An Investigation of Aggression in Methamphetamine Users

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Abstract

Methamphetamine (MA) use is associated with increased aggression, though the specific mechanisms through which this association operates remain unclear. Research has demonstrated an association between MA, heightened impulsivity and reduced behavioural control. Similarly, there is strong evidence of a link between MA use and psychotic symptoms. Despite research efforts examining these independent associations, there is a paucity of studies examining how these factors interact to influence the relationship between MA use and aggression. Study 1 was conducted to examine the independent and combined roles of impulsivity and positive psychotic symptoms on the relationship between MA and aggression. In this study, 237 injecting MA users completed a range of self-report measures of hostility, impulsivity, and dependence. Higher levels of MA dependence were associated with increased hostility, higher levels of impulsivity and greater positive psychotic symptoms. Furthermore, the relationship between MA use and aggression was mediated by both impulsivity and positive symptoms of psychosis. Synergistic effects of impulsivity and positive psychotic symptoms on hostility were also observed, with substantially higher levels of hostility being associated with the presence of positive symptoms in conjunction with heightened impulsivity. The results of Study 1 are important in increasing our current understanding of the relationship between MA dependence and aggression, but the reliance on self-report measures does present a problem because of the susceptibility of such measures to report biases. Study 2 was therefore designed to extend previous research by exploring the relationship between MA and aggression using a behavioural measure of aggression, as well as self-report measures. Behavioural and self-report measures of impulsivity were also included. Results revealed that MA users, compared with non-MA users, not only reported higher levels of aggression, but they actually
behaved more aggressively when competing with a (fictitious) opponent. Furthermore, some evidence of a dose-response relationship between MA and aggression was apparent. Unexpectedly, no group differences were evident on either the behavioural or self-report measures of impulsivity. Taken together, the present research attests to the robustness of the relationship between MA use and aggression, and points to the need for increased understanding of the complex and multifaceted nature of factors such as impulsivity and positive psychotic symptoms that contribute to this relationship.
Statement of Originality

This work has not previously been submitted for a degree or diploma in any university. To the best of my knowledge and belief, the thesis contains no material previously published or written by another person except where due reference is made in the thesis itself. Data collected for Study 1 was part of a larger project and assistance was provided by a team of research assistants. Data collected for Study 2 was undertaken by the author alone.

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Kely Lapworth

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<td>AQ</td>
<td>Aggression Questionnaire</td>
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<tr>
<td>ATODS</td>
<td>Alcohol Tobacco and Other Drugs Service</td>
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<tr>
<td>BIS-11</td>
<td>Barrett Impulsiveness Scale – Version 11</td>
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<tr>
<td>BPRS</td>
<td>Brief Psychiatric Rating Scale</td>
</tr>
<tr>
<td>CSF</td>
<td>Cerebrospinal fluid</td>
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<tr>
<td>DSM-IV-TR</td>
<td>Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (Text Revision)</td>
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<tr>
<td>fMRI</td>
<td>functional Magnetic Resonance Imaging</td>
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<tr>
<td>MA</td>
<td>Methamphetamine</td>
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<tr>
<td>Mg/kg</td>
<td>milligrams per kilogram</td>
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<tr>
<td>O-LIFE</td>
<td>Oxford Liverpool Inventory of Feelings and Experiences</td>
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<tr>
<td>PRISM</td>
<td>Psychiatric Research Interview for Substance and Mental Disorders</td>
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<tr>
<td>SCID-IV</td>
<td>Structured Clinical Interview for the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition</td>
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<tr>
<td>SDS</td>
<td>Severity of Dependence Scale</td>
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<td>SSRT</td>
<td>Stop signal reaction time</td>
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<tr>
<td>STOP-IT</td>
<td>Stop Signal Task</td>
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<tr>
<td>TAP</td>
<td>The Taylor Aggression Paradigm</td>
</tr>
<tr>
<td>TLFB</td>
<td>Timeline Follow Back Method</td>
</tr>
<tr>
<td>TPH</td>
<td>Tryptophan hydroxylase</td>
</tr>
<tr>
<td>5-HIAA</td>
<td>5-hydroxyindoleacetic acid</td>
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<tr>
<td>5-HT</td>
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CHAPTER 1

Overview and Aims of the Thesis

There has been a significant increase in the use of methamphetamine (MA) across the Western world in recent years (Favrod-Coune & Broers, 2010). This has been particularly evident in Australia which has seen a steady increase in the use of MA with more than 1 million (6.3%) Australians reporting lifetime use (Australian Institute of Health and Welfare, 2008). Similar increases have been seen in other countries including the United States, which in turn, has resulted in a growing number of people admitted to treatment programs with a 255% increase in individuals presenting for MA related treatment during the period of 1997 to 2007 (U.S. Department of Health and Human Services Substance Abuse and Mental Health Services Administration, 2003).

The consequences of MA use are extensive and include involvement in criminal activities, adverse physical effects of chronic use and profound, and perhaps enduring, psychological effects (Salo et al., 2010). One particular consequence, aggressive and hostile behaviour, is a primary concern, and forms the focus of this thesis.

There is increasing evidence from cross-sectional studies that many MA users report high rates of aggressive and hostile outbursts, elevated levels of violent and aggressive behaviours and great difficulty controlling their anger (Cohen et al., 2003; Darke, Torok, Kaye, Ross, & McKetin, 2010; Hall, Hando, Darke, & Ross, 1996; Sekine et al., 2006; Vincent, Schoobridge, Ask, Allsop, & Ali, 1998; Wright & Klee, 2001; Zweiben et al., 2004). This increase in aggression is evident in current MA users and also in abstinent users who were previously MA dependent (Sekine et al., 2006). Research from animal and human laboratory studies has provided further support for an association between MA and aggression, using more methodologically rigorous research designs (Payer, Lieberman, & London, 2011; Sekine et al., 2006).
Despite the growing body of evidence demonstrating a link between MA and aggression, the factors that may contribute to the expression of aggression in MA users and influence the strength of the relationship are still relatively unknown. Nonetheless, what is clear is that MA use is also associated with difficulties relating to impulse control and behavioural inhibition and to the experience of subclinical and clinical psychotic symptoms. Both of these factors have been linked to the propensity to act aggressively across a range of substances including MA.

In relation to the first of these factors, the association between stimulant use and impaired inhibitory control is now well established. MA users demonstrate impairment in the inhibition of behavioural responses (Fillmore & Rush, 2002; Li, Milivojevic, Kemp, Kwangik, & Sinha, 2006; Monterosso, Aron, Cordova, Xu, & London, 2005; Salo et al., 2002; Simon et al., 2000) and an increase in risk taking behaviours compared to non-drug using individuals (Leland & Paulus, 2005). Further support comes from evidence of alterations in brain function in areas of the frontal and prefrontal cortex, known to be involved in the regulation of behaviour (Leland, Arce, Feinstein, & Paulus, 2006; Paulus, Lovero, Wittmann, & Leland, 2008). Finally, a number of studies have found elevated scores in MA users on self-report measures designed to tap constructs related to behavioural control or impulsivity (Coffey, Carlin, Lynskey, Li, & Patton, 2003; Moeller et al., 2004; Semple, Patterson, & Rant, 2005).

MA use is also clearly linked to subclinical and clinical psychotic symptoms (e.g., Mahoney, Kalechstein, De La Garza, & Newton, 2008) which can occur during acute MA intoxication or withdrawal. Typically such symptoms relate to persecutory delusions and when expressed as full psychotic symptoms, resemble paranoid schizophrenia (Iwanami et al., 1994). Higher rates of psychotic symptoms, particularly positive psychotic symptoms, are noted to occur in MA users compared to non-using
individuals (D. Harris & Batki, 2000; Leamon et al., 2010; McKetin, McLaren, Lubman, & Hides, 2006; Nakama et al., 2008; Srisurapanont et al., 2003). Further, this effect remains constant, even after taking into account history of psychotic disorders (McKetin, McLaren, Lubman, et al., 2006). For most MA users, the psychotic symptoms tend to be transient in nature and typically occur during heavy binge use periods or throughout the withdrawal period. Nevertheless, there is a smaller subset of individuals who continue to experience recurring symptoms for months and years after cessation of MA use (Chen et al., 2003; 2005).

Hospital morbidity data highlights an alarming rise in the number of psychotic disorders due to stimulant use. For instance, based on hospital admissions in Australia, there were 200 cases of stimulant-induced psychosis reported during 1998 to 1999, compared with 1028 cases in the year of 1999 to 2000. Higher rates have continued to be observed with 1510 cases reported during 2004 to 2005 and 1636 reported cases during the year of 2006 to 2007 (AIHW, 2008). Such data shows a clear increase in the incidence of stimulant-induced psychosis over the past decade.

Importantly, studies have reported higher rates of aggression and violence in psychotic populations compared to those with other psychiatric illnesses (Foley et al., 2005; Pearson, Wilmot, & Padi, 1986; Tardiff, Marzuk, Leon, & Portera, 1997). In early psychosis patients, the reported incidence rates of violence and aggression have ranged from 29% to 75% (Foley et al., 2007; Steinert, Wiebe, & Gebhardt, 1999) depending on definitions used. Further, these studies have identified substance use as a significant predictor of aggressive behaviour (Foley et al., 2005). Thus, individuals who use MA and experience psychotic symptoms have a greater risk of behaving aggressively.
Overall, there is a paucity of studies examining the potential mechanisms through which MA use operates to facilitate aggression in some users. Even less attention has been paid to how two of these possible mechanisms – impulsivity and positive psychotic symptoms – might interact to influence the strength of the relationship between MA use and aggression.

Another shortcoming in this area of research is that aggressive and hostile behaviour has often been measured by simply asking respondents whether they have felt or acted in an aggressive or hostile manner, as opposed to using standardised measures of aggression or hostility, with known reliability and validity. Of those studies that have employed standardised measures, very few have attempted to disentangle whether the aggression preceded initial MA use or could be viewed as a consequence of such use. Clearly, it is possible that for many MA users, pre-existing characteristics, including increased levels of impulsivity, may play an etiological role in substance abuse as well as being exacerbated by subsequent MA use (Hoaken & Stewart, 2003).

Finally, while there have been a number of studies using behavioural measures of aggression in populations of substance users, including alcohol (Giancola et al., 2009; Godlaski & Giancola, 2009), heroin (Gerra et al., 2007) and ecstasy (Gerra et al., 2001), with the exception of one recent study, there has been little behavioural investigation of aggression in MA users specifically (Payer et al., 2011; Sekine et al., 2006).

The current thesis aimed to extend previous work by investigating the links between MA use, behavioural disinhibition, psychotic symptoms and aggression. In the first study the influence of MA dependence, impulsivity, and positive psychotic symptoms on aggressive and hostile behaviour were investigated in a sample of MA users ($n = 237$), using valid and reliable self-report measures. Study 1 further examined
the interacting effects of impulsivity and positive symptoms of psychosis on the association between MA dependence and aggression.

The second study aimed to replicate and extend Study 1 by examining the relationship between MA use and impulsivity and aggression by utilising both behavioural and self-report measures of the latter two constructs. Study 2 employed a between-subjects design in which the responses of MA users ($n = 21$) were compared with a carefully selected group of non-MA users ($n = 21$) who did not differ on important demographic variables including age, sex, education, and general intelligence. Study 2 thus sought to clarify whether there were differences in the behavioural expression of impulsive behaviour and aggressive behaviour in MA users and non-MA users on both self-report and laboratory measures of aggression and impulsivity. Because of difficulties in measuring psychotic symptoms using behavioural measures, the influence of psychotic symptoms on the MA-aggression relationship was not examined in Study 2.
CHAPTER 2

The Relationship Between Aggression and Methamphetamine Use

Multiple sources have documented that repeated use of MA affects many aspects of psychological functioning (for reviews see Darke, Kaye, McKetin, & Duflou, 2008; Marshall & Werb, 2010; Maxwell, 2005; Shrem & Halkitis, 2008). These include heightened rates of aggression, psychosis, mood and anxiety disorders, and cognitive deficits. It would appear that up to 25% of those with regular use report severe and debilitating mental health issues (McKetin, McLaren, & Kelly, 2005; Zweben et al., 2004).

