



**ME** **METHAMPHETAMINE**

**FACT VS. FICTION AND LESSONS FROM THE CRACK HYSTERIA**

**METHAMPHETAMINE: FACT VS. FICTION AND LESSONS FROM THE CRACK HYSTERIA**

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## **Executive Summary**

The purpose of this report is to provide a critical examination of the available evidence on illicit methamphetamine use and its consequences in the United States and internationally. It is the aim of this report to dispel some of the myths about the effects of methamphetamine and other illicit drugs using the best available scientific data. Further, it is our hope that this analysis will lead to more rational policies for dealing with both legal and illegal amphetamine. The report begins with an examination of the lessons learned from the “crack cocaine scare” in the 1980s. In this way, the reader can draw parallels between society’s response to crack cocaine then, and methamphetamine now. The report then describes distinctions and similarities between methamphetamine and other amphetamine-type stimulants. Also examined is the prevalence of methamphetamine use and public policies in response to the perceived increased use of the drug and perceived drug-related problems. Finally, the report critically reviews the scientific literature on the effects of methamphetamine on the brain, physiology, and behavior. The data show that many of the immediate and long-term harmful effects caused by methamphetamine use have been greatly exaggerated just as the dangers of crack cocaine were overstated nearly three decades ago. Recommendations are made in an effort to remedy this situation and to enhance public health and safety.

## Introduction

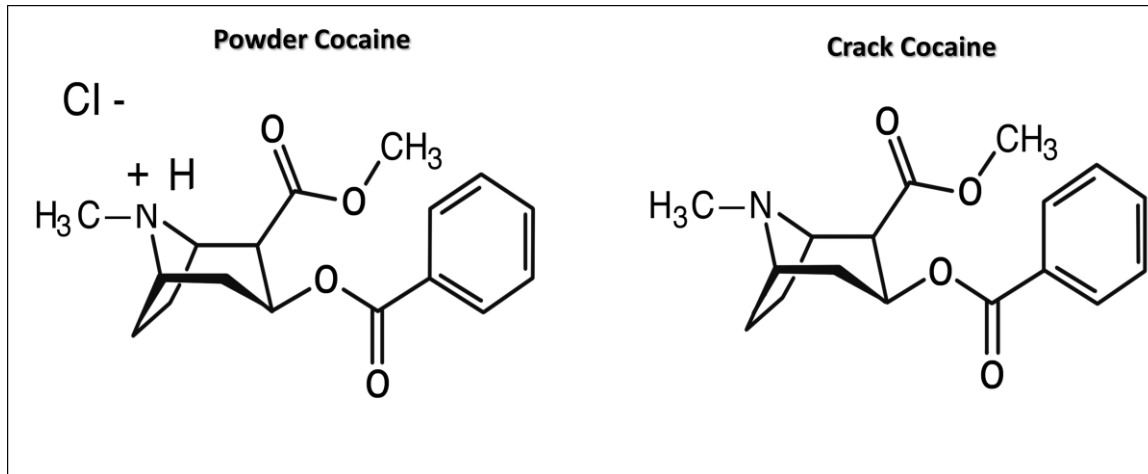
### *Lesson Learned from Crack in the United States*

The emotional hysteria that stems from misinformation related to certain illegal drugs often leads to more harm than the drugs themselves. In the United States during the mid-1980s, for example, crack cocaine was believed to be so powerfully addictive that even first-time users would become addicted. Even more worrisome was the perception that the drug produced unpredictable and deadly effects. Despite the fact that there was virtually no real evidence supporting these claims, in 1986, the United States Congress passed the now infamous Anti-Drug Abuse Act setting penalties 100 times harsher for crack than for powder cocaine convictions. The law stated that a person convicted of selling 5 grams of crack cocaine was required to serve a minimum sentence of five years in prison; convictions involving 50 grams of crack carried a minimum sentence of 10 years. To receive similar sentences for trafficking in powder cocaine, individuals needed to have been caught selling (or intending to sell) 500 grams and 5 kilograms of cocaine, respectively.

By 1988, concern about the drug had increased so much that penalties of the 1986 law were extended to persons convicted of simple possession. Even first-time offenders faced such stiff mandatory sentences. Simple possession by a first-time offender of any other illegal drug, including powder cocaine or heroin, carried a *maximum* penalty of one year in prison.

The driving force behind passage of these anti-crack laws was the exaggerated claims made in the media on a near-daily basis. Multiple stories warned of “crack-crazed” addicts. In the months before the 1986 elections, more than 1,000 stories on cocaine appeared in the national press, including five cover stories in *Time* and *Newsweek*. *Time* magazine called crack cocaine the issue of the year. When the furor about crack had settled, two things were clear. First, media accounts about the effects of the drug were inconsistent with the scientific data. There are no pharmacological differences between crack and powder cocaine to justify their differential treatment under the law. Figure 1 shows the chemical structures of cocaine hydrochloride and cocaine base (crack). The structures are nearly identical.

**Figure 1 Chemical structure of cocaine hydrochloride (powder cocaine) and cocaine base (crack)**



The two forms of cocaine produce identical effects; these effects are predictable (Hatsukami and Fischman, 1996). That is, as the dose is increased, so are the effects, whether they are blood pressure and heart rate *or* subjective “high” and addictive potential. The way the drug is taken differs based on its form, however. Crack is smoked, whereas powder is swallowed, snorted, or injected. More intense effects are observed when the drug is smoked or injected intravenously, but the drug itself remains the same. To punish crack users more harshly than powder users is analogous to punishing those who are caught smoking marijuana more harshly than those caught eating marijuana-laced brownies.

Another consequence that became apparent was that the differences between crack and powder laws disproportionately targeted blacks. A whopping 85 percent of those sentenced for crack cocaine offenses were black, despite the fact that the majority of users of the drug were white (USSC 1995, 1997, 2002, 2007). Frustrated by the lack of willingness of lawmakers to eliminate the disparity between crack and powder cocaine penalties, in 2007, even presidential candidate Barack Obama voiced strong concerns: “...let’s not make the punishment for crack cocaine that much more severe than the punishment for powder cocaine when the real difference between the two is the skin color of the people using them...That will end when I am President.” On August 3, 2010, President Obama signed legislation that decreased, but did not eliminate, the sentencing disparity between crack and powder cocaine offenses. The new law reduced the sentencing disparity from 100:1 to 18:1.

While we recognize the improvement this compromise represents, it remains an inadequate solution because the scientific evidence does not support disparate penalties for crack and powder cocaine violations. And when one considers the perception of the egregious racial injustice that occurred in enforcing these laws, it is not difficult to draw a similar conclusion to the one drawn by Malcolm X in 1964 when asked whether the U.S. had made sufficient progress towards racial equality. He said “If you stick a knife in my back nine inches and pull it out six inches, there is no progress... The progress is healing the wound.”

*Methamphetamine: the new crack*

There are multiple signs indicating that methamphetamine is the current drug about which exaggerated media claims are made. As with the “crack scare” of the 1980s, many high-profile stories about methamphetamine have appeared in the global press. On August 8, 2005, for example, *Newsweek* ran a dramatic cover story called “The Meth Epidemic.” Use of this drug, according to the magazine, had reached epidemic proportion. The evidence suggested otherwise. At the height of methamphetamine’s popularity, there were never more than a million current users of the drug in the United States. This number is considerably lower than the 2.5 million cocaine users, the 4.4 million illegal prescription opioid users, or the 15 million marijuana smokers during the same period. In the United States, the number of methamphetamine users has never come close to exceeding the number of users of these other drugs (SAMHSA 2012).

