



## Depression and executive dysfunction contribute to a metamemory deficit among individuals with methamphetamine use disorders



K.B. Casaletto<sup>a</sup>, L. Obermeit<sup>a</sup>, E.E. Morgan<sup>b</sup>, E. Weber<sup>a</sup>, D.R. Franklin<sup>b</sup>, I. Grant<sup>b</sup>, S.P. Woods<sup>b,\*</sup>,  
The Translational Methamphetamine AIDS Research Center (TMARC) Group

<sup>a</sup> SDSU/UCSD Joint Doctoral Program in Clinical Psychology, San Diego, CA, USA

<sup>b</sup> Department of Psychiatry, University of California, San Diego, La Jolla, CA, USA

### HIGHLIGHTS

- Compared awareness of memory deficits (metamemory) in methamphetamine (MA) users vs. non-users
- MA users were more impaired, but less aware of memory impairments than non-users
- MA and depression interacted such that MA users with depression had the most inaccurate metamemory
- Poor metamemory was associated with executive dysfunction and lower cognitive reserve in MA users

### ARTICLE INFO

Available online 28 August 2014

#### Keywords:

Neuropsychology  
Memory  
Drug/substance abuse

### ABSTRACT

**Objective:** Chronic methamphetamine (MA) use is associated with moderate deficits in learning and memory, but the extent to which MA users are aware of such memory deficits (i.e., metamemory) is not known. **Methods:** In the current study, 195 participants with lifetime MA use diagnoses (MA+) and 195 non-MA-using comparison subjects (MA-) underwent comprehensive neuropsychiatry research assessments, including performance-based and self-report measures of episodic memory. **Results:** MA use disorders, major depressive disorder (MDD), and their interaction were uniquely associated with metamemory functioning, such that MDD increased the likelihood of a metamemory deficit among MA+ participants. Within the MA group, individuals who overestimated their memory abilities demonstrated greater executive dysfunction and lower cognitive reserve. **Conclusions:** Chronic MA use is associated with reduced awareness of objective deficits in memory acquisition and recall, which is particularly exacerbated by the presence of major depression. Efforts to enhance metamemory accuracy and deployment of compensatory mnemonic strategies may benefit substance abuse treatment outcomes.

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### 1. Introduction

Methamphetamine (MA) has a preferential neurotoxic effect on the frontostriatal systems (Earnst, Chang, Leonido-Yee, et al., 2000) that contributes to both emotion dysregulation (London, Simon, Berman, et al., 2004) and neurocognitive impairment (Scott, Woods, Matt, et al., 2007). MA-related neurocognitive deficits most commonly include episodic memory and executive functions (Woods, Rippeth, Conover, et al., 2005) and are associated with poorer functioning in daily activities (Henry, Minassian, & Perry, 2010), including unemployment (Weber, Blackstone, Iudicello, et al., 2012). One mechanism by which such MA-related neurocognitive deficits may impact daily

functioning is via poor awareness of the nature and extent of one's impairment. For example, a MA dependent individual who is unaware of a memory deficit would be much less likely to use a compensatory strategy during daily tasks (e.g., using a calendar or alarm to help them remember to take a medication), and is therefore more vulnerable to experience critical memory failures in real life (e.g., medication nonadherence).

According to Nelson and Narens (1990), awareness of memory abilities (i.e., metamemory) may be disrupted at several time points during the acquisition, retention, and/or retrieval of new information. Specifically, the overall correspondence between an individual's perceived memory abilities and his actual memory capacity is postulated to be influenced by how well the material has been learned (e.g., do I need to continue studying?), initiation and termination of recall search strategies, and recall selection choices (e.g., how confident am I that this is the memory I am searching for?); all of which require internal self-

\* Corresponding author at: University of California, San Diego (UCSD), HIV Neurobehavioral Research Program, 220 Dickinson Street, Suite B, San Diego, CA 92103-8231, USA. Tel.: +1 619 543 5004; fax: +1 619 543 1235.

regulation. Such processes utilize prefrontal systems, and the combination of executive and memory dysfunction appears to confer greater metamemory inaccuracy (Pannu & Kaszniak, 2005). These same systems are commonly disrupted among MA users suggesting a potential vulnerability to metamemory dysfunction in this population (Ersche, Williams, Robbins, et al., 2013). Yet only two studies to date have examined metacognition in chronic MA users; Cattie, Woods, Iudicello, et al. (2012) found that self-reported symptoms of executive dysfunction in daily life were not related to objective laboratory measures of executive dysfunction among individuals with MA dependence, while Kirkpatrick, Metcalfe, Greene, et al. (2008) illustrated that increasing doses of intranasal methamphetamine administration among MA users disrupted accuracy of metacognitive judgments.