While the use of various substances has been linked to violent and aggressive behaviour, there is increasing evidence of a particularly strong link between MA use and aggression, hostility and violence. Studies which have employed a range of self-report measures have found that a large proportion of MA users reported a high incidence of aggressive outbursts, elevated levels of violent and aggressive behaviours and great difficulty controlling their anger (Cohen et al., 2003; Darke et al., 2010; Hall et al., 1996; McKetin, McLaren, Riddell, & Robbins, 2006; Sekine et al., 2006; Vincent et al., 1998; Wright & Klee, 2001; Zweben et al., 2004). For instance, Sommers, Baskin, and Baskin-Sommers (2006) found that 35% of MA users admitted to behaving aggressively or violently while intoxicated. Even higher rates of aggression and violence were reported by Brecht, O’Brien, von Mayrhofer, and Anglin (2004) and Zweben et al. (2004) who found that 57% and 43% of MA users respectively, reported high rates of aggression.

An English study examined links between violent crime, aggression, and MA use in a group of 86 users (Wright & Klee, 2001). Almost half (47%) of the heavy MA users reported committing a violent crime, defined as the infliction of physical harm on
another. Users commonly identified MA intoxication (34%), provocation (26%), and the experience of paranoid delusions (17%) as precipitants of violence and aggression. In this study, aggression was defined as hostile or destructive behaviour that did not involve physical harm to another. Although limited to self-report, a quarter of participants believed that their MA use and violent crime were directly linked, and they acknowledged the withdrawal period as being a high risk time for committing aggressive and violent acts. Almost two-thirds (62%) of MA users reported ongoing problems with aggression that they believed were associated with their MA use (Wright & Klee, 2001).

A similar investigation was undertaken by Sommers and Baskin (2006) who examined the incidence of violence in a sample of 205 MA users. Participants were interviewed at length about specific violent or aggressive events and the context in which they occurred, using a structured, open-ended technique. Similar to the study above, violence consisted of “any form of deliberate physical harm inflicted on another individual” (Sommers & Baskin, 2006, p. 83). A quarter (27%) of participants reported an act of violence while intoxicated by MA. The majority (65.5%) of those reporting violence were male and half (51.4%) of the cases of violence involved domestic relationships. Predictors of violence were exposure to family of origin deviance (e.g., child abuse), a prior history of violence in the context of substance use, poor social functioning, initial age of MA use, and the expression of aggression during childhood.

Other research has shown that a high incidence of MA and aggression is also evident in very young MA users. For example, a recent study which examined the prevalence of MA use and aggression in a group of South African adolescents found significantly higher aggressive behaviour scores among those who used MA relative to their non-drug using counterparts (Pluddemann, Flisher, McKetin, Parry, & Lombard,
In order to confirm the significance of this finding and to estimate the size of the effect after adjusting for potential confounders, an ordinal regression was performed. Using the measure of aggressive behaviour as the outcome variable, and adjusting for cannabis, alcohol and tobacco use in the past 12 months, the researchers found that MA use almost doubled the risk of aggression. Interestingly, research has shown that high rates of aggression are not only evident in current MA users, but are also noted in abstinent users who were previously MA dependent (Sekine et al., 2006). Using an unpaired t-test, results revealed significantly higher mean aggression scores on a self-report measure of aggression among a group of abstinent MA abusers compared with sex, age and education matched controls.

Anecdotal information collected from health workers also attests to the notion of a strong relationship between MA use and aggression. For instance, emergency staff at hospitals report that MA intoxicated individuals frequently present as violent, agitated and aggressive and often require police intervention (Bunting, Fulde, & Forster, 2007). Further, there was consensus among some workers in the field that MA users have actually become more aggressive over time (Bunting et al., 2007), which possibly coincides with more potent forms of the drug being made available. Topp, Degenhardt, Kaye, and Darke (2002) conducted semi-structured interviews (based on the World Health Organisation questionnaire used to assess cocaine trends in Sydney) with 229 ‘key informants’ between 1998 and 2001. The key informants consisted of general health workers, needle syringe program workers, police and outreach workers who had maintained contact with a minimum of 10 different MA users and/or at least weekly contact with MA users during the past 6 months. Information was collected from key informants in relation to commonly reported side effects from MA users. There was consensus among all key informants that those MA users who had injected or smoked
more potent forms of the drug (e.g., crystal MA and base), generally experienced
greater levels of psychological disturbance than users accessing less potent forms
(powder). Further, these users were consistently described as more chaotic, aggressive,
agitated, paranoid, and difficult to work with than users of MA powder.

While there is increasing agreement among researchers of an association
between MA and aggression, much of the evidence relies on anecdotal reports and/or is
derived and generalised from qualitative studies that focus primarily on violence
committed by MA users (Butler, Wheeler, & Sheridan, 2010; Darke et al., 2010;
McKetin, McLaren, Riddell, et al., 2006). In contrast to using valid and reliable
standardised measures of aggression or violence, these studies have typically used
structured interviews to ask MA users about whether they have acted in an aggressive or
violent manner. Further, there does not appear to be a clear and consensual definition of
aggression or violence among studies, which has resulted in these constructs being
measured in different ways. While there was consistency across two studies in their
definition of violence (Sommers & Baskin, 2006; Wright & Klee, 2001), only one of
these studies went on to define aggression (Wright & Klee, 2001). Further, the
definition of aggression in this study, as hostile or destructive behaviour that did not
involve physical harm to another, clearly contrasts with other studies which refer to
violence and aggression more broadly and interchangeably. For example, Hamilton and
Goeders (2010, p. 314) adopted a broader definition of violence as a “complex,
multifaceted, and dynamic aspect of human interaction that occurs in multiple forms
and patterns.”

Boles and Miotto (2003) on the other hand, referred to three types of violence.
First, psychopharmalogical violence was defined as violence which occurs as a direct
result of the neurotoxic effects MA has on altering cognitions and behaviour which may
cause an individual to perpetrate violence. Second, systemic violence was defined as acts of interpersonal aggression which occur among individuals involved in the drug scene. Third, economic compulsive violence was defined as intentional acts of violence and criminal activity performed to obtain resources to support MA use. Other studies investigating the link between MA use and violence have measured the number of violent acts committed in a specified period, such as the past 30 days. Respondents were asked about how often they had engaged in a range of violent behaviours, including assaulting someone or robbing someone to obtain cash, goods or drugs, and threatening someone, with or without a weapon (Butler et al., 2010; Darke et al., 2010; McKetin, McLaren, Riddell, et al., 2006; Torok, Darke, Kaye, Ross, & McKetin, 2008).

Further, few studies have dissected rates of aggression prior to MA use with aggressive acts reported following initial MA use. For instance, Zweben et al. (2004) examined co-occurring psychiatric symptoms in a sample of 1016 MA outpatients. Measures of violent behaviour, including self-reports of legal charges and difficulty controlling violent behaviour or anger, were collected using items from the Addiction Severity Index (ASI; McLellan et al., 1992). Forty-three percent of MA users reported that they had experienced problems controlling their violent behaviour throughout their lifetime. While this finding would seem to support an association between MA use and aggression, the study did not specify whether these problems preceded or followed the use of MA.

The Temporal Relationship between Methamphetamine Use and Aggression

Some studies have attempted to tease out the temporal relationship between MA use and aggression. Hall et al. (1996) found that while there was a positive association between reported acts of violence prior to any MA use and incidents following initial MA use, individuals still reported greater occurrences of aggressive acts following first
use of MA compared with when they had not used MA at all (39% versus 44%). In another study, Sommers et al. (2006) expanded on their earlier work by collecting information from MA users about acts of violence committed before they began using MA and after they had initiated use. Structured, open-ended interviews were used to collect a narrative account of how MA use was related to violent events. Of 106 participants, 38% of males and 30% of females reported committing acts of violence while under the influence of MA. Among those who had committed violence, 46% reported that they had not committed a violent act prior to MA use. Of the 54 aggressive acts reported by MA users, 62% involved domestic violence, 17% were related to drug disputes, 13% were gang related and a further 9% were random acts of aggression such as road rage and assault on strangers.

In this same study, additional information was collected regarding acts of violent crime committed throughout lifetime. Results showed that 37% of participants had committed assault, 16% had undertaken robbery, 54% carried weapons, 16% had been charged with attempted murder, and 7% had been charged with murder (Sommers et al., 2006). The authors concluded that MA use increased the risk of violence and aggression by converting day-to-day interactions into challenges of social control and retribution. MA use resulted in an exaggerated and often aggressive response to personal violations, such as perceived lack of respect, invasion of personal space and difference of opinion or verbal challenges. Further, MA use commonly resulted in paranoia (62%) and a perceived threat of danger from others. This, in turn, led to defensive responses which commonly involved aggression. MA users described a loss of control over their behaviour and spoke of experiencing outbursts of anger and rage. Overall, the authors concluded that MA use decreased behavioural control, increased
level of arousal and intensity of emotions, and distorted perceptions of everyday interactions (Sommers et al., 2006).

Another study that attempted to explore pre-existing rates of aggression, rates of aggression following MA use and typical patterns of aggression was recently undertaken by Hamilton and Goeders (2010). This study focused solely on MA-related aggression and violence among a group of 30 female users receiving treatment for MA dependence. Fifty-seven percent of female users reported behaving violently or aggressively towards others. This behaviour typically occurred in the context of ‘coming down’ or withdrawing from the drug. Further, 29% of the sample attributed their aggression directly to MA, while 59% acknowledged they had existing anger issues that were exacerbated by their use of MA (Hamilton & Goeders, 2010).

In a study focusing specifically on violent crime (murder, manslaughter, robbery, and assault), Cartier, Farabee, and Prendergast (2006) compared recurrent acts of violence and other criminal offences in 202 prison inmates with a history of MA use with 202 inmates who had no history of MA use. The follow up period was one year post release. During this time, MA users were more likely to commit a violent crime and also return to custody for other offences than non-MA users. Notably however, despite the fact MA users reported engaging in more violent crime following their release from prison, they were not more likely than non-MA users to return to custody for this reason.

Recently, Torok et al. (2008) sought to examine the prevalence and types of violent crime committed by a large sample (n = 118) of MA users in Australia. Using a structured interview, information was collected regarding participants’ frequency of involvement in various types of crime, including property crime, fraud and violent crime in the preceding month, as well as history of arrests for any violent offences.
Eighty-one percent of MA users admitted to engaging in violent crime throughout their lifetime, compared with 41% in the past year. Further, approximately half of all MA users had faced criminal charges for committing a violent crime, including 44% for physical assault, 14% for aggravated assault and 24% for threatening behaviour. Three quarters of MA users reported behaving violently on more than a single occasion, with violent users reporting an average of eight offences (Torok et al., 2008).

The same researchers then went on to compare rates and types of violent crime committed by MA users with a sample of heroin users and a sample that used both MA and heroin regularly. They found that the MA group had engaged in significantly more violent crime in the past year than heroin users or participants in the heroin/MA group. Results further demonstrated that participants who reported the heaviest MA use had committed the greatest number of violent acts (Torok et al., 2008). Interestingly, gender was not found to be associated with increased risk of violent offending. This is surprising given the large body of literature with non-drug abusing populations which typically reports higher rates of violence among men (Karer & Langan, 2001; Kellerman & Mercy, 1992; Neale, Bloor, & Weir, 2005). The authors explained the absence of gender differences by suggesting that involvement in the illicit drug market as a result of using MA, coupled with situational factors that accompany a drug dependent lifestyle, led to a universal and increased risk of being involved with violence (Torok et al., 2008).