Media coverage has been filled with accounts of desperate users turning to crime to support their use of the “dangerously addictive” drug. Many articles focused on the “littlest victims.” The *New York Times* headlined one story, “Drug Scourge Creates Its Own Form of Orphan,” describing an apparent rise in related foster care admissions and reports of addicted biological parents who were impossible to rehabilitate. The paper quoted a police captain who said methamphetamine “makes crack look like child's play, both in terms of what it does to the body and how hard it is to get off” (Butterfield 2004). The paper also claimed: “Because users are so highly sexualized, the children are often exposed to pornography or sexual abuse, or watch their mothers prostitute themselves” (Zernike 2005).

Then United States Attorney General Alberto Gonzales called it “the most dangerous drug in America,” and President George W. Bush proclaimed November 30, 2006 National Methamphetamine Awareness Day. Back in 1986, President Ronald Reagan proclaimed the entire month of October Crack/Cocaine Awareness Month.

The parallels are clear enough to demonstrate that history is repeating itself—mistakes and all. The emotional hysteria driving public policy on methamphetamine is causing great harms that are avoidable. In an effort to avoid making many of the same mistakes that were made with crack cocaine with regard to stigma, public policy and general misinformation, the purpose of this report is to provide a critical examination of the available evidence on methamphetamine use and its consequences in the United States and internationally. It is the aim of this report to dispel some of the myths about the effects of methamphetamine and other illicit drugs using the best available scientific data. Further, it is our hope that this analysis will lead to more rational policies for dealing with both legal and illegal amphetamine.

### **What is methamphetamine?**

Amphetamine is a class of chemical compounds that includes drugs used for both medical and recreational purposes. Of this class, *d*-amphetamine and methamphetamine are approved in several countries to treat a variety of disorders, including attention-deficit hyperactive disorder (ADHD), narcolepsy, and obesity. These drugs as well as other amphetamines [e.g., 3,4-methylenedioxyamphetamine (MDA), and 3,4-methylenedioxymethamphetamine (MDMA: as known as ecstasy, Molly, or Mandy)] are used recreationally.

In recent years, a range of other substances has also been grouped with amphetamine to comprise a class of drugs known as amphetamine-type stimulants (ATS). In addition to the amphetamines mentioned, other ATS include, but are not limited to, methcathinone, fenetylline, ephedrine, pseudoephedrine and methylphenidate. The major rationale for the inclusion of these drugs as ATS is that they are synthetic stimulants. Cocaine is not an ATS because it is not manufactured synthetically, although it is classified as a stimulant. From a behavioral or neuropharmacological perspective, the grouping of ATS makes little sense. Methylphenidate (Ritalin) and cocaine produce similar effects on human behavior and on monoamine neurotransmitters, which modulate mood and other functions.

Yet, only methylphenidate is included as an ATS. Hence, the ATS grouping seems somewhat arbitrary. Another concern is that such grouping facilitates the exaggeration of the extent of global amphetamine use because a large number of different drugs are all included in one class.

For purposes of this report, the primary focus will be on methamphetamine. However, because global ATS prevalence data include other compounds as well, an exclusive focus on methamphetamine is nearly impossible. With this caveat in mind, our rationale for limiting the focus on methamphetamine, where possible, is because it has generated the greatest amount of concern. Indeed, periodically there are statements in the scientific and popular literature attesting to methamphetamine's greater potency and "addictive" potential, relative to other amphetamines. Such statements, however, are inconsistent with the empirical evidence. In carefully controlled laboratory studies of human research participants, *d*-amphetamine and methamphetamine produce nearly identical physiological and behavioral effects (Martin et al. 1971; Sevak et al. 2009; Kirkpatrick et al. 2012). They both increase blood pressure, pulse, euphoria, and desire to take the drug in a dose-dependent manner. Essentially, they are the same drug.

One reason for the unfounded beliefs about the drugs might be related to the fact that methamphetamine is more readily available on the illicit market due to its apparent easier synthesis. A quick search of the internet can provide the surfer with dozens of "How to make meth" recipes. According to these recipes and law enforcement personnel, methamphetamine can be 'easily' made from a few common products, the most important of which is the over-the-counter cold medication, pseudoephedrine. This makes methamphetamine all the more accessible to the poor, the marginalized, those wishing to avoid the medical establishment, and other interested consumers. There is a large underclass of people who cannot afford to see a physician in order to receive a referral to a psychiatrist to obtain a prescription for legal amphetamine. Moreover, there are many people who are skeptical of psychiatrists, who consider it a stigma to be under psychiatric care, or who, quite rationally, do not want any mental health issues to appear on their medical records.



Undoubtedly, there are also many people who have no medical or neurological condition that would qualify them for a prescription, but who nonetheless seek stimulants for personal reasons, be they work-related or recreational. As a result, it is not surprising that methamphetamine is the most frequently abused amphetamine.<sup>1</sup> But this has less to do with pharmacology and more to do with access or availability.

### **Global extent of methamphetamine use**

Nonetheless, over the past two decades excessive illicit amphetamine use has become a major global concern. According to data from the United Nations Office on Drugs and Crime (UNODC) in 2011, ATS were used at rates higher than any other drug class with the exception of cannabis (UNODC, 2013). The number of cannabis users worldwide (on an annual basis) is estimated to be about 180 million, ATS users about 34 million, opiate users 16.5 million, and cocaine users about 17 million (UNODC 2013). These estimates are based on information provided by individual governments to UNODC, and confidence intervals around them are wide. The high side of the confidence interval for ATS use, for example, indicates that as many as 50 million people may use it during a year. Data from some governments distinguish methamphetamines from other ATS, while others do not

In many countries, there are no population-based data on the extent of illicit drug use. Since illicit drug use in many countries is subject to severe criminal sanctions, it is likely that a substantial proportion of illicit drug users are unwilling to disclose information about their use, especially to government-sponsored survey takers. Of course, these factors affect illicit drug use estimates generally, but data collection on consumption of ATS is even more fraught with difficulty than with respect to other drugs. This is because, unlike the processing of coca into cocaine and of opium into heroin and other opiates, the manufacture of methamphetamine does not rely on primary products that can be grown only in special locations. As noted by Degenhardt and colleagues (2010), methamphetamine can be and is manufactured in a much wider variety of locations and “under more clandestine conditions and for comparatively less cost.”