Given the prefrontal and striatal predilection of MA-associated neural disruption, it is not surprising that mood dysregulation, such as major depression, is a highly comorbid (Conway, Compton, Stinson, et al., 2006) and functionally impactful condition among chronic MA users (Glasner-Edwards, Marinelli-Casey, Hillhouse, et al., 2009). As postulated in the Nelson and Narens model of metamemory (1990), an individual's belief regarding the difficulty level of the information to be learned (i.e., self-efficacy) in combination with his motivation to learn directly influences his memory behaviors (e.g., to study the information or not). Given that depressive symptoms directly impact self-efficacy and motivation and are related to increased prevalence of memory symptoms in the general population (Ponds & Jolles, 1996), major depressive disorder (MDD) may moderate metamemory accuracy by lowering self-perceived memory abilities and motivation to learn. Therefore, it may not be surprising that memory self-efficacy is consistently a stronger indicator of memory complaints than actual memory test performance (Dellefield & McDougall, 1996; Ponds & Jolles, 1996). In fact, depression has been consistently associated with inaccurate under-estimation of actual memory abilities in the general population (Kalska, Punamaki, Makinen-Pelli, et al., 1999). For example, depression demonstrated a stronger relationship with reported memory symptoms than memory test performance among a cohort of older healthy adults (Bolla, Lindgren, Bonaccorsy, & Bleecker, 1991), and increasing levels of depression are consistently associated with greater number of memory symptoms among both younger and older adults, regardless of actual memory capacities (Bolla et al., 1991; Kalska et al., 1999; Niederehe & Yoder, 1989).

Considering the frontostriatal systems that are disrupted in MA use and depression and the role of such systems in metamemory, MA + individuals may be particularly susceptible to inaccurate perceptions of their memory abilities; yet, no studies to date have examined this construct among substance users. Therefore, we aim to determine the independent and additive impact of MA use and MDD on metamemory, as well as explore other factors that may affect metamemory processes within the MA + cohort (e.g., executive dysfunction).

## 2. Materials and methods

### 2.1. Participants

The sample was composed of 390 participants who participated in National Institute of Drug Abuse (NIDA) funded research studies conducted from 1999 to 2012 that were approved by the University of California, San Diego's Human Research Protections Program. All participants provided written informed consent prior to study participation. MA use disorder diagnoses were determined via the Composite International Diagnostic Interview Version 2.1 (CIDI; World Health Organization, 1997) or the Structured Clinical Interview for the DSM-IV-TR (SCID; First, Spitzer, & Gibbon, 1991), which follow the Diagnostic and Statistical Manual version IV-text revised (DSM-IV-TR; American Psychiatric Association, 2000) criteria; participants who met criteria for lifetime MA dependence and MA abuse or dependence within the past 18 months were included in the MA + group ( $n = 195$ ). One hundred ninety five participants who did not meet DSM-IV-TR criteria for MA

abuse or dependence currently or in the past, and also did not meet DSM-IV-TR criteria for other drug or alcohol abuse or dependence within the past 18 months were included as a MA— comparison sample.

For both groups, participants were not enrolled in the study if they presented with a positive urine toxicology result for any illicit substances (excluding marijuana) on the day of testing, or if they qualified for dependence of alcohol or other drugs (excluding marijuana and prescribed medications) within the past year. Specific to marijuana, participants were excluded if they reported use the morning before testing (i.e., acutely intoxicated). Additionally, participants were excluded if they had histories of primary psychotic disorders (e.g., schizophrenia; substance-induced psychosis was allowed), severe medical problems (e.g., seizures, TBI), or if they scored <70 on the Wide Range Achievement Test—reading subtest (WRAT-3 Reading; Wilkinson, 1993).

### 2.2. Psychiatric assessment

Mood disorders and other substance use diagnoses (e.g., alcohol) were determined based on the CIDI or the SCID. Participants were classified as having lifetime Major Depressive Disorder (MDD) if they ever met DSM-IV-TR criteria for MDD (including currently). A semi-structured timeline follow-back interview (Rippeth, Heaton, Carey, et al., 2004) was used to determine MA use characteristics (e.g., frequency, quantity).

### 2.3. Neuromedical evaluation

All participants completed a medical evaluation including a standard medical history interview, structured neurological and medical examination, and laboratory testing of blood and urine samples (Heaton, Franklin, Ellis, et al., 2011).

### 2.4. Neurobehavioral assessment

A comprehensive neuropsychological battery, designed to capture the primary cognitive domains affected by MA dependence (Scott et al., 2007), was administered to all participants. This battery included measures of episodic memory, executive functions, psychomotor speed, attention/working memory, verbal fluency, and motor functioning (Rippeth et al., 2004).

#### 2.4.1. Episodic memory assessment

Learning and memory were specifically evaluated using the Brief Visuospatial Memory Test (BVMT-R; Benedict, 1997) and Hopkins Verbal Learning Test (HVLN-R; Benedict, Schretlen, Groninger, et al., 1998). Learning (i.e., trials 1–3) and delayed recall scores on each measure were calculated and converted into scaled scores ( $M = 10$ ,  $SD = 3$ ) and averaged across the two measures to create summary learning and delayed recall scores. Participants were classified as “memory impaired” if they received a scaled score of <7 on learning and/or delayed recall. Given that the primary aim of this study was to determine accuracy of one's memory appraisal (i.e., one's actual memory abilities in everyday life), we used demographically-uncorrected scaled scores in order to best represent absolute memory functioning (versus memory functioning expected for one's age, education, gender, and ethnicity). However, group differences in demographic (i.e., education, ethnicity, gender) and neuropsychiatric functioning (i.e., WRAT-3 reading, lifetime MDD, lifetime alcohol or other substance use disorders) were covaried in all between-group analyses.

#### 2.4.2. Perceived memory functioning

Perceived memory ability in everyday life was assessed using the memory domain (first 10 items) of the Patient's Assessment of Own Functioning (PAOFI; Chelune, Heaton, & Lehman, 1986). On the PAOFI, each item (e.g., “How often do you forget people whom you met in the last day or two?”) is rated from 1 (“almost always”) to 6

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