**Severity of Psychological Effects**

One factor that appears to influence the severity of psychological effects, including violent and aggressive behaviour, is the form and purity of the drug. Compared with amphetamine, methamphetamine has stronger and longer lasting subjective effects (Degenhardt & Topp, 2003). It can be used in multiple ways
including snorting, smoking, injecting or swallowing. Methamphetamine powder or speed has relatively low purity and is generally snorted or injected. Pills can represent pharmaceutical grade stimulants (e.g., Dexamphetamine), or methamphetamine powder that has been compacted into tablets (e.g., ‘Speed tabs’ or ecstasy). The base (a sticky, gluggy, oily form of damp paste) and crystalline (yellowish/brownish crystal-like substance) forms of methamphetamine have even greater purity. Clear crystal methamphetamine, commonly referred to as Ice, tends to be of the highest purity, is generally imported from South-East Asia and is readily available in Australia (Topp & Churchill, 2002).

Indeed, research has shown that the use of more potent forms of methamphetamine, such as base and crystal meth, tends to be associated with more rapid deterioration in psychological and physical deterioration than less pure forms, such as powdered methamphetamine (Degenhardt & Topp, 2003). A study of 45 crystal methamphetamine (i.e., ice) users found that 91% of the sample perceived at least one psychological side effect related to their use, with an average number of 4 effects reported (Degenhardt & Topp, 2003). The most commonly reported symptoms were anxiety (70%), paranoia (64%), depression (62%), irritability (62%) and violent behaviour (24%). The researchers concluded that compared with a sample of longer-term, heavier, and predominantly injecting amphetamine users, crystal methamphetamine users appeared more likely to experience significant psychological effects at a much more recent and lower level of use. Thus, emerging evidence suggests that severity of psychological effects experienced by methamphetamine users might be related to the form and potency of the drug.”
Is Methamphetamine Use Linked to Aggression: Evidence from Animal Laboratory Studies

Animal laboratory research provides the opportunity to investigate the relationship between MA administration and aggression in highly controlled environments. Over the past three decades, several laboratory-based animal studies have examined the effects of acute MA administration on aggressive behaviour, with some studies finding a positive association (Miczek & Tidey, 1989; Mori, Ito, Kita, & Sawaguchi, 2004; Shintomi, 1975; Sokolov & Cadet, 2006; Sokolov, Schindler, & Cadet, 2004), while others found no evidence of a relationship (Miczek & O’Donnell, 1978) and at least one study found mixed results (Crowley, 1972). An early, rigorous study found strong evidence of a relationship between MA and aggression when comparing fighting in mice injected with MA to non-drug exposed mice (Shintomi, 1975). Seven male mice received three injections – (i) no MA, (ii) a single dose of MA (5 milligrams per kilogram of body weight [mg/kg]), (iii) a second dose of MA (5mg/kg). The mice were placed together in a confined space and fighting behaviours were observed and rated across 30 minute intervals, over a two hour period, on a total of four occasions. A dose-dependent effect was demonstrated where an absence of fighting behaviour was observed at baseline, compared with slight fighting and hyperactive behaviours (running and jumping) following the first dose, and significant fighting and hyperactive behaviours following the second dose. Further, mice exposed to MA demonstrated significantly higher rates of aggressive behaviour compared to control mice (Shintomi, 1975).

In another early study, Crowley (1972) observed a dose-dependent increase in fighting time in rats following acute administration of MA, but only at concentration levels of up to 1 mg/kg. Groups of rats were given doses of 0, 0.25, 0.5, 1.0, 2.0 or 4.0
mg/kg of MA. Fighting behaviour, time spent fighting and motor activity was measured when pairs of rats were placed on an electric shock grid and received a series of shocks. Interestingly, MA was found to increase fighting behaviour in rats in a dose-dependent manner up to 1 mg/kg, but was observed to reduce fighting behaviours when administered at higher doses. Crowley (1972) concluded that depending on the dose, MA can both increase and decrease aggressive behaviours in rats.

In sharp contrast, Miczek and O'Donnell (1978) reported that a single dose of MA below 8 mg/kg did not increase aggressive or hostile behaviour among mice, compared to mice not injected with MA. This study also differed in that aggressive behaviour was induced by placing a male, ‘intruder mouse’ into the cage of an isolated male mouse or a mouse that was housed with another female mouse. It would seem that these mice were placed in a less painful and possibly less stressful situation or environment than the rats which were placed together and received electric shocks in Crowley’s (1972) study. Indeed, the discrepant findings reported above are thought to reflect variations in experimental conditions, such as drug (MA versus other amphetamine derivatives), dose, acute versus chronic administration, type of animal (i.e., mice or rats), and differences in environment (i.e., confined and overcrowded spaces versus isolation, pain induced versus pain free).

A more recent study has attempted to address these inconsistent results. Sokolov, et al. (2004) compared the effects of long-term intermittent (over eight weeks) exposure to MA, acute exposure and no exposure (saline placebo) on fighting behaviours among mice. Mice were randomly assigned to either receive repeated MA injections, a series of saline injections and a single MA injection, or a series of saline injections alone. Fighting behaviour was measured as the proportion of animals that initiated a bite attack. The amount of time before the first bite was also recorded. As in
the study conducted by Miczek and O’Donnell (1978), aggression was induced by placing an ‘intruder’ mouse into the cage of another ‘resident’ mouse, which was also considered the experimental mouse. Measurements were taken 15 minutes and 20 hours after the final injection of MA. Results showed that a single injection of MA (6mg/kg) had no effect upon fighting behaviour among mice. In contrast, chronic intermittent doses of MA significantly increased the number of bite attacks initiated by mice. Specifically, 70% (21 of 30) of mice in the chronic MA group attacked an intruder when tested 15 minutes after MA injection, while 83% (20 of 24) displayed fighting behaviour 20 hours after drug administration. In contrast, only 14% (2 of 14) of mice in the single injection group initiated a bite attack when tested 15 minutes later, compared with 25% at 20 hours following drug administration. Finally, 12% (5 of 42) of mice in the control group (chronic saline treatment) initiated a bite attack in the experiment.

These findings were replicated in a later study (Sokolov & Cadet, 2006). Consistent with the study above, male mice were randomly assigned to one of three conditions: chronic MA (repeated MA injections), acute MA (repeated saline injections and a single MA injection) or control condition (repeated saline injections). Aggressive behaviour was measured as the time latency before the first bite attack using the resident-intruder paradigm. Aggressive behaviour was assessed during 45 minute observation periods which occurred 15 minutes and 20 hours post injection. Results revealed significantly increased aggression in mice chronically treated with MA at both follow up time points. In contrast, mice that received a single injection of MA were not significantly more aggressive than mice who received saline exclusively.

It is worth noting that the two studies above (Sokolov & Cadet, 2006; Sokolov et al., 2004) attempted to mimic the binge pattern of use that is typical of human MA users, including gradual increases of the drug in the initial stages of use followed by
large doses in short time periods (Yen, Ko, Yen, & Liu, 2005). In the two animal studies above, the mice in the chronic MA condition received increasing amounts of MA over a binge period of three days. They were injected with smaller amounts of MA at two intervals throughout day one (1mg/kg and 2mg/kg, respectively), slightly higher amounts of MA at two intervals on day two (3mg/kg and 4mg/kg, respectively), and even higher concentrations at two intervals on day three (5mg/kg and 6mg/kg, respectively). Given that animal studies typically involve the administration of lower doses of MA than humans would normally use, questions have been raised as to the generalisability of these findings to humans. Nonetheless, these two studies have considerable similarity to human patterns of MA use and thus provide the most conclusive findings regarding the direct impact of MA on aggression. Despite some inconsistency across earlier animal studies, emerging evidence strongly suggests that repeated injection of MA is associated with increased aggression.

Is Methamphetamine Use Linked to Aggression: Evidence from Human Laboratory Studies

Despite the growing body of self-report based research showing that both chronic MA use (Kosten & Singha, 1999; Moss, Salloum, & Fischer, 1994) and acute MA intoxication which might occur during a binge period (R. P. Allen, Safer, & Covi, 1975; Cherek, 1981; Licata, Taylor, Berman, & Cranston, 1993) are significantly associated with heightened aggression in users, only two laboratory studies have directly assessed aggression specifically in MA users. The first of these studies used neuroimaging and a self-report measure of aggression to examine the effects of MA on aggressive behaviour in users, with a particular focus on the role of serotonin in this process (Sekine et al., 2006). The second and most recent study extended Sekine’s study by not only utilising neuroimaging techniques and a self-report measure of
aggression, but also included a behavioural task measuring aggression (Payer et al., 2011). This study was not concerned with the specific role of serotonin in inducing aggression in MA users. Rather, the researchers examined the role of brain function, particularly the function in the amygdala and prefrontal cortex, in the expression of aggression in MA users (Payer et al., 2011). These two studies will be discussed in greater detail below.

In the first study, Sekine and colleagues (2006) examined the complex relationship between MA use, serotonergic functioning and aggression in humans using positron emission tomography (Sekine et al., 2006). The researchers compared 12 currently abstinent, former MA users (5 women / 7 men) to 12 non-drug using controls who were matched according to age, gender and number of years of education. It is worth noting that while the study did not report the amounts of MA formerly used by participants in the MA group, it did specify that they were recreational users recruited from the community who did not have a history of toxic or high dose use. The Structured Clinical Interview for the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (SCID-IV; First, Spitzer, Gibbon, & Williams, 1997) was used to confirm that MA users were not engaging in any other illicit drug use, had no history of mental disorders (including antisocial personality disorder), or history of increased aggression prior to MA use. Notably, retrospective interviews were undertaken with each MA user and their family to ensure accuracy of information regarding duration of MA use and the history of psychiatric symptoms. The SCID-IV was also used to confirm that control participants had no history of MA use or any other illicit drug use, and did not meet criteria for any psychiatric illness in accordance with the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV-TR). The Aggression Questionnaire (AQ; Buss & Perry, 1992) was used to obtain self-
reported scores of aggression, ranging from 29 (low aggression) to 145 (severe aggression). The Brief Psychiatric Rating Scale (BPRS; Ventura, Lukoff, et al., 1993) was used to collect information regarding the presence of negative and positive symptoms of psychosis.

Results revealed that abstinent MA users were more aggressive than control participants. Specifically, the mean total score on the AQ was significantly higher in MA users than in controls ($t = -11.1; P < .001$). Further, of the 12 MA users, 4 demonstrated severe aggression and 4 reported an absence of any psychiatric symptoms except for aggressive behaviour. While all MA users reported previously experiencing psychosis during MA use, none had been hospitalised or received psychiatric treatment. It is worth noting that two participants in the MA group had experienced persistent psychotic symptoms, including persecutory delusions and auditory hallucinations. There were no significant reports of negative psychotic symptoms among either group. Positron emission tomography results revealed that the density of the serotonin transporters in widespread brain regions (e.g., midbrain, cerebellum, amygdala, putamen, thalamus, temporal cortex and occipital cortex) of abstinent MA users was significantly lower than the control group. Furthermore, these reductions in serotonin transporters were even evident in nine MA users who had remained abstinent for at least one year (30% reduction observed, compared with controls). Findings demonstrated that longer durations of MA use were associated with lower serotonin transporter densities. Finally, a particularly relevant finding was that observed decreases in serotonin density in the brain not only corresponded with duration of MA use, but were also associated with increased reports of aggression. The authors concluded that in this study, MA use appeared to be an antecedent to aggressive behaviour, thus suggesting a causal link, with MA use leading to aggressive behavior (Sekine et al., 2006).
As mentioned previously, there is only one other laboratory study investigating the MA-aggression link in humans (Payer et al., 2011). This recently published study extended an earlier investigation in which the same authors investigated MA users’ neural responses to different facial expressions displayed on a computer screen. Results revealed significant activation differences in the right inferior frontal gyrus, an area of the brain involved in both inhibitory control and emotion regulation (Payer et al., 2008). Despite highlighting potential social-cognitive deficits, the initial study was limited in that it did not directly assess the ability to regulate emotion.