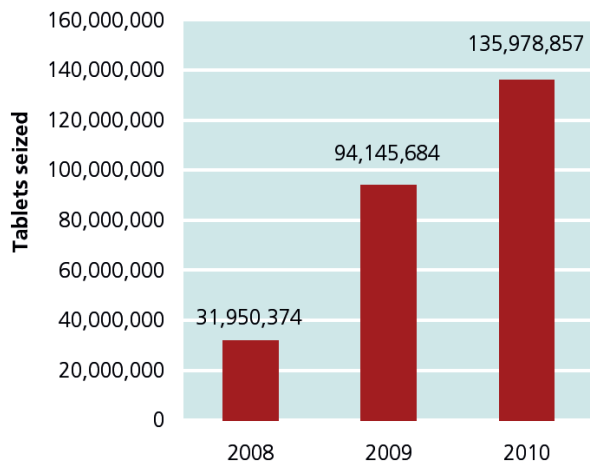
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<sup>1</sup> The terms “abuse” and “dependence”, as they are used throughout this report, conform to the Diagnostic and Statistical Manual of Mental Disorders 4<sup>th</sup> Edition (DSM-IV-TR) and International Statistical Classification of Diseases and Related Health Problems (ICD-10) definitions of substance abuse and dependence. DSM-IV-TR and ICD-10 terminology are used to avoid the use of pejorative words and terminology that have multiple meanings.

A good illustration of this point is the production of homemade methamphetamine (known as pervitin) in the Czech Republic. In the early 1970s, according to Záborský (2007), one individual perfected a simple process for producing pervitin from an ephedrine-containing cough medicine available over the counter and the technique quickly spread, even at the risk of harsh punishment during the period of Soviet control. Pervitin use and production are perceived to be extensive in the Czech Republic even today (Mravcik et al. 2011), although data supporting this perception are lacking.

While absolute numbers of users of ATS at a given moment is difficult to estimate, better trend estimates of use can be discerned when prevalence data are combined with government seizure statistics over time. Based on such data, UNODC's most recent report concluded that markets for methamphetamine are growing faster than for other ATS, fueled in part by significant increases in East and Southeast Asia, the United States and Mexico in the last five or six years (UNODC 2012). Figure 2 illustrates the dramatic increase in seizures in East and Southeast Asia since 2008. Methamphetamine markets in Europe, while small relative to those of North America, have also expanded since 2009 based on seizure data, especially in Scandinavia and Turkey (UNODC 2012). Seizure data alone may be misleading in that increases in seizures may reflect better investments in policing rather than real increases in production, and declines in seizures may reflect producers' improved ability to evade capture rather than a real decline in production. This point highlights the importance of combining multiple types of data to shed light on drug use prevalence rates.

On behalf of the United Nations Reference Group on HIV and Injecting Drug Use, Degenhardt et al. (2010) conducted an extensive review of the literature on the extent of methamphetamine and ATS use and means of consumption around the world. They observed that all regions of the world have some documented illicit ATS use, including methamphetamine use. While production was highest in the Middle East, Southeast Asia and North America (including Mexico), production in Africa, especially South Africa, was increasing (Degenhardt et al. 2010). Some have suggested that stringent measures taken in the United States to limit access to methamphetamine precursors (e.g., pseudoephedrine) have decreased United States production but have increased production in Mexico (Colfax et al. 2010).

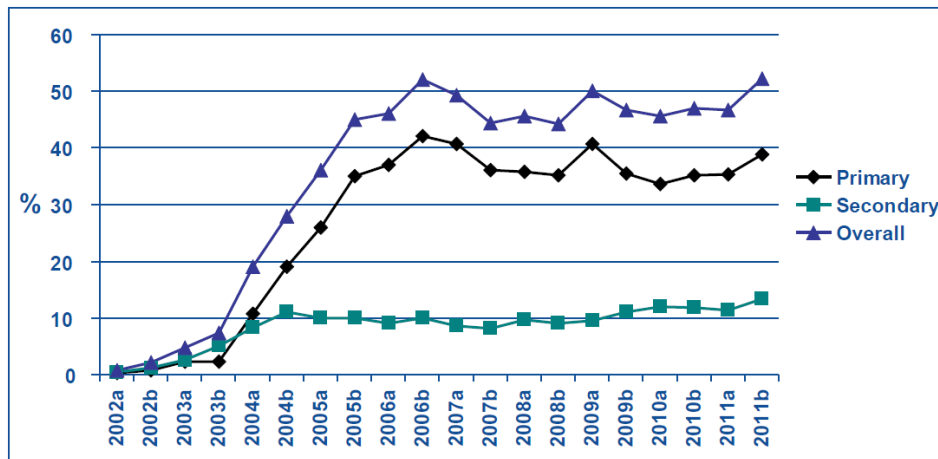
**Figure 2. Methamphetamine tablets seized in East and South-East Asia, 2008-2010**

Source: UNODC, *World Drug Report 2012*, p 53.

Data from drug treatment centers that track the preferred drugs among those seeking treatment can also aid in determining ATS prevalence rates. But, as is the case with seizure data, this information should be used in combination with other information such as drug use prevalence data because it too, when used alone, has serious limitations. For example, it is likely that many marginalized drug abusers are under-represented in such data because drug treatment may not be available to them for a variety of reasons ranging from an inability to pay for services to a lack of treatment facilities. On the other hand, some individuals may be encouraged to exaggerate the extent of their drug use in order to receive drug treatment in lieu of receiving a prison sentence. So, it is conceivable that a large proportion of ATS users are excluded from this type of data or the drug use rates of treatment-seekers may be less than accurate. These caveats notwithstanding, Figure 3 shows that the percentage of individuals seeking treatment for methamphetamine use dramatically increased in the Western Cape Province of South Africa between 2002 and 2011 (SACENDU 2012). Based on the available information, it might be supposed that the number of clandestine amphetamine laboratories has risen along with the number of people seeking treatment for amphetamine use problems. As we noted above, the increased number of amphetamine seizures could simply reflect a greater focus, in terms of resources and attention, by various law enforcement agencies to this problem. Similarly, in recent years the presence of more treatment facilities may also have increased the number of people seeking amphetamine-related treatments.

Even with these caveats, it seems clear that the extent of illicit methamphetamine use deserves of the attention of public health officials. The strategies for addressing problems related to illicit amphetamine use and abuse, however, should be mindful of the strengths and limitations of our current data.

**Figure 3 Methamphetamine as primary and secondary drug of use among people seeking treatment in Western Cape Province, South Africa**



Source: SACENDU Research Brief 15(1), 2012

### **Policy, law and programs: national and multinational responses to methamphetamines**

#### *Methamphetamine scare: Scare tactics sold as drug prevention*

Like crack cocaine, methamphetamine in North America has been the object of sensational “scare” campaigns in the media, fed by nearly everyone -- from scientists to healthcare professionals to law enforcement personnel to politicians. Jenkins (1994) describes an early such flood of media reports on “ice” or smokable methamphetamine, which resulted in Congressional hearings and a national panic, though consumption of ice was largely confined to Hawaii. Media reports suggested that the whole of the US would be deluged by “epidemic” levels of use of ice in a short time, ushering in an “ice age” (Jenkins 1994). Jenkins concluded that the demonization of crack versus powder cocaine paved the way for the demonization of ice versus other amphetamines, and that the ice scare served a particular political purpose for certain politicians in Hawaii and beyond who needed to portray themselves as “tough on drugs.” Indeed, as discussed above, a similar situation occurred with crack cocaine in the late 1980s.

Both liberal and conservative politicians added their voice to the cacophony that blamed crack for everything from premature death to child abandonment and neglect to high crime and unemployment rates.