This limitation was addressed in a more recent study which not only used an affect matching and labelling task, but also included self-report measures of aggression and alexithymia, neuroimaging techniques and, unlike the study by Sekine and colleagues (2006), employed a task designed to behaviourally measure aggression (Payer et al., 2011). Alexithymia is defined as difficulty with identifying and distinguishing between feelings and difficulty with using words to describe feelings and communicate emotional distress to others (Taylor, Bagby, & Parker, 1997). The research specifically examined neurobiological correlates of affect processing and aggression in 39 MA dependent individuals (16 females, 23 males) who had remained abstinent for seven to ten days and 37 non-drug using controls (18 females, 19 males). A revised version of the AQ (Buss & Warren, 2000) was used to gauge participants’ views regarding the extent to which each specified aggressive behaviour was characteristic of their own behaviour.

It is important to note that while the total sample completed self-report measures, only 12 participants in the MA group and 15 in the control group completed the behavioural task. This subset of individuals participated in a competitive reaction time task in which they were able to choose to subject a fictitious opponent to varying
levels and amounts of aversive noise on trials they won by being the fastest to react to a cue. The program was set so that participants did not win all trials, and instead, on several trials they were led to believe they had responded slower than their fictitious opponent and were consequently blasted with noise themselves. This behavioural measure of aggression, described by the authors as “The Competitive Reaction Time Task” (Payer et al., p. 273), is similar to the Taylor Aggression Paradigm (TAP; S. Taylor, 1976).

Finally, functional magnetic resonance imaging (fMRI) studies were also used to examine links between brain function, aggression and affect regulation in both MA users and non-users, with a particular focus on examining the roles of the amygdala and prefrontal cortex. Again, this procedure was employed with a subset of the total sample (25 MA dependent participants and 23 control participants).

Results revealed that MA dependent individuals self-reported elevated levels of aggression compared to controls (Payer et al., 2011). Further, this finding was supported by performance on the behavioural measure of aggression in which MA users clearly displayed increased aggression, compared with control participants, under conditions of high provocation. More specifically, despite displaying similar levels of aggression in the early stages of the task (where participants were exposed to low and moderate levels of provocation), MA users displayed a sharp increase in aggressive responding following high levels of provocation (i.e., following trials in which they were blasted with loud noise by their fictitious opponents).

Further, while completing an affect matching task, in which participants were required to choose between two faces, the one which they believed matched the emotional expression of the target face, fMRI revealed no significant group differences in relation to amygdala activity. Significant differences were observed, however, in the
bilateral ventral inferior frontal gyrus, with MA users demonstrating lower activation than controls. No group differences were observed when participants completed the labelling affect component of the task which required them to select the emotion or word from two possible choices, which they believed matched the emotional expression on the target face. Both MA users and controls appeared to engage the dorsal inferior frontal gyrus and displayed reduced amygdala activity – neural processes which are indicative of successful emotion regulation. Although decreased amygdala activity was associated with elevated displays of aggression on the behavioural task in both MA users and controls, it was only related to increased self-reported aggression in controls (Payer et al., 2011).

In brief, although the study failed to find evidence of deficits in emotion regulation in MA users, the authors suggested that poor emotional insight may underlie the relationship between MA use and aggression. This notion was supported by the finding that MA users experienced greater difficulties in identifying feelings than control participants, which in turn, correlated with increased self-report and behavioural measures of aggression. Further support was obtained from imaging data which revealed dysfunction in the ventral inferior frontal gyrus of MA users, a region of the brain that is implicated in emotional insight. Notably, the authors demonstrated that the results of this study were not related to acute intoxication (given the study tested recently abstinent MA users), historical patterns of MA use or withdrawal, and concluded that MA-related aggression is clearly mediated by other factors, including personality characteristics (Payer et al., 2011).

While not focusing on MA specifically, another notable study has provided evidence for the role of a different stimulant, ecstasy (3,4-methylenedioxymethamphetamine), in influencing aggression in dependent users (Gerra et al., 2001).
Gerra and colleagues (2001) used the Point Subtraction Aggression Paradigm (PSAP) to compare the behavioural responses of 12 male, long term ecstasy users, who had remained abstinent for 3 weeks prior to testing, with 20 male, non-drug using controls. The PSAP represents a behavioural measure of aggression and involves participants competing against a fictitious opponent on a computer-based task to earn money. The fictitious partner provokes aggressive responding from the participant by subtracting money unexpectedly. The participant is able to respond by subtracting money in retaliation, with the amount deducted providing the dependent measure of aggressive responding. Results demonstrated that the ecstasy users made significantly more aggressive responses than controls (Gerra et al., 2001). Notably, this study employed a rigorous experimental design and while it focused on an ecstasy using population rather than a MA using one, both drugs are stimulants and are likely to have similar effects on behaviour. This study, taken together with the two laboratory studies described above, which have directly assessed aggression in MA users, have provided strong evidence of a link between stimulant abuse, including MA and increased aggression.

Neurobiological Processes Involved in Aggression and Methamphetamine Use

It is well documented that MA has a neurotoxic effect on serotonergic pathways in animals and humans (see Nelson & Chiavegatto, 2001, for review). Serotonin, or 5-hydroxytryptamine (5-HT), is produced in the brain from an amino acid tryptophan and plays an important role in a number of behavioural functions, including the sleep/wake cycle, appetite, sensory-motor activity, learning and inhibiting impulsive responses to frustration, such as aggression. Tryptophan hydroxylase (TPH) is an enzyme that controls the rate of synthesis of the neurotransmitter serotonin. It can limit the production of serotonin since it is the only catalyst in the reaction producing serotonin. Thus, reduced serotonergic functioning is linked to the deficiency of TPH. Serotonergic
activity can be determined by measuring the levels of 5-hydroxyindoleacetic acid (5-HIAA) in cerebrospinal fluid (CSF).

Animal experiments have shown that MA use reduces serotonin transporter density in rats (Kovachich, Aronson, & Brunswick, 1989). Further, there is an established link between decreased serotonergic activity and increased aggression in animals. For example, destruction of brain serotonin (5-HT) cells and pathways has been associated with an increase in aggression in rodents (Feldman, Meyer, & Quenzer, 1997). Conversely, administration of serotonin agonists reduced isolation-induced, resident-intruder and maternal aggression among rodents (Feldman et al., 1997). Higley et al. (1996; 1996; 1996) studied the relationship between serotonergic pathways and aggressiveness in rhesus monkeys living in large, free ranging colonies, using the number of wounds, scars and aggressive encounters monkeys displayed as a measure of behavioural aggression in addition to CSF to measure 5-HIAA levels – a metabolite of serotonin (5-HT). A negative correlation was found between 5-HIAA and aggression. Specifically, male monkeys with low CSF 5-HIAA concentrations displayed higher rates of severe, unrestrained, and unprovoked aggression than monkeys with high CSF 5-HIAA concentrations. They engaged in prolonged chases and physical attacks on other monkeys and the aggression they displayed typically escalated to a dangerous level. In contrast to these findings, diminished serotonergic functioning was not associated with overall rates of aggression, which included the expression of less intense, restrained aggression which is typically exercised in self-defense.

In addition to inhibiting aggression, serotonin also appears to exert a strong influence on impulsive and risky behaviour. In the studies described above, Higley et al. (1996; 1996) also found that low 5-HIAA was associated with high risk-taking behaviour (e.g., unprovoked, long leaps at dangerous heights and repeatedly being
captured in baited traps), including very high levels of aggression towards older and larger animals. Further, high rates of impulsive behaviour were positively correlated with unprovoked and unrestrained aggression. These aggressive monkeys were also noted to engage in a range of risk taking behaviours and displayed reduced behavioural control (Higley, King, et al., 1996; 1996; 1996). For instance, monkeys with low levels of serotonergic activity were more likely to initiate fights they could not win and 46% died as a result of attacks from mature males. Direct observations revealed that the deceased monkeys had initiated escalated aggressive behaviour towards the other monkeys, unlike the surviving monkeys who did not initiate aggression. Further, examination of the deceased bodies revealed wounds and scars that indicated a history of fierce and aggressive behaviour. The authors concluded that low 5-HT turnover may reflect poor or reduced impulse control rather than increased aggression per se. It was suggested that decreased serotonergic activity reduces animals’ inhibitory control (thereby increasing impulsivity) across a number of behavioral domains and thus, the high rates of violent aggression shown by these monkeys was possibly a product of impaired impulse control (Higley, King, et al., 1996; 1996; 1996).

Indeed, a large body of research conducted prospectively has clearly demonstrated that free-ranging rhesus monkeys with low levels of serotonin, as measured through samples of CSF 5-HIAA, displayed deleterious behaviour patterns including impulsive, unrestrained aggression, excessive risk-taking and social isolation (Kaplan, Fontenot, Berard, Manuck, & Mann, 1995; Mehlman et al., 1994, 1995; 1997). Further, these behaviour patterns have been shown to remain stable over extended periods of time. Howell et al. (2007) undertook a longitudinal investigation of the relationship between serotonin and aggression in 104 free-ranging rhesus monkeys. The monkeys were observed over a 10 year period, from infancy (24 to 36 months at
baseline) through to adulthood (12 to 13 years). As in earlier studies, monkeys with low CSF 5-HIAA displayed high levels of aggression over a 10 year period. Further, these monkeys were also more likely to die prematurely as a result of behaving aggressively and engaging in high risk behaviours.

It is worth noting that a majority of the animal laboratory studies have focused on male primates. While less attention has focused on examining the serotonin-aggression link in female animals, the small body of emerging literature suggests a similar pattern. In one study, 44 juvenile female monkeys living freely on a large sea island were observed over an 18 month period (Westergaard et al., 2003). Among other behaviours (including proximity, social grooming and submission), observers recorded the frequency with which the monkeys engaged in impulsive behaviour which was measured as unprovoked leaping between trees at dangerous heights, and aggression. Unprovoked leaping behaviour was scored as frequency data and fell into one of three categories of escalating danger – short leaps – up to one metre, medium leaps – one to three metres in distance, long leaps – greater than three metres. A short, medium and long leap ratio was calculated by dividing the number of either short/medium/or long leaps by the total number of all combined leaps. Aggressive behaviours were defined and categorised by intensity into displacements, stationary threats, chases, and physical assaults. The frequency of these behaviours was rated per hour and behaviours were classified into one of two levels of aggression – moderate aggression (displacements and threats) and severe aggression (chases and assaults). Physiological data was also collected including CSF 5-HIAA concentrations in blood samples. Univariate, linear analyses were conducted by correlation z-tests and regression to test for correlations between physiological measures, behavioural variables and possible confounding
factors. Monkeys with values two or more standard deviations above or below the mean were excluded from analyses.