A decade later – in the late 1990s – the methamphetamine hysteria in the United States began to generate considerable interest among public officials; some were even making explicit connections with crack cocaine in their demonizing of the drug. Then Oklahoma Governor Frank Keating characterized methamphetamine in this way: “It’s a white trash drug -- methamphetamines largely are consumed by the lower socio-economic element of white people and I think we need to shame it. “Just like crack cocaine was a black trash drug and is a black trash drug” (Senate Communications Division, 1999).

Since 2000, concerns about methamphetamine have further intensified. Most media portrayals of methamphetamine use emphasized unrealistic effects and exaggerated the harms associated with the drug. For example, in January 2010, National Public Radio (NPR) ran a story entitled, “This is your face on meth, kids” (NPR, 2010). The story described a California sheriff who was trying to stop young people from experimenting with methamphetamine. With the help of a programmer, he developed a computer program that digitally altered teenagers’ faces to show them what they would look like after using methamphetamine for 6, 12, and 36 months. These young people watched their images change from those of healthy, vibrant individuals to faces marred by open scabs, droopy skin, and hair loss. They were told that these were the direct physiological effects of using methamphetamine. Ninety percent of individuals who tried methamphetamine once, they were also told, would become “addicted.”

There is no empirical evidence to support the claim that methamphetamine causes physical deformities. Of course, there have been the pictures of unattractive methamphetamine users in media accounts about how the drug is ravaging some rural town. The infamous “meth mouth” images (extreme tooth decay) have been widely disseminated. But, consider this: methamphetamine and Adderall are essentially the same drug. Both drugs restrict salivary flow leading to xerostomia (dry mouth), one proposed cause of “meth mouth.” Adderall and generic versions are used daily and frequently prescribed – each year they are among the top 100 most prescribed drugs in the United States – yet there are no published reports of unattractiveness or dental problems associated with their use. The physical changes that occurred in the dramatic

depictions of individuals before and after their methamphetamine use are more likely related to poor sleep habits, poor dental hygiene, poor nutrition and dietary practices, and media sensationalism. With regard to the addictiveness of methamphetamine, the best available information clearly shows that the overwhelmingly majority of people who try methamphetamine will not become addicted (O'Brien and Anthony 2009). Less than 15 percent of those who have ever used the drug will become addicted. The bottom line is that the overwhelming majority of methamphetamine users use the drug without problems.

Although the hysteria about methamphetamine and crack cocaine use shared many similarities, the perceived users of the respective drugs differed, as indicated above by former governor Keating. Crack users were believed to be black and urban, whereas methamphetamine users were seen as white and rural. As a result, at the start of the new millennium an intense electronic media campaign was initiated targeting many rural communities in the western United States. The stated goal was to prevent young people from experimenting with methamphetamine. In 2005, the state of Montana adopted a graphic advertising campaign called the *Montana Meth Project*. The advertisements show in horrifying details a young person who uses methamphetamine for the first time, and then ends up engaging in some unthinkable act such as prostitution or assaulting strangers for money to buy methamphetamine. At the end of the advertisement, printed on the screen is the message: "Meth, not even once." A year after its inception, the Montana Meth Project was recognized by the White House for its innovative approach to drug prevention. Indeed, preliminary findings from a report by the Montana Department of Justice suggested that the campaign was successful at decreasing methamphetamine use among teens. This apparent success led several other states to join the "Meth Project" and adopt identical advertising strategies.

Is it true that scare tactic campaigns decrease drug use? More complete evidence would suggest not. A recent critical review of the impact of the Montana Meth Project on methamphetamine use indicated that the advertisement campaign had no effect when preexisting downward trends in methamphetamine use were taken into account (Anderson 2009).

One potential reason for the lack of success is that the population most likely to use methamphetamine finds the advertisements laughable because they exaggerate methamphetamine-related harmful effects. These individuals most likely know people who have used the drug, and the information presented in the advertisements is inconsistent with their own knowledge. Here, the parallel is to early propaganda about marijuana and the film “Reefer Madness.” The film is now a ‘cult classic’ because it is widely regarded as a comedy as well as a cautionary tale of official propaganda.

This raises the question, what are the potential negative consequences of presenting exaggerated or misleading information about drugs to young people? Some educators and healthcare professionals have expressed concern that the types of embellishment used by the Montana Meth Project decrease their credibility and relevance and lead many young people to reject other drug-related information from “official” sources, even when the information is accurate. Despite these concerns and no evidence of preventing drug use, the Meth Project remains popular with some officials and continues to use these types of scare tactics (see, <http://www.methproject.org/>).

### ***Laws and policies***

Veracity aside, the anti-methamphetamine media campaign and incredible statements made about the drug has led to considerable concern by the general public, which, in turn, inspired policy-makers to pass new legislation. In Canada, for example, a particularly concerted media “scare” campaign on methamphetamine was followed quickly by the legal reclassification of both methamphetamine and its constituent ingredients or precursors, as noted by Boyd and Carter (2010):

Substances commonly used in the production of methamphetamine, such as red phosphorus and hydriodic acid, were added to the list of Class A [i.e. most highly criminalized] precursors....In August 2005, methamphetamine was rescheduled from a Schedule II to a Schedule I drug under the *Controlled Drugs and Substances Act*. Thus the maximum penalties now apply to methamphetamine, including the possibility of life sentences for trafficking, and production and possession can garner sentences of up to seven years.

Since Boyd and Carter's analysis was published, the government of Canada passed into law a wide-ranging crime bill that imposes mandatory minimum sentences for many categories of drug crimes (Cohen 2012). This measure will likely mean that for minor methamphetamine convictions, for example, a judge inclined to give a lighter sentence will not be able to do so.

In some countries, the perception of problems associated with the abuse of amphetamine has become so worrisome that even more drastic measures have been taken. Topping the list is Iran, which has a mandatory death penalty for possession of 30 grams of methamphetamine in a regime with extremely harsh penalties for all drug offenses. Moreover, the executions can be drawn out and extremely painful (Mogelson, 2012). In response to reports of precipitous increases in methamphetamine abuse, in 1996 the government of Thailand banned all uses of amphetamine, including those for medical purposes (Pilley and Perngpam, 1998). Other governments have also taken steps to restrict legal uses of amphetamine, although most have not been as extreme as those taken in Iran and Thailand. For example, in the United Kingdom and New Zealand, while *d*-amphetamine remains available for medical purposes, any use of methamphetamine (including medical use) has been banned. Recall that *d*-amphetamine and methamphetamine are essentially the same drug (e.g., Kirkpatrick et al. 2012).

In the United States, new laws have also been implemented in response to the increased negative advertising campaign waged against the drug. These laws focus on both methamphetamine and compounds used to make the drug illicitly (i.e., precursors). Amphetamine tablets were available over the counter in the United States until the early 1950s (Maxwell and Rutkowski 2008). In 1970, in response to perceived abuses of the drug in the 1960s, amphetamine was placed under Schedule II of the newly passed Controlled Substances Act. This meant that all amphetamines were classified under the most restrictive category for drugs available by prescription. Other Schedule II drugs include cocaine and morphine. More recently, the Comprehensive Methamphetamine Control Act of 1996 increased criminal penalties for trafficking and producing methamphetamines. The law also restricted access to precursors, including ephedrine and pseudoephedrine, which were key ingredients in over-the-counter cold medicines (Franco 2005).