Results revealed that long leaping between trees was positively correlated with both moderate and severe aggression, with the correlation stronger for severe aggression (severe aggression: $r (39) = 0.46, p<0.002$; moderate aggression: $r (39) = 0.35, p<0.03$). There was no relationship found between short or medium leaping behaviour and aggressive behaviour. Further, consistent with their male counterparts, female monkeys with low CSF 5-HIAA concentrations displayed more impulsive and risk taking behaviours than females with high concentrations. Interestingly, however, low levels of serotonin were not associated with more severe, high-intensity, impulsive aggression. Rather, in female monkeys, low CSF 5-HIAA was associated with less intense (i.e., moderate), restrained aggression that is typically used to defend status. This gender discrepancy might be understood in light of early research which suggests that while male and female rhesus monkeys tend to engage in relatively equal overall rates of aggression in their natural habitats, females are less likely to display severe aggression than males (Lindburg, 1971). Further, given that females tend to spend more time in family groups, aggressive incidents are more likely to occur within this context and so might also be characterized by less severe aggression. Others have suggested that female macaques simply appear to engage in less severe or intense aggression on the basis that they are smaller than their male counterparts, and possess smaller canines which produce less severe trauma and wounding (Westergaard et al., 2003).

Irrespective of the differences in intensity of aggression, animal research has clearly demonstrated a link between serotonergic functioning and increased impulsivity and aggression in non-human primates.
Similar patterns of association between low serotonin levels and aggression have been observed in other animal species, including canines. A very recent study examined the link between serotonin and aggression in dogs (Rosado et al., 2010). The study compared 80 dogs, which had been referred to animal behavioural services as a result of behaving aggressively towards people, to 19 control dogs who had no history of aggression towards people or other dogs. Analysis of blood samples revealed that aggressive dogs had significantly lower serum concentrations of 5-HT (serotonin) with no observed gender differences. Numerous other studies have reported similar links between depleted serotonin and increased aggression among canines (Cakiroglu, Meral, Sancak, & Cifti, 2007; Peremans et al., 2003; Rosado et al., 2010).

More than two decades of research has demonstrated that a reduction in brain serotonin may play a similar role in human aggression (Coccaro, 1996; Coccaro & Siever, 2002). Studies employing a variety of paradigms, including measurement of CSF serotonin metabolites, hormonal response to serotonergic probes, and imaging metabolic changes with serotonergic agents, have found that individuals with abnormally low levels of serotonin are more violent and impulsive than those who have normal serotonergic activity (Coccaro, Kavoussi, Hauger, Cooper, & Ferris, 1998; Davidson, Putnam, & Larson, 2000; Frankle et al., 2005; Kish et al., 2009; Sekine et al., 2006). In early studies, reduced CSF concentration of the serotonin metabolite 5-HIAA was found in individuals who reported a lifetime history of aggression. Brown and colleagues (1979) were among the earliest researchers to demonstrate this relationship when they examined serotonergic functioning in 24 male naval recruits with a lifetime history of aggression (Brown et al., 1979). Specifically, they found a strong inverse relationship ($r = -0.78$) between aggression and CSF 5-HIAA concentrations among these men.
These findings have been replicated in a number of studies involving clinical and prison populations. For instance, Limson et al. (1991) reported an inverse correlation between CSF 5-HIAA and a life history of aggression in abstinent alcoholics and controls, while Brown et al. (1982) reported similar findings in their study of 12 naval recruits with a diagnosis of Borderline Personality Disorder. Further, numerous studies have demonstrated that males with psychopathological conditions associated with impaired impulse control, such as explosive aggression, impulsive violence and social impairment had low CNS serotonin 5-HT activity (Brown et al., 1982; Brown, Goodwin, Ballenger, Goyer, & Major, 1979; Kruesi et al., 1990; Linnoila et al., 1983; Virkkunen, Kallio, et al., 1994; Virkkunen, Rawlings, et al., 1994). Linnoila et al. (1983) found that incarcerated men who had committed multiple violent acts had lower levels of serotonin than those who had engaged in a single, non-impulsive, premeditated act of violence. This finding suggested that 5HT functioning may be primarily associated with an impulsive or reactive subtype of aggressive behaviour. In a more recent study, Frankle et al. (2005) examined the regional serotonin transporter distribution in the brains of 10 individuals with impulsive aggression and 10 age and sex matched healthy controls, using positron emission tomography with the serotonin transporter radiotracer[^sup 11^C]McN 5652. Results indicated that serotonin transporter availability was significantly reduced in the anterior cingulated cortex of those individuals with impulsive aggression compared to controls. Further, this region of the brain is known to play an important role in affect regulation (Frankle et al., 2005). Overall, many studies have demonstrated that reduced serotonergic functioning is associated with reduced behavioural control and increased aggression in individuals with a range of clinical and psychopathological conditions.
Despite the numerous studies investigating the link between serotonin and aggression in humans, only two studies have focused on the link between serotonin and aggression in MA using populations. The first of these studies, conducted by Sekine et al. (2006) has been discussed in detail above (see p. 17) and has provided strong evidence of a link between MA, serotonin reduction and aggression in MA users. Of particular interest was the finding that longer durations of MA use were associated with the lowest levels of serotonin transporter density.

Similar findings were reported in a markedly different study by Kish et al. (2009). Post-mortem analyses of the brains of 16 chronic and recent MA users (11 males and 5 females) and 24 healthy controls (21 males and 3 females) with no evidence of brain pathology were compared. They found modest decreases in serotonin transporter protein expression in the striatum of MA users compared with controls. Further, concentrations were significantly decreased in orbitofrontal and occipital cortices of MA users. These findings support the proposition that MA causes dysfunction in areas of the brain, particularly the orbitofrontal cortex, which are known to be associated with impaired behavioural control. Taken together, these two studies provide evidence that MA affects serotonergic functioning, which in turn, is associated with increased aggression in users.

Kish et al. (2010) extended this study by examining whether the same damage to serotonin systems was observed in living ecstasy users. The researchers compared brain serotonin transporter binding using [(11)C] N,N-dimethyl-2-(2-amino-4-cyanophenylthio) benzylamine in a group of 49 recently abstinent (average of 45 days), ecstasy users with low to moderate use (average of four years use of 1 to 2 ecstasy tablets per fortnight), with 50 non-ecstasy using control subjects. Hair scalp analysis was used to confirm reported levels of ecstasy use and to rule out the presence of other
stimulants which impact on brain serotonin systems, including MA and cocaine. Results revealed that 18% of ecstasy users had recently used MA and 29% had also used cocaine. A magnetic resonance image and structural analyses were collected for all participants. Serotonin transporter binding in ecstasy users was significantly reduced throughout all cerebral cortices (ranging from 19% to 46%) and the hippocampus (21%). These reductions paralleled years of drug abuse and dose level, such that longer periods of use and greater amounts of the drug were associated with greater reductions. Further, ecstasy users who also used MA displayed a slight left-hemispheric biased cortical thinning that was not observed in any other group. Compared to controls, heavier ecstasy users reported more marked mood shifts following cessation of use and demonstrated greater reductions in serotonin transporter binding. The ecstasy group performed more poorly than controls on tests of attention, executive function and memory, which was also associated with decreased serotonin transporter binding. Overall, findings suggested that low to moderate doses of ecstasy resulted in mild to marked loss of serotonin transporter density in the cerebral cortex/hippocampus in the range of that observed in Parkinson's disease, which is not related to gender or recent use of other drugs (Kish et al., 2010).

In summary, it has been clearly demonstrated that deficits in central serotonin (5-HT) are associated with increased aggression and reduced behavioural control in both the animal and human laboratory. Further, it has been suggested that low levels of serotonin show long term inter-individual trait-like stability over time (Higley, King, et al., 1996; Howell et al., 2007; Lenzenweger, 1999). Given the knowledge that MA use results in depletion of serotonin, it seems reasonable to propose that individuals who use this drug repeatedly, chronically or in very high concentrations, are more likely to display heightened aggression. Alternatively, it seems equally reasonable to posit that
MA use may result in a more disinhibited pattern of behaviour generally, which presents itself as aggression in some users. Further clarification is needed to determine whether the aggression in MA users is best understood as a specific, direct effect of MA use, or as a byproduct of overall reduced behavioural control or inhibition more generally.

**Gender Differences in the Expression of Aggression**

The association between gender, aggression and MA use is somewhat unclear. Some studies have found similar rates of aggressive and violent behaviour among male and female MA users. For instance, one study found that among a group of MA users ($n = 39$), equal numbers of males and females reported incidents of MA related violence including arguments, fights, assaults, and property damage. Both genders attributed their violent behaviours to drug-related disputes, feeling agitated and angry during withdrawal, and experiencing paranoid thinking and hallucinations (Sexton, Carlson, Leukefeld, & Booth, 2009). Similarly, Brecht et al. (2004) found that equally high numbers of male and female MA users reported violent and aggressive behaviours with 57% of a large sample ($n = 350$) reporting such problems.

Despite the above mentioned studies reporting an absence of gender differences, there is some evidence suggestive of higher rates of aggression and violence among male MA users. For instance, in the study previously described, Sommers et al. (2006) found that more male MA users reported perpetrating violence than female MA users (38% versus 30%, respectively). In contrast, a small number of studies have suggested that although the association between MA use and aggression is evident among both males and females, higher rates of MA related violence are observed among females (Dluzen & Liu, 2008; Hamilton & Goeders, 2010; Venios & Kelly, 2010). For instance, Zweben et al. (2004) found that 46% of women compared with 40% of men reported
experiencing significant difficulties in controlling their anger and violent behaviour. A longitudinal study involving more than 1000 MA users also found that female MA users reported greater difficulty controlling their aggression than their male counterparts, with 13% of females versus 10% of males reporting problems (Hser, Evans, & Huang, 2005). Interestingly, research suggests that the relationship between MA use and aggression may be bidirectional in females. For instance, a substantial proportion of female MA users have reported histories of abuse, particularly partner aggression (Cohen et al., 2003). Similarly high numbers of incarcerated, MA using women have also reported a history of sexual abuse in childhood, adulthood or both (Vik & Ross, 2003). Furthermore, among the 77 women interviewed, the highest rates of sexual assault were reported by those women who injected MA compared to non-injecting MA users and non-stimulant drug users. In a longitudinal study involving 1073 MA users, female users were more likely than male users to report incidents of physical abuse and sexual abuse in the past 30 days (Hser et al., 2005). These studies suggested that some female MA users may have a greater propensity than male MA users to not only be exposed to aggression, but to also engage in aggressive behaviour. In brief, the literature concerning gender differences in MA-related violence and aggression is mixed with some studies reporting differences in favour of men, some reporting differences in favour of women, and others reporting equally high and alarming rates of aggression among both male and female MA users.

**Summary**

MA use has been strongly associated with heightened aggression, hostility and violence. While the majority of research has used self-report data, the results of these studies have been supported by a large number of animal-based laboratory, field, and experimental studies and a smaller number of human experimental studies. Despite
evidence of a strong link between MA use and aggression, it is also apparent that aggression does not occur amongst all MA users. More specifically, while a large number of MA users have reported engaging in increased aggressive and hostile behaviour, others, including some who used large quantities of the drug, did not. One possible explanation for this is that there are other, yet to be identified factors influencing the association between MA and aggression.

**Interpreting the Methamphetamine-Aggression Link – The General Aggression Model**

Several possible explanations, each with their own theoretical underpinnings have been described above in an attempt to understand the relationship between MA use and elevated levels of aggression. The General Aggression Model, proposed by Anderson and Bushman (2002), is also useful in understanding this phenomenon. For the purpose of this thesis, aggression is defined as “any action toward another person that is elicited by provocation, driven by anger, and intended to cause harm” (Payer et al., 2011, p. 271). According to the General Aggression Model, the expression of aggression reflects a complex process in which an individual first experiences an internal reaction to a particular situation, and then engages in appraisal and decision making processes aimed at making sense of the situation and one’s own internal state. The outcome of these appraisal and decisional making processes then determines whether or not an individual responds aggressively. Based on the assumptions of this model, aggressive behaviour is more likely to occur in individuals with reduced cognitive capacity, poor emotional insight and emotional processing abilities, and a reduced ability to regulate uncomfortable emotions such as hostility and anger, than in individuals who are able to engage the required cognitive resources and have sufficient emotional insight to work through a problem to reach a thoughtful outcome.
As will be discussed in the next chapter, MA use has been associated with a range of cognitive deficits including, but not limited to, deficits in response inhibition (Monterosso et al., 2005; Salo et al., 2005), which is a component of the General Aggression Model (Anderson & Bushman, 2002). Further, other studies have demonstrated that MA users experience difficulties with identifying facial emotions (Henry, Mazur, & Rendell, 2009; Payer et al., 2008), describing their own feelings (Payer et al., 2011), and with theory of mind (Y. T. Kim et al., 2010; Y. T. Kim, Kwon, & Chang, 2011); or more broadly, the ability to see things from another’s perspective (Henry et al., 2009). This is supported by other research which has shown that MA users demonstrate poor emotional insight and self-awareness (R. Z. Goldstein et al., 2009; Homer et al., 2008; Verdejo-García & Pérez-García, 2008). In accordance with the General Aggression Model, MA users may be at increased risk of behaving aggressively given the apparent cognitive deficits they possess – deficits which play a vital role in determining whether an individual reacts impulsively with aggression, or is able to work through a situation both cognitively and emotionally to achieve a non-aggressive outcome.
CHAPTER 3

Factors Influencing the Expression of Aggression in Methamphetamine Users

Current understanding and knowledge of why an association between MA use and aggression is observed in some users, but not in others, is extremely limited. While many studies clearly demonstrate an association between MA use and aggression, there has been relatively little attention paid to factors that may influence the propensity to act aggressively. Two variables which have been strongly, although separately, associated with both MA use and aggression are impulse control (the ability to inhibit behaviour) and psychotic symptoms. Despite these independent associations, research is yet to determine whether these factors play a mediating or moderating role on the expression of aggressive behaviour in MA users. In the following section, studies that have investigated each of these factors will be reviewed.

Methamphetamine Use, Impulsivity and Disinhibition

In relation to the first of the above factors, the association between MA use and impaired inhibitory control is well established. Studies investigating the relationship between MA use and impulsivity can be divided into three broad categories. First, there are studies which have used behavioural measures of impulsivity to demonstrate that MA users show impairments in the inhibition of behavioural responses (Baicy & London, 2007; Fillmore & Rush, 2002; Li et al., 2006; Monterosso et al., 2005; Salo et al., 2002; Simon et al., 2002), which can also be seen as an increase in risk taking behaviours, compared to non-drug using individuals (Leland & Paulus, 2005). Second, there are studies which have used neuroimaging techniques to demonstrate neurobiological and metabolic changes in the frontal and prefrontal cortices of MA users, which are implicated in inhibitory control (Leland et al., 2006; Paulus et al., 2008). Finally, a number of self-report based studies have also found elevated scores
on measures assessing impulsivity and related constructs (Clark, Robbins, Ersche, & Sahakian, 2006; Coffey et al., 2003; Ersche, Roiser, Robbins, & Sahakian, 2008; Moeller et al., 2004; Semple, Zians, Grant, & Patterson, 2005).

A growing number of studies have confirmed an association between impulsivity and stimulant use - a link that appears to be bidirectional (Dawe & Loxton, 2004; De Wit, 2009). While some researchers have confirmed the role of impulsivity as a risk factor in initiating and maintaining stimulant use, other studies have focused upon the effects of stimulant use on behavioural control or levels of impulsiveness in users (see Perry & Carroll, 2008, for a review). Impulsivity has been defined in a variety of ways, including a “tendency to act spontaneously and without deliberation” (Carver, 2005, p. 313). Recently, Moeller, Barratt, Dougherty, Schmitz, and Swann (2001), offered the alternative term of behavioural impulsivity to refer to this “predisposition toward rapid, unplanned reactions to internal or external stimuli without regards to the negative consequences of these reactions to themselves or others” (p. 1784). Despite various definitions, there is general consensus in the literature that impulsivity is a multifaceted construct involving several key elements including acting rashly, acting without adequate thought, acting with less forethought than someone with equal knowledge, acting before considering the possible consequences, failing to plan ahead, failing to perseverate, and of particular relevance to the current thesis, failing to withhold a response once a behaviour is initiated (Cross, Copping, & Campbell, 2011; Dawe & Loxton, 2004).

Three broad categories of laboratory tasks have been developed to behaviourally measure impulsivity. First, punishment and/or extinction paradigms measure impulsivity by assessing the perseverance of a response that is punished or unrewarded. Second, in reward-choice paradigms, impulsivity is measured by demonstrating a
preference for a small immediate reward over a larger delayed reward. Third, response disinhibition/attentional paradigms tap impulsivity by measuring responses that are premature and the inability to withhold a response (Moeller et al., 2001).

Indeed, the construct impulsivity is often equated with a description of reduced behavioural inhibition, otherwise known as disinhibition. The term disinhibition typically refers to a top-down cognitive process in which control mechanisms that are responsible for the suppression of automatic and reward-driven responses fail to operate adequately and result in a predisposition towards behaving impulsively (Verdejo-García, Lawrence, & Clark, 2008). The opposite process, referred to as behavioural or response inhibition, is defined as “the ability to prevent any form of planned physical response” (Eagle, Bari, & Robbins, 2008, p. 439). Individuals with low levels of inhibition experience problems with self-regulation and difficulties in withholding inappropriate responses in situations in which environmental contingences impel them to do so.

Broadly speaking, several studies demonstrating a relationship between MA, impulsivity and reduced behavioural control have employed a combination of neuroimaging techniques, such as fMRI, positron emission tomography, and behavioural measures of response inhibition. These studies have investigated the neurotoxic effects of MA on neurotransmitters and/or brain structures in MA users, and the resulting neuropsychological impairments, including reduced behavioural inhibition and impaired decision making (Leland et al., 2006; Salo, Nordahl, Buonocore, et al., 2009; Salo, Nordahl, Galloway, et al., 2009; Salo, Ursu, Buonocore, Leamon, & Carter, 2009).

**Methamphetamine use, effects on the brain and disinhibition.** According to Baicy and London (2007), the way in which an individual responds to people and events
in their environment is influenced by complex cognitive processes, including the interplay of cortical and subcortical interactions. While subcortical activity is associated with an individual being able to respond rapidly to a situation, cortical involvement is often necessary and useful in assisting an individual to evaluate the situation at hand and thus guide their behavioural response. This ensures that individuals don’t just take quick, rash action, but rather they are able to make a guided decision about how to behave or respond.

**Brain imagining and behavioural studies – metabolic and structural changes in the brain.** Brain imaging and behavioural studies have shown that chronic MA users demonstrate metabolic changes in the brain which are possibly associated with behavioural and cognitive control issues. One of the ways in which chronic MA use might influence brain structures and functioning to bring about reduced behavioural control relates to the changes it causes in frontocortical blood flow and glucose utilization (Berman et al., 2008; S. J. Kim et al., 2005; London et al., 2005; London et al., 2004; Robinson & Berridge, 2003; Volkow et al., 2001). Frontocortical blood flow is an index of brain function and as such, is useful in examining differences in cognitive functioning between MA users and their non-drug using counterparts. Studies have found that blood glucose perfusion rates in chronic MA users are below control values in certain brain structures, including the insula (Chang et al., 2002). This finding is important in so far as the insula plays an important role in an individual’s ability to recognise both their own internal state and that of others and thus regulate behaviour (Critchley, 2005). Research involving clinical populations who experience difficulties with impulse control, such as Borderline Personality Disorder, has shown that dysfunction of the insula cortex is associated with increased impulsivity, particularly
impulsive aggression (Lamm & Singer, 2010; Soloff, Meltzer, Greer, Constantine, & Kelly, 2000; Takahashi et al., 2009).

A recent study by S. J. Kim et al. (2005) examined the changes in regional cerebral glucose metabolism (rCMRglc) in abstinent MA users and the impact of these changes on frontal executive function. Thirty three abstinent MA users and 21 healthy gender matched controls completed the Wisconsin Card Sorting Test (WCST) and were scanned using positron emission tomography. The WCST is a test of executive function measuring the ability to form abstract concepts, to shift and maintain set, and to utilise feedback (Greve, Stickle, Love, Bianchini, & Stanford, 2005; Yoo et al., 2000). Results revealed significant decreases in rCMRglc in the frontal white matter in MA users relative to the healthy controls. Further, unlike healthy controls, MA users demonstrated significant impairment in frontal executive function, as evidenced by more total errors, perseveration errors, and non-perseveration errors in the WCST. Other studies have reported similar results (London et al., 2004; Volkow et al., 2001; Wang et al., 2004) and taken together, point to metabolic and cortical abnormalities that could underlie a range of impulsive and disinhibited behaviours shown by individuals who use MA.

In addition, neuroimaging and behavioural studies have also shown that chronic MA users demonstrate prominent functional and/or structural abnormalities within several regions of the brain, including the limbic and paralimbic cortices, subcortical areas, and prefrontal cortices (Baicy & London, 2007). These deficits are in turn, associated with behavioural impairments including reduced inhibition. More specifically, chronic MA use has been shown to be associated with alterations in brain function in the amygdala and prefrontal cortex, which in turn, are associated with reduced behavioural control (Leland et al., 2006; Paulus et al., 2008). While the
AMYGDALA has been shown to play a vital role in regulating rapid, automatic responses to environmental and social stimuli (Adolphs, 2003; LeDoux, 2007), the prefrontal cortex is involved in regulating more deliberative aspects of emotional processing, response selection and behavioural control (Adolphs, Tranel, & Baron-Cohen, 2002; Posamentier & Abdi, 2003). Effective emotional stress regulation and inhibitory control is dependent on top-down control from the prefrontal cortex over subcortical regions involved in reward and emotion (Heatherton & Wagner, 2011; Li & Sinha, 2008). Thus, self-regulation of behaviour fails whenever the balance is tipped in favour of subcortical areas, either due to particularly strong impulses, or when prefrontal function itself is impaired (Heatherton & Wagner, 2011).

Paulus, Hozack, Frank, Brown, and Schuckit (2003) used fMRI to compare the neural activity of 14 MA dependent males with 14 age-matched non-drug using males while they completed a two-choice prediction task. MA dependent males were found to demonstrate dysfunction in their decision making such that irrespective of success rate, the MA-dependent males demonstrated a rigid, stimulus response pattern including making more win-stay, lose-shift consistent responses than controls. Further, neuroimaging results showed that, unlike controls, MA users displayed the highest rates of activation in key brain regions when the outcome was most unpredictable. Nevertheless, compared with controls, MA dependent males demonstrated less task related activation in orbitofrontal cortex, dorsolateral prefrontal cortex, anterior cingulated and parietal cortex. This study clearly demonstrated that MA use was associated with reduced activation in frontal regions of the brain known to be associated with executive functions, including decision making and behavioural control.

In another study, Thompson et al. (2004) used high resolution MRI and new computational brain-mapping techniques to examine the pattern of structural brain
alterations associated with chronic MA use in human subjects and related these deficits to cognitive impairment. Regional abnormalities were mapped in the cortex, hippocampus, white matter, and ventricles in 22 MA dependent users and 21 age-matched, healthy controls. Cortical maps revealed severe gray-matter deficits in the cingulate, limbic, and paralimbic cortices of MA users (averaging 11.3% below control; \( p < 0.05 \)). MA users had hippocampal volumes that were 7.8% smaller (on average) than controls and significant white-matter hypertrophy (7.0%). Hippocampal deficits were mapped and correlated with memory performance on a word-recall test (\( p < 0.05 \)). MRI-based maps indicated that chronic MA use causes a selective pattern of cerebral deterioration that contributes to impaired memory performance. Further, MA may selectively damage the medial temporal lobe and, consistent with metabolic studies, the cingulate-limbic cortex. This study adds further support to a growing literature base demonstrating an association between MA use and neuropsychological dysfunction, which in turn, is possibly associated with compromised executive function.