Another change in the United States -- the Methamphetamine Penalty Enhancement Act of 1998 -- lowered the cut-off that would trigger mandatory sentences for methamphetamine trafficking. The Methamphetamine Anti-Proliferation Act of 2000 imposed restrictions on access to precursors, including blister packs for over-the-counter ephedrine and pseudoephedrine. The Combat Methamphetamine Epidemic Act of 2005 increased restrictions on pseudoephedrine. Pharmacists and sellers of medications containing pseudoephedrine were required to place these medications behind the counter and buyers were required to show a state-issued identification card and sign a log that could be used to track their purchases (Gonzales et al. 2010). Fearing that this inconvenience would decrease sales, many pharmaceutical companies simply replaced pseudoephedrine with phenylephrine. Unfortunately, compared with pseudoephedrine, phenylephrine is a less effective nasal decongestant, the condition for which these medications are most often used (Eccles 2007). This important unintended consequence is rarely discussed among supporters of these laws.

Given the fact that new laws in the United States restrict availability of an effective cold and flu medication (pseudoephedrine), it is of interest to know whether this approach is effective at decreasing the availability of methamphetamine. Dobkin and Nicosia (2009) studied this question by focusing on two major seizures of pseudoephedrine in the United States in 1995. They concluded that this intervention substantially disrupted the supply of methamphetamine, but that the effect was only temporary. Within four to 18 months, the methamphetamine market had returned to pre-intervention levels. This result suggests that legislation aimed at restricting precursors such as pseudoephedrine may have only short-term effects on illicit drug markets, while it permanently reduces the ability of the whole population to obtain effective cold medications.

### **Methamphetamine effects on the brain**

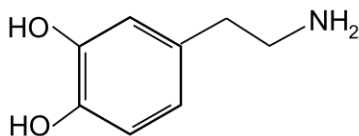
The intense focus on restricting access to methamphetamine suggests that it must be one of the most dangerous drugs with respect to the potential damage to the brain and behavior. Over the past several decades, data from basic research have contributed to an increased understanding of methamphetamine-related effects on cells in the brain.

A comprehensive review of the effects of methamphetamine on the brain is beyond the scope of the current report, and excellent reviews already exist (e.g., Sulzer et al. 2005; Fleckenstein et al. 2007). Here, we present a brief overview because we think it will be useful for the reader to have some understanding of the neurotransmitters involved in the actions of methamphetamine. In this way, we hope to provide the reader with tools to evaluate more accurately “brain statements” made about methamphetamine in the popular media and scientific literature.

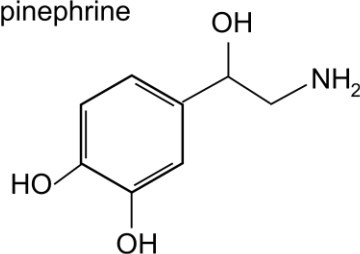
As Figure 4 indicates, amphetamine-related drugs bear a striking resemblance to the catecholamine neurotransmitters dopamine and norepinephrine. Catecholamine neurotransmitters are known to play an important role in several behaviors, including movement and mood regulation. They are also known to be involved in malfunctions that lead to disease states. For example, a prominent theory guiding the treatment of clinical depression proposes that too little activity of the neurotransmitters dopamine, norepinephrine, and/or serotonin (together known as monoamine neurotransmitters) can cause depression and too much can cause a manic state. This theory also postulates that drugs such as methamphetamine lead to abuse because they stimulate the “dopamine reward system,” which is responsible for telling the rest of the brain “that’s good—do that again.” Thus, the structural similarities between amphetamine and catecholamine neurotransmitters provide clues about the drugs’ mechanisms of action.

**Figure 4 Chemical structures of neurotransmitters and amphetamines**

Dopamine

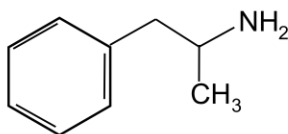


Norepinephrine



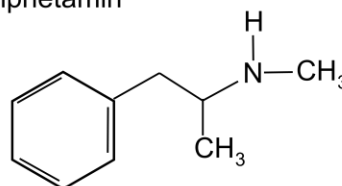
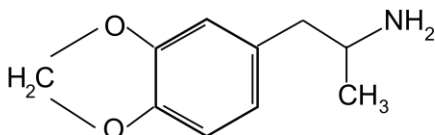
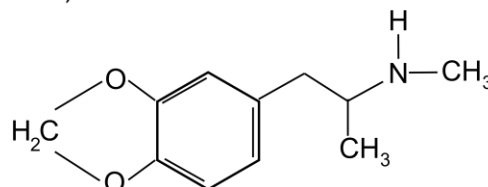
Amphetamin

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Methamphetamin

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3,4-Methylenedioxyamphetamin  
(MDA)3,4-Methylenedioxymethamphetamin  
(MDMA)

Multiple lines of evidence demonstrate that amphetamine causes a release of monoamines from brain cells. Behaviorally, this can have the effect of elevating mood, increasing alertness and vigilance, while decreasing tiredness and sleepiness (e.g., Hart et al. 2003, 2005). These effects can be beneficial for individuals required to work extended hours or workers who have to perform at the circadian nadir of alertness. Undoubtedly, this is the reason that several nations' militaries have used (and continue to use) amphetamine since World War II (Caldwell and Caldwell 2005). The drug helps soldiers fight better and longer. The performance enhancing benefits are widely recognized by students and professionals in most every field from athletics to zoology.

A downside to amphetamine effects on monoamine neurotransmitters is that the drug may also produce toxicity to cells containing these neurotransmitters. An accumulating amount of evidence shows that amphetamine, when administered repeatedly in large doses, promotes the formation of free radicals that damage brain cells. Following release of dopamine, for example, certain enzymes inactivate the neurotransmitter. But, abnormally enhanced dopamine activity – caused by repeated administration of large amphetamine doses – may produce an increased formation of free radicals and thereby cause cell injury (Cadet and Krasnova 2009). This, in turn, could lead to persistent deficits in the functioning of dopamine-containing cells.

This knowledge has raised concerns about the potential harmful consequences of methamphetamine abuse on the human brain and behavior. Dopamine-rich areas serve a wide range of important human functions ranging from mood to movement to learning and memory. Indeed, a substantial database collected in laboratory animals suggests that acute and long-term administration of amphetamine produces disruptive effects in several cognitive domains, including learning and memory (for review, see Marshall et al. 2007). There is, however, an important limitation associated with many of these studies when extrapolating the findings to humans: the dosing regimens used did not capture key elements of human recreational amphetamine use, specifically gradual dose escalation. Typically, investigators administered large doses of methamphetamine repeatedly for one or more consecutive days to drug-naïve animals, whereas human recreational drug users usually start with smaller amounts and increase their doses gradually as their drug use progresses. This difference is not trivial because the harmful neurobiological and behavioral changes that occur in response to repeated large doses of methamphetamine can be prevented with prior exposure to several days of escalating doses (Segal et al. 2003; O’Neil et al. 2006; Belcher et al. 2008). Given this situation, it is critically important to employ more ecologically relevant models in future animal studies investigating the impact of amphetamine use on cognitive functioning. These issues underscore the importance of carefully assessing behaviors of interest in humans under rigorous conditions.