Research has shown that MA users may experience dysfunction in these essential brain structures and cortical systems as a result of the neurotoxic effect of the drug on dopaminergic systems. While MA-induced neurotoxicity has been shown to impact on several neurotransmitter systems (i.e., serotonergic and non-monoaminergic systems), it exerts a particularly strong influence on dopaminergic pathways (Cass, 1997; J. C. Scott et al., 2007). A series of acute and chronic doses of MA have been shown to significantly deplete dopamine levels, destroy dopamine nerve terminals, and cause long-term reductions in other markers of dopamine terminal integrity in users (Harvey, Lacan, Taniouis, & Melega, 2000; Lee, Groman, London, & Jentsch, 2007; Lee et al., 2009; Miller & O’Callaghan, 2003; van Gaalen, van Koten, Schoffelmeer, & Vanderschuren, 2006). Dopamine plays a crucial role in a number of functions,
including control of movement, regulation of emotional responses, and regulation of the reward system. Dopamine also influences impulsivity and behavioural inhibition (Lee et al., 2009). Numerous in vivo positron emission tomography studies have shown that chronic MA users exhibit lower levels of dopamine transporters in the striatum (McCann et al., 1998; Sekine et al., 2001; Volkow et al., 2001), orbitofrontal and dorsolateral prefrontal cortex and amygdala (Sekine et al., 2003) – areas of the brain implicated in behavioural control. For instance, Volkow et al. (2001) found that dopamine transporter levels in the striatum of the brain were 24% lower in MA users than controls. Further, MA users performed poorer on tests measuring brain function associated with the striatum including fine motor skills and gross motor skills. This reduction in performance was proportional to the deficits in dopamine transporters. Similarly, Sekine et al. (2001; 2003) found lower dopamine transporter availability in the striatum and prefrontal cortex of MA users compared with non-drug using controls, with levels ranging from 20% to 33%.

Recently, Lee et al. (2009) assessed the link between dopamine and impulsivity in a group of MA dependent individuals (n = 51) and healthy controls (n = 66). While MA addiction has been shown to be associated with both impulsivity and striatal dopamine D2/D3 receptor deficits, this was the first laboratory study to directly test this (Lee et al., 2009). More specifically, although previous research reported correlations of striatal D2/D3 receptor availability with glucose metabolism in the prefrontal cortex (Volkow et al., 2001; 1993; 2008), a potential moderator of impulsivity (Brennan & Arnsten, 2008; Dalley, Mar, Economidou, & Robbins, 2008), these studies were limited to providing indirect support for this relationship. In the study conducted by Lee et al. (2009), both groups completed the Barrett Impulsiveness Scale (BIS-11), a self-report measure of impulsiveness, and dopamine D2/D3 receptor availability was assessed.
using positron emission tomography scanning. Group differences were examined on the three BIS-11 subscales using multivariate analysis of covariance (MANCOVA). Holm-Bonferroni corrections were applied to post hoc independent-samples analysis of covariance (ANCOVA). Results revealed that MA dependent users scored significantly higher on than controls on all BIS-11 subscales (all $F_{(1,115)} \geq 13.1$, all $p$ values < 0.001).

Further, a MANOVA was also used to examine differences in levels of dopamine available in critical regions of the brains in MA users compared with controls. Results revealed that MA users exhibited lower striatal D2/D3 receptor availability in the left caudate nucleus (16.1% lower), the putamen (12.6% lower), and in the nucleus accumbens (8.4% lower). Post hoc, independent ANCOVA confirmed significant differences for the caudate nucleus ($F_{(1,49)} \geq 11.1$, $p < 0.001$) and the putamen ($F_{(1,49)} \geq 6.6$, $p < 0.01$). Finally, the relationship between BIS-11 scores and striatal D2/D3 receptor was examined. For the MA group, a significant negative correlation ($r = -1.71$, $p = 0.004$) was found between lower striatal D2/D3 receptor availability and higher impulsiveness. Upon examining the three specific regions of the brain of interest, Holm-Bonferroni corrections were applied and a significant post hoc correlation was obtained for the caudate nucleus ($r = -0.63$, $p = 0.002$).

In summary, chronic MA users have been shown to experience metabolic changes in frontocortical blood flow and glucose systems, alterations in neurotransmitters and have demonstrated prominent functional and structural abnormalities within several regions of the brain that play a central role in regulating how an individual will respond behaviourally to their environment.

**Brain imaging and behavioural studies – a review of key findings.**

According to Jentsch and Taylor (1999), the brain changes and abnormalities associated with chronic MA use, particularly the associated dysfunction in frontal regions of the
brain, play a critical role in explaining the relationship between MA use and impulsivity. Research has shown that frontocortical systems are involved in executive processes including decision making, considering consequences of one’s actions and providing inhibitory control over behaviour (Davidson et al., 2000). Although researchers have investigated a range of cognitive deficits in MA users, including deficits in working memory, decision making and cognitive flexibility, the current thesis is particularly interested in the studies which have examined response/behavioural inhibition in stimulant users (Fillmore & Rush, 2002; Fillmore, Rush, & Hays, 2002; Li et al., 2006), particularly in MA users (Monterosso et al., 2005; Salo, Nordahl, Buonocore, et al., 2009; Salo, Nordahl, Galloway, et al., 2009; Salo, Ursu, et al., 2009). Indeed, behavioural studies investigating the link between MA use and inhibitory behavioural control have clearly demonstrated that MA users display reduced response inhibition, compared to non-MA users.

In a review of the literature, Jentsch and Taylor (1999) argued that chronic MA use results in decreased cognitive and response inhibitory control over behaviour, such that those individuals with prefrontal damage are unable to inhibit approach behaviour once they have begun to act. It is this inability to desist from action once initiated that is proposed to influence drug use such that thoughts about drug use result in drug-seeking behaviour without regard to the consequences. Further, it might also explain why some MA users have a greater propensity to display disinhibited, impulsive, or poorly considered behaviour.

The four most widely used tests to directly measure the ability to suppress an automatic or prepotent behavioural response in substance using populations are the Stroop Task (Stroop, 1935), the Stop Signal Task (STOP-IT; Logan, 1994), the Go/No Go Task (Vocci, 2008) and measures of commission errors on Continuous Performance
Tests (Logan et al., 1997). Nevertheless, the majority of studies which have examined behavioural inhibition in MA using populations specifically, have used the Stroop Task (Stroop, 1935), which is a test of selective attention that requires subjects to engage cognitive control to inhibit a prepotent but task-irrelevant response (i.e., word reading) and execute the task-relevant response. More specifically, the individual is required to read aloud the ink colour of a row of X’s as quickly as possible (control condition). The individual is then required to name the ink colours in which a series of words is printed – which is challenging because the words themselves, are actually the names of colours (interference condition). Each colour name (e.g., blue) is different from the ink colour it is printed in (e.g., red). The two conditions are compared and the difference between them provides a measure of the time taken to resolve the conflict between an automatic, non-desired response (e.g., word reading) and an effortful, desired response (e.g., colour naming). A larger interference score is indicative of reduced behavioural control.

In one of the earliest studies to assess response inhibition in MA users, Salo et al. (2002) used a computer-based version of the Stroop Task to compare the performance of 8 currently abstinent MA users and 12 age matched, non-drug using controls. In the computerized version of the Stroop, participants are required to select their responses using their computer keyboard, rather than reading them out aloud. Individuals in the MA group had used the drug for an average of approximately 15 years and had been abstinent for a period of two to four months. Results clearly demonstrated that MA users displayed significantly greater Stroop reaction time interference than controls.

In another study, Salo et al. (2007) used the computer-based Stroop Test to measure the abilities of 36 currently abstinent MA users and 16 non-substance using control participants to direct their attention to specific tasks while ignoring distracting variables. Although control participants were matched for age, they had received more
education and had higher estimated pre-morbid intelligence than MA users. Participants in the MA group had been abstinent for a minimum of three weeks and had previously used MA for an average of 12 years. Results showed that MA users demonstrated reduced attentional control compared to non-drug users. The study further examined the relationship between Stroop performance and brain metabolite levels in frontostriatal regions using proton magnetic resonance spectroscopy. MA users exhibited abnormally low $N$-acetyl-aspartate creatine and elevated cho $N$-acetyl-aspartate levels in the anterior cingulate cortex compared to controls, which in turn, was related to significantly worse attentional control on the Stroop Task.

Salo, Nordahl, Galloway, et al. (2009) found a link between chronic MA use and deficits in behavioural control when comparing the performance of three groups: recently abstinent MA users (n = 38), MA users who had remained abstinent for greater than one year (n = 27), and non-MA using controls (n = 33) on a Stroop reaction time task. Specifically, recently abstinent MA users demonstrated greater Stroop reaction time interference compared to the long-term abstinent MA users and controls. The authors concluded that the cognitive dysfunction (i.e., increased disinhibition) associated with MA use may improve following longer periods of abstinence but is clearly evident at in the short term (Salo, Nordahl, Galloway, et al., 2009).

Expanding on this study, some of the same researchers utilised both a behavioural measure of inhibition and neuroimaging techniques to examine the effects of MA use on behavioural control (Salo, Nordahl, Buonocore, et al., 2009). This study investigated the relationship between behavioural regulation or control and working memory deficits in 37 currently abstinent MA users and 17 non-MA using, age-matched controls. Using the Stroop selective attention task to measure cognitive inhibition and diffusion tensor imaging to obtain indices of working memory microstructure within the
callosal genu and splenium regions of the brain, the researchers found that MA users exhibited greater Stroop reaction time interference (a measure of cognitive inhibition/control) than controls. Further, results from diffusion tensor imaging indices showed lower fractional anisotropy within the frontal brain regions of MA users. This finding was consistent with another study which showed that the same brain abnormality led to worse performance on the Wisconsin Card Sorting Test, another test of frontal executive function (Chung et al., 2007).

Many of the same researchers went on to perform another study where they examined the patterns of behavioural control relevant to MA addicts (Salo, Ursu, et al., 2009). This study compared patterns of behavioural inhibition among 12 lifetime dependent MA users (abstinent for at least three weeks) and 16 age matched non-MA using controls (Salo, Ursu, et al., 2009). Neurological patterns of behavioural control were examined using fMRI. A single-trial Stroop Task was used to compare MA users with controls using measures of error rates, reaction time conflict and the level of trial-to-trial adjustments observed following incongruent trials. It was found that on a series of trials measuring the ability to use exposure to conflict situations to regulate behaviour, MA users demonstrated reduced reaction time adjustments and reduced activation in the right prefrontal cortex compared with controls. This finding is consistent with an emerging body of literature demonstrating that MA use and chronic use results in impaired inhibitory control.

Recently, Simon, Dean, Cordova, Monterosso and London (2010) used the Stroop Task as part of a larger cognitive test battery to examine the neuropsychological functioning of 27 recently abstinent (four to nine days) MA users and 28 non-MA using control participants. The two groups were matched for age, gender and estimated premorbid IQ, but participants in the MA group had significantly fewer years of
education than controls and more MA participants smoked cigarettes. The battery assessed five broad cognitive domains including attention and processing speed, learning and memory, working memory, timed executive functioning and untimed executive functioning. While results revealed a trend effect showing that MA users performed worse than controls across all five cognitive domains, the attention and processing speed domain was the only one to reach statistical significance. Participants in the MA group performed significantly worse on the Stroop colours subtest, although this effect was reduced to non-significance when education was entered as a covariate. Notably, a subsample of participants in the MA group \( (n = 18) \) who agreed to remain abstinent for a period of one month following initial testing, and 21 participants in the control group were reassessed using the same test battery. Although results demonstrated slightly greater performance in MA users than controls across the five cognitive domains, the effects were non-significant. These findings suggested that while cessation of MA use resulted in improved cognitive functioning, including increased inhibition, these improvements did not appear rapidly.