## **Methamphetamine effects on human physiology and behavior**

### ***Immediate effects***

There is now a large database investigating the direct effects of methamphetamine on human physiology and behavior (for review, see Hart et al. 2012). These laboratory studies are designed to document the immediate and short-term effects of the drug on measures of cognitive functioning, mood, sleep, blood pressure, heart rate and the drug's addictiveness. These studies employ carefully controlled, within-participant designs, during which participants: (1) complete a baseline battery of tests, which includes the measures of interest; (2) are administered a methamphetamine dose (ranging from placebo to 50 mg); and (3) are reassessed on the battery at predetermined time points for several hours after drug administration. Also, all of the drug doses are given in a double-blind manner – the research participants don't know whether they are getting a placebo or real methamphetamine, nor do the medical staff monitoring the sessions.

The findings to date can be summarized in the following way. After methamphetamine administration, participants reported feeling more euphoric and their cognitive functioning was improved. These effects lasted about four hours. The drug also caused significant increases in blood pressure (BP) and heart rate that lasted for up to 24 hours. The maximum levels were about 150/90 (BP) and 100 (beats per min.). While these elevations were statistically significant, they were well below levels obtained when engaged in a rigorous physical exercise, for example. Another finding was that the drug reduced the amount of time participants slept (Perez et al. 2008). For example, when they took placebo, participants got about eight hours of sleep, but when the 50 mg dose was given, they got only about six hours. Together, the results indicate that a large dose of methamphetamine produced expected effects. The drug didn't keep people up for consecutive days, it didn't dangerously elevate their vital signs, nor did it impair their judgment.

Tellingly, the above human laboratory data are at odds with anecdotal reports and conventional wisdom. Perhaps researchers had not asked the right question. One of the most popular beliefs about methamphetamine is that it is highly addictive, more so than any other drug. So, in another set of experiments, Hart and colleagues set out to address this issue. Under one condition, methamphetamine-dependent individuals were given a choice between taking a big hit of methamphetamine (50 mg) or \$5 in cash. They chose the drug on about half of the opportunities. But, when we increased the amount of money to \$20, they almost never chose the drug (Kirkpatrick et al. 2012). Similar results were observed among crack cocaine addicts in an earlier study (Hart et al. 2000). Thus, the addictive potential of methamphetamine was not as had been claimed; its addictiveness was not extraordinary. These results also showed that methamphetamine addicts, just like crack addicts, can and do make rationale decisions, even when faced with a choice to take the drug or not. This was consistent with the literature assessing cognitive functioning of methamphetamine users, but as noted below, only if one looked carefully and guarded against much of the biased information that influences public perceptions about methamphetamine users (Hart et al. 2012).

While the above results suggest that many of the immediate harmful effects caused by methamphetamine use may have been greatly exaggerated, there are real documented potential negative consequences associated with this drug. For instance, methamphetamine abuse is associated with multiple deleterious medical consequences, including paranoia mimicking full-blown psychosis (Grelotti et al. 2010) and hypertensive crisis leading to stroke (Ho et al. 2009). While serious, such cases are rare, and entail the long-term use of extremely large doses. Still, in order to better understand the potential consequences of long-term use of methamphetamine, in the next section we review the literature on methamphetamine-related brain and cognitive effects by examining individuals who have used the drug illicitly for several years.

### *Long-term effects*

What are the long-term effects of methamphetamine on intelligence and brain functioning of addicts? These are people who had used the drugs for many years. In these studies, abstinent methamphetamine addicts and a control group (usually non-drug users) completed a comprehensive set of cognitive tests over the course of several hours, and the results were compared to determine whether or not the cognitive functioning of the methamphetamine addicts was normal. Of course, normality is a relative concept that is determined not only by comparing performance of the methamphetamine group with the performance of a control group, but also by comparing the methamphetamine group's scores with those from a normative dataset, taking into consideration each individual's age and level of education. These requirements are important because they allow us to take into account the relative contribution of age and education in terms of the individual's score and adjust the score accordingly. Simply stated, it would be inappropriate to compare the vocabulary scores of a 16-year-old high school dropout with those of a 22-year-old college graduate. The older college graduate would be expected to outperform the younger high school dropout.

A biased and less than careful reading of the scientific literature might lead one to conclude that methamphetamine addicts have severe cognitive impairment. In one study by Sara Simon and colleagues, the apparent impairments observed among methamphetamine users were so bad that it led the researchers to warn: "The national campaign against drugs should incorporate information about the cognitive deficits associated with methamphetamine...Law enforcement officers and treatment providers should be aware that impairments in memory and in the ability to manipulate information and change points of view (set) underlie comprehension... methamphetamine abusers will not only have difficulty with inferences... but that they also may have comprehension deficits... the cognitive impairment associated with [methamphetamine abuse] should be publicized..." (Simon et al. 2002). But, as one reads this and similar papers more critically there are strong grounds for skepticism. While the data showed that the control participants outperformed methamphetamine users on a few tests, the performance of the two groups was not different on the majority of tests.

More importantly, when the cognitive scores of the methamphetamine addicts in the Simon study are compared against scores in a larger normative dataset, none of the methamphetamine users' scores fall outside the normal range (Hart et al. 2012). Based on all of the data, this indicates that the cognitive functioning of the methamphetamine users is normal. This should have tempered the researchers' conclusions and prevented them from stating such dire warnings. Unfortunately, the inappropriate conclusions drawn by Simon and colleagues are not atypical. The methamphetamine literature is filled with similar unwarranted conclusions. As a result, the apparent methamphetamine addiction-cognitive impairment link has been widely publicized - numerous articles have appeared in scientific journals and the popular press. We contend that this has helped shape an environment in which there is an unwarranted and unrealistic goal of eliminating methamphetamine use at any cost to amphetamine users and to the public.

The reporting of brain imaging findings has been especially misleading. On July 20, 2004, for example, *The New York Times* printed an article entitled, "This Is Your Brain on Meth: A 'Forest Fire' of Damage." It stated: "People who do not want to wait for old age to shrink their brains and bring on memory loss now have a quicker alternative—abuse methamphetamine . . . and watch the brain cells vanish into the night." This conclusion was based on a study that used magnetic resonance imaging (MRI) to compare brain sizes of methamphetamine addicts with those of non-drug-using healthy individuals (Thompson et al. 2004). The researchers also assessed the correlation between memory performance and several brain structural sizes. They found that methamphetamine users' right cingulate gyrus and hippocampus were smaller than controls by 11 and 8 percent, respectively. Memory performance on only *one* of four tests was correlated with hippocampal size (i.e., individuals with larger hippocampal volume performed better). As a result, the researchers concluded, "chronic methamphetamine abuse causes a selective pattern of cerebral deterioration that contributes to impaired memory performance." This interpretation, as well as the one printed in *The New York Times* article, is inappropriate for several reasons.

First, brain images were collected at only one time point for both groups of participants. This makes it virtually impossible to determine whether methamphetamine use *caused* "cerebral deterioration," because there might have been differences between the groups even before methamphetamine was ever used. Second, the non-drug users had significantly higher levels of education than methamphetamine users (15.2 vs. 12.8 years, respectively);



it is well established that higher levels of education lead to better memory performance. Third, there were no data comparing methamphetamine users with controls on any memory task. This, in itself, precludes the researchers from making statements regarding impaired memory performance caused by methamphetamine. Nonetheless, the only statistically significant cognitive finding was a *correlation* of hippocampal volume and performance on one of the four tasks. This finding is the basis for the claim that methamphetamine users had memory impairments, because the hippocampus is known to play a role in some long-term memory; but other brain areas are also involved in processing long-term memory (e.g., overlying temporal neocortex). The size of these other areas was not different between the groups. Finally, the importance for everyday functioning of the brain differences is unclear because an 11 percent difference between individuals, for example, is still most likely within the normal range of human brain structure sizes.

This example is not unique. The brain imaging literature is replete with a general tendency to characterize any brain differences as dysfunction caused by methamphetamine (as well as other drugs), even if differences are within the normal range of human variability (Hart et al. 2012). It would be like comparing the brains of police officers who only completed high school with those of college professors who had obtained a Ph.D. and, then concluding that the officers are cognitively impaired as a result of any differences that might be noted. This simplistic thinking is the main thrust behind the notion that drug addiction is a brain disease. It certainly isn't a brain disease like Parkinson's disease or Alzheimer's disease. In the case of these illnesses, one can look at the brains of affected individuals and make good predictions about the illness involved. We are nowhere near being able to distinguish the brain of a drug addict from that of non-drug addict.

### ***Other Health Concerns***

Methamphetamine abuse has been shown to exacerbate AIDS pathology, including cognitive deficits (Cherner et al. 2005; Carey et al. 2006), and is associated with reduced immunological response to secondary infections such as hepatitis C (Gonzales et al. 2006).

In addition, concerns about methamphetamine users contracting blood-borne illnesses such as HIV and hepatitis C as a result of their drug use have increased interest in determining the extent to which methamphetamines are injected intravenously. Relative to heroin and cocaine, methamphetamine is injected less frequently (Colfax 2010), but injection rates vary widely from region to region. Degenhardt et al. (2010) observed higher methamphetamine injection rates in parts of East and Southeast Asia, North America, northern and central Europe, the former Soviet states, Australia and New Zealand than in Thailand and the United States.

One factor that may influence whether methamphetamine is injected or consumed by other routes is the purity of the drug. If a drug is less pure, containing only 5-10 percent of the desired compound, for example, then, of course, there is less of it to waste. Moreover, taking a drug by mouth can lead to only 10 to 20 percent bioavailability of the ingested dose. Bioavailability is the proportion of the dose of the drug that reaches its target -- in this case, the brain. One reason that a drug's bioavailability is lower following oral administration is because enzymes in the liver specialize in breaking down chemicals, including methamphetamine, in order to protect the brain and to make any poisons we eat less destructive. This process is called *first-pass metabolism*. It can significantly reduce the effective dose of a drug taken orally. First-pass metabolism can be circumvented by injecting or smoking a drug. In general, illicit methamphetamine purity is high (greater than 80 percent: USDOJ, 2011) compared to drugs such as heroin and cocaine. This might be one reason that intravenous use of the drug has been consistently lower than that of these other drugs. Thus, as public health officials consider consequences for various measures taken to limit illicit amphetamine use, they should also be mindful of the fact that their interventions might alter the route by which methamphetamine users take the drug. And a new route might be potentially more dangerous not only to the user, but also to the general public because of blood-borne diseases.

***Treatment with a focus on agonist replacement therapies***

While several behavioral/psychosocial therapies have been demonstrated to reduce illicit amphetamine abuse, a substantial proportion of individuals do not benefit from these interventions (Lee and Rawson 2008).

This suggests the importance of developing pharmacotherapies for those individuals who may be less responsive to behavioral treatments alone. Pharmacotherapies may be used alone, in combination with behavioral/psychosocial therapies, or in a staged manner following inadequate response to behavioral/psychosocial therapies. Most researchers/clinicians recognize that pharmacotherapies alone will not cure a chronic, relapsing disorder such as substance dependence, in part because the problem of substance dependence is expressed behaviorally. Hence, a major goal is that pharmacotherapies will provide a window of opportunity by relieving withdrawal symptoms, for example, so behavioral/psychosocial interventions can be more effectively implemented.

A key argument for the development of effective methamphetamine abuse treatment medications is that they would reduce illicit drug use in HIV-infected individuals in an effort to slow the progression of the disease or reduce the likelihood of HIV-negative individuals engaging in behaviors that might increase the chances of contracting the disease. The underlying theoretical assumption guiding the use of agonist therapies is that maintenance on a pharmacologically similar medication will induce cross-tolerance to the abused drug. Methadone, a long-acting  $\mu$ -opioid agonist for opioid dependence and nicotine replacement medications for tobacco dependence have been used as agonist maintenance treatments to prevent relapse and cravings in individuals attempting to maintain abstinence. Agonist maintenance medications typically use safer routes of administration (e.g., oral or transdermal) and produce diminished psychoactive effects.

In contrast to medication development efforts for opioid and nicotine dependence, in which the neurobiological mechanisms mediating reinforcement are fairly well understood, the neuronal mechanisms of action for amphetamine are more complicated. As stated above, amphetamine increases the activity of monoamine neurotransmitters. Accordingly, it has been reasoned that medications that more closely mimic the actions of the methamphetamine on monoaminergic activity may be useful in treating methamphetamine abuse.

A number of published single-blind studies and case reports indicate that maintenance on oral amphetamines may be helpful in the treatment of methamphetamine abuse (Fleming and Roberts 1994; 1994; Pates et al. 1996; Bradbeer et al. 1998; Shearer et al. 2001). These studies have reported that amphetamine maintenance has many positive outcomes, including reductions in illicit amphetamine use and in injecting as well as improvements in general health. Such programs increase treatment retention and the number of users presenting for healthcare services. Importantly, reported incidence of adverse effects during amphetamine maintenance has been extremely low. However, it is important to note that most data were collected under non-blind conditions, which increase the likelihood of positive results. To address this concern, Longo et al. (2009) completed a randomized, double-blind placebo-controlled trial assessing the effectiveness of sustained-release *d*-amphetamine (average dose 80 mg/d) for treating amphetamine dependence. Positive results were obtained on two of the three outcome measures: the amphetamine maintenance group had significantly better treatment retention and a lower degree of methamphetamine dependence compared with the placebo group. On the third outcome measure (self-reported drug use), there was a trend toward reduced amphetamine use in the amphetamine maintenance group, but it did not reach statistical significance.

The above observations suggest that carefully controlled, double-blind investigations of long-acting oral amphetamine should be expanded. Such therapies may not only be important for curtailing illicit methamphetamine use, but may also be critical for reducing public health risk associated with contraction, progression, and transmission of HIV. It is important to note that prior to initiating such therapies, clinicians should be cognizant of the fact that our knowledge is incomplete and should be prepared to alter their actions as new, more complete knowledge dictates.

***Harm reduction***

As described in this report, we have learned a great deal about the conditions under which either positive or negative effects are more likely to occur with amphetamine use. Low to moderate doses of amphetamine can improve mood, enhance performance, and delay the need for sleep. Repeated administration of large doses of the drug can severely disrupt sleep and lead to psychological disturbances, including paranoia. Unfortunately, this knowledge is rarely disseminated to the public in an unbiased manner, primarily because of the irrational belief that it might lead one to engage in drug use. In light of the fact that there may be as many as 50 million people who use illegal ATS on an annual basis, it seems that a rational approach – one that aims to reduce drug-related harms – would be to share what we have learned with drug users and those in positions to help keep them safe. Otherwise, we may do society a major disservice.

If more amphetamine users were aware of a few simple facts that we have learned, this would substantially enhance public health and safety. First, inexperienced amphetamine users should be discouraged from taking these drugs in the manner in which experienced users do. Experienced users tend to take drugs in ways that get them to the brain quickly, i.e., smoking or intravenous injections. Because smoking and injecting produce more potent effects, the likelihood of harmful consequences is increased with these methods. Alternatively, taking a drug by mouth is usually safer than other ways of consuming drugs for two reasons: 1) the stomach can be pumped in case of an overdose; and 2) some of the drug will be broken down before reaching the brain, resulting in a muted effect.

This is the idea behind an innovative harm reduction program for methamphetamine users in the Czech Republic, which takes its cue from users' own practices (Mravčík et al. 2011). Some people who use methamphetamines in the Czech Republic engage in what they call “parachuting” or swallowing a bolus of pervitin wrapped in a piece of plastic. The main practitioners of this method were people who wanted to inject less or who had trouble injecting because of damaged veins. Service providers in low-threshold facilities are facilitating this process by making available gelatin capsules, which are superior to an improvised plastic wrapper as something to swallow (Mravčík et al. 2011). Czech service providers hope that this method will encourage some people who use methamphetamines to shift from injection to oral use.

Second, healthy sleep habits should be stressed for all drug users because prolonged sleep loss can cause deterioration of mental functioning. In severe cases of sleep deprivation, even without drugs, hallucinations and paranoia may also occur. Because amphetamines reliably reduce fatigue and offset performance decrements, some may repeatedly take these drugs to lessen problems associated with sleep loss. This is a less than ideal approach. One of the most consistent effects of stimulants is the disruption of sleep, which means that repeated use could exacerbate problems related to sleep loss. Given the vital role that sleep plays in healthy functioning, regular users of amphetamine should be mindful of their sleep durations and avoid drug use near the sleep period. In cases of severe sleep disruptions caused by amphetamine, medical healthcare professionals should be consulted to determine whether a sleeping aid is appropriate.

Third, people who inject methamphetamine should be provided access to clean injection equipment. The effectiveness of needle exchange programs for HIV prevention among people who inject other drugs is supported by a large database (Wodak and Cooney 2004). There is little available information on needle exchange programs targeting amphetamine users, despite the fact that UNODC recommends needle and syringe programs as a central element of HIV prevention (UNODC 2012).

Alternatively, for individuals who smoke amphetamine, harm reduction services should include access to clean pipes, plastic tips, filters, lip balm and smoking foil. Presumably, these measures will decrease the likelihood of exchanging bodily fluids and encourage other health promoting behaviors. For example, researchers in Ottawa, Canada, found that cocaine users -- who used the drug via both smoking and injection -- tended to inject less when smoking equipment was readily available; they also engaged in less sharing of potentially contaminated smoking equipment (Leonard et al. 2008).

## **Conclusions and Recommendations for Policy and Research**

Evidence presented in this report suggests that illicit ATS use, primarily methamphetamine use, has become the new crack cocaine with respect to sensational media reporting about the extent of ATS use and hyperbolic description of the disastrous consequences of methamphetamine addiction. It has taken nearly three decades for the public to come to a superficial understanding that the deleterious effects of crack cocaine were greatly exaggerated in mass media and government statements. The monetary and human costs of our earlier misunderstandings about crack cocaine are incalculable. Today, unfortunately, much of the public information about methamphetamine has little foundation in evidence. Overblown worst-case anecdotes are usually disseminated uncritically by the popular press and accepted as sound evidence by an undiscerning public.

Recent implementation of laws and policies aimed at curtailing methamphetamine use in several countries, including the United States, are based on less than accurate assumptions. For example, major themes in anti-methamphetamine advertisements are that the drug is instantly addictive and that use of methamphetamine leads to extreme tooth decay. There is no evidence to support either of these myths. Another common lore is that illicit methamphetamine use leads to neuroanatomical pathologies that cause cognitive impairments. This supposition is also not supported by evidence.

These campaigns have reached the point where they undermine themselves perhaps because methamphetamine users and their acquaintances know that the information is unfounded in all but the rarest cases, as happened for decades with public education materials that suggested that marijuana was a direct route to harmful addiction to hard drugs.

There is an urgent need to revisit policies and information programs related to methamphetamines both nationally and with respect to international norms. In particular:

- Countries that apply harsh criminal sanctions to methamphetamine use and possession should urgently revisit those laws and policies. There is no empirical evidence that suggests that even long-term users of methamphetamines pose a threat to those around them. Nor is there evidence to suggest that methamphetamine users should be incarcerated in order to deal with the “threat” they pose.
- Growing evidence suggests that treatment and harm reduction measures in the health sector may be helpful for some persons who are methamphetamine-dependent, though science and best practices are evolving. Specialized health services and social support informed as much as possible by the best science, rather than criminal sanctions, should be the dominant response to methamphetamine addiction in national policy. Cognitive-behavioral therapies should not be denied to people who need them based on the flawed idea that methamphetamine users are too cognitively compromised to benefit from these interventions.

There is an urgent need for national authorities and donors to invest in development of affordable, accessible and scientifically appropriate health and social services in this area.

- National regulatory authorities and the World Health Organization should restudy the costs and benefits of suppressing access to amphetamine used for legitimate medical purposes in the name of methamphetamine control. Blanket bans on amphetamine for all purposes are unjustified and may have high costs in terms of disease burden in the population. Moreover, these restrictions provide the impetus for the illicit proliferation of “meth labs,” which pose hazards to public safety.
- Precursor control may hold some promise for short-term supply reduction of methamphetamine, but is unlikely to be effective beyond the short run. Like eradication of coca in the Andes, precursor markets made illicit are likely to manifest a “balloon effect” whereby they may be shut down in the short term in one location, but they will pop up quickly in another location. As suggested by the expert Global Commission on Drug Policy (2011) based on evaluation of decades of supply-control efforts in many parts of the world, it is unlikely that illicit precursor markets will be controlled in the long term without some level of state regulation that eschews criminal sanctions.
- National and international institutions and civil society organizations should stop supporting wasteful and ineffective campaigns of misinformation on methamphetamine that use exaggerations and fabricated data as scare tactics. These campaigns are not effective, and their dishonesty renders them counter-productive. There is an urgent need for correcting misinformation on methamphetamine in formats that both policy-makers and the public can understand and use.
- Where injection of methamphetamines is prevalent, access to sterile injection equipment for all who need it must be a policy and program priority. Needle and syringe programs continue to be undermined by paraphernalia laws in many countries and by tolerance of disruption of these services by the police. Denial of the reality of drug injection in prisons and refusal to provide sterile injecting equipment in prisons and other detention facilities undermine HIV and hepatitis control in many countries (Lines et al. 2005). Investment in ensuring access to sterile injection equipment remains a lagging element of national and international HIV programs in spite of decades of evidence demonstrating the effectiveness of these programs.



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