In contrast to the large number of studies reviewed above which have used the Stroop Task (Stroop, 1935) to measure behavioural inhibition in MA users, there appears to be only one study (Monterosso et al., 2005) which has used the STOP-IT (Verbruggen, Logan, & Stevens, 2008). The STOP-IT directly measures an individual’s ability to inhibit or suppress a pre-potent behavioural response in the presence of conflicting GO and STOP signals. More specifically, the task engages participants in such a way that they are required to respond to GO signals on some trials and to inhibit their response on other trials when the GO signal is followed by a STOP signal. The STOP signal is presented occasionally and at unpredictable intervals (i.e., a shape on the screen without any sound = GO signal and push response key; a shape on the screen
followed by a beep sound = STOP signal and withhold response). The task provides a measure of the time that is required to inhibit a response, once it has already commenced (Stop Signal Reaction Time; SSRT). The STOP-IT is unique because of its’ direct assessment of the ability to inhibit an automatic behavioural response, and some have argued that it provides a more direct assessment of inhibitory control than other tests which claim to assess inhibition (Monterosso et al., 2005; Quay, 1997).

Further, research has shown that the task is sensitive to specific elements of inhibition that are implicated in self-control in populations with impulse or self-control disorders, including Attention Deficit Hyperactivity Disorder (Oosterlaan & Sergeant, 1996; Schachar, Tannock, & Marriott, 1995).

Using the STOP-IT, Monterosso et al. (2005) compared response inhibition in 11 MA users who had been abstinent for five to seven days, with two control groups consisting of 14 non-MA users who used tobacco and 29 non-MA and non-tobacco users. The decision to recruit control participants who used tobacco and others who did not was made on the grounds that cigarette smoking is common among MA users. Results showed that MA users demonstrated significantly slower response inhibition relative to controls, as evidenced by their SSRT scores (which indicate the latency time to inhibit an initiated motor response). In contrast, the MA group did not perform differently to either comparison control group on ‘go’ trial reaction time or the number of discrimination errors, which measure motor speed and decision-processes, respectively. These results indicated that in this particular study, MA use was associated with a specific deficit in inhibiting a pre-potent response. Consistent with the findings of an earlier study (see Thompson et al., 2004), Monterosso et al. (2005) also found that MA users displayed a volume reduction in the inferior frontal gyrus, a
region of the brain that is associated with response inhibition deficits in neurological patients (Aron, Fletcher, Bullmore, Sahakian, & Robbins, 2003).

Several other studies, however, have used the STOP-IT to demonstrate a relationship between cocaine, a similar stimulant to MA, and impaired inhibitory control. In one of the earliest studies, Fillmore et al. (2002) examined the effects of low dose oral cocaine on the ability to inhibit behaviour in eight individuals with a history of cocaine abuse. Performance on the STOP-IT was tested just before and one hour after receiving a randomised, double-blind administration of 0 mg (placebo), 50 mg, 100 mg, and 150 mg of oral cocaine HCl. Results showed the cocaine significantly reduced participants’ ability to exercise inhibitory control over behavioural impulses. There was no significant dose-response effect found which was most likely associated with the low doses of cocaine utilised in the study. Findings further indicated that cocaine did not affect the speed and accuracy with which participants were able to execute a response. Overall, findings showed that low, acute doses of cocaine can impair the ability to inhibit behaviour at doses that do not affect the ability to respond.

Extending on this study, Fillmore and Rush (2002) compared the inhibitory control of cocaine users with age-matched non-cocaine using controls. Again, the ability to inhibit and execute behavioural responses was measured with the STOP-IT. Compared with controls, cocaine users were found to perform significantly worse on the STOP-IT. Cocaine users found it more difficult to inhibit their responses and required more time to inhibit their responses as estimated by their SSRT scores. The two groups did not differ, however, in their ability to execute responses, and displayed similar reaction times and response accuracy scores.

Similarly, in another more recent study (Li et al., 2006) cocaine users were found to display increased disinhibition. Eighteen abstinent cocaine dependent
participants were compared with 41 age and education matched control participants on the STOP-IT. Given the authors’ view that performance on the STOP-IT may involve processes during sensorimotor transformation that are distinct from inhibitory control and yet can directly impact response inhibition performance, they also measured response readiness and performance monitoring. Thus, SSRT scores were calculated as the index of response inhibition function (a measure of how long it takes for the stop signal to be processed so a response can be stopped), fore-period effect scores were calculated as an index of response readiness, and post-signal slowing effect scores were calculated to measure performance monitoring. It was hypothesised that greater response readiness (as measured by fore-period scores) to the GO signal would increase SSRT, whereas greater performance monitoring elicited by the STOP signal would decrease SSRT. Findings indicated that relative to controls, cocaine users demonstrated increased SSRT scores and decreased post-signal slowing scores. The authors suggested that reduced performance monitoring may be a vital cognitive mechanism underlying the increased disinhibition observed among cocaine users.

In one notable study, Kjome et al. (2010) compared 66 dependent cocaine users with 20 non-drug abusing control participants on a questionnaire measure of impulsivity (the BIS-11) and laboratory tasks measuring both behavioural inhibition (the Immediate Memory Task) and decision making (the Iowa Gambling Task). In extending the researcher further, correlations between the questionnaire measure of impulsivity and the behavioural tasks were also examined. Results indicated that cocaine-dependent users reported significantly higher total scores and subscale scores on the BIS than controls. Further, the cocaine group had higher commission errors on the memory task compared to controls, which equated to a poorer ability to inhibit behavioural responses among cocaine users. Performance on the Iowa Gambling Task also differed...
significantly between the two groups, with cocaine users shifting to advantageous decks of cards more slowly and less frequently than controls. Upon reviewing the interrelationships between measures, it was found that there was a significant relationship between performance on the memory task and self-reported scores on the BIS-11. In contrast, performance on the gambling task was not related to responses on the BIS-11.

To summarise, there is a growing body of evidence supported by neuroimaging studies and laboratory tasks which has clearly shown that chronic MA use is associated with neuropsychological deficits, with MA users showing impairments of frontal lobe functions that are thought to be important in the control and regulation of behaviour of these individuals. Research has shown that a primary function of this brain region is to control behaviour via inhibitory processes that normally serve to regulate behaviour by suppressing or terminating prepotent (i.e., environmentally-triggered) responses. The same deficits have been observed in cocaine users. Given the substantial size of this literature base, a detailed examination of several key studies has been reviewed above. A more thorough list of studies which have examined behavioural inhibition in MA and cocaine abusers is summarised in Table 1. An interesting question that remains unanswered is whether the impairments in inhibitory control that MA users experience, in turn, impact on their propensity to engage in a range of poorly controlled behaviours, including possible displays of aggression.
Table 1.

**Summary of Key Studies Investigating Behavioural Inhibition in Methamphetamine and Cocaine Users**

<table>
<thead>
<tr>
<th>Reference</th>
<th>Participants</th>
<th>Behavioural Task Used</th>
<th>Results</th>
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</thead>
<tbody>
<tr>
<td>Kjome et al. (2010)</td>
<td>66 cocaine dependent subjects (56 male, 10 female) 20 controls</td>
<td>Iowa Gambling Task Immediate and Delayed Memory Task (a version of the Continuous Performance Test)</td>
<td>Cocaine dependent participants made more disadvantageous choices on the IGT, made more commission errors on the IMT and reported higher scores on self-report measures of impulsivity.</td>
</tr>
<tr>
<td>Simon et al. (2010)</td>
<td>27 recently abstinent MA users 28 non-substance abusing controls match on age, education, and premorbid IQ</td>
<td>A cognitive battery assessing 5 domains including attention/processing speed, learning/memory, working memory, timed and untimed executive functioning</td>
<td>Compared to controls, MA dependent individuals performed significantly worse on a test of processing speed and lower global battery scores. A subsample of participants (18 MA users &amp; 21 controls) was retested following MA users abstaining for one month. Results revealed that although MA users showed greater improvement in cognitive performance over the entire test battery compared to controls, this effect was not statistically significant.</td>
</tr>
<tr>
<td>Salo, Nordahl, Buonocore, et al. (2009)</td>
<td>37 abstinent MA users 17 non-substance abusing controls</td>
<td>Stroop Task Diffusion Tensor Imaging (DTI)</td>
<td>MA users displayed greater Stroop reaction time interference and reduced cognitive control.</td>
</tr>
<tr>
<td>Salo, Nordahl, Galloway, et al. (2009)</td>
<td>38 recently abstinent MA users (3 weeks to 6 months) 27 long-term abstinent MA users (&gt; than 1 year) 33 non-substance abusing controls</td>
<td>Stroop Task</td>
<td>Recently abstinent MA users demonstrated greater Stroop reaction time interference compared with the other two groups. Longer-term abstinent MA users did not perform differently to controls. Stroop reaction time interference correlated positively with duration of drug use and abstinence.</td>
</tr>
<tr>
<td>Salo, Ursu, et al. (2009)</td>
<td>12 MA dependent users 16 age matched, non-substance abusing controls</td>
<td>Stroop Task fMRI</td>
<td>MA users exhibited reduced reaction time adjustments and reduced activation in the right prefrontal cortex on conditions that measured the ability to use exposure to conflict situations to regulate behaviour.</td>
</tr>
<tr>
<td>Reference</td>
<td>Participants</td>
<td>Behavioural Task Used</td>
<td>Results</td>
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<tr>
<td>Paulus et al. (2008)</td>
<td>12 non-dependent MA users (used stimulants at least twice in past 6 months) 12 age and education matched controls</td>
<td>Two-Choice Prediction Task fMRI</td>
<td>Stimulant users displayed less strategy adjustment to different error rates (i.e., less likely to stay with winning responses and to shift away from losing responses) and exhibited different activation patterns as a function of error rate in the left insular and bilateral dorsolateral prefrontal cortex.</td>
</tr>
<tr>
<td>Leland, Arce, Feinstein, &amp; Paulus (2006)</td>
<td>11 non-dependent stimulant users (type of stimulant not specified; had used stimulants on 2 to 50 occasions) 11 aged and education matched controls</td>
<td>Card Prediction Task fMRI</td>
<td>Non-dependent stimulant users displayed enhanced striatal activation response to uncertainty such that they had longer reaction times and made more incorrect card predictions on uncertain trials.</td>
</tr>
<tr>
<td>Leland &amp; Paulus (2005)</td>
<td>19 non-dependent stimulant using university students (stimulants including cocaine, amphetamine, methamphetamine &amp; methylphenidate) 108 non-stimulant using matched controls</td>
<td>Risky Gains Task (RGT)</td>
<td>Overall, stimulant-users made more risky responses than controls ($p &lt; .02$), however they displayed the same inhibition effect of punishment on next-trial risky responding. Thus, stimulant users showed increased risk taking but were not less sensitive to punishment than controls. Risk-taking in the task correlated with measures of sensation-seeking and impulsivity, but not other personality measures, anxiety, or tendency toward alcohol use disorders. Given that the RGT required participants to select from a sequence of individual options presented according to a fixed schedule, rather than allowing deliberation between simultaneously available options, this task may have modeled a different sort of risk-taking than other tasks.</td>
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<tr>
<td>Monterosso, Aron, Cordova, Xu, &amp; London (2005)</td>
<td>11 recently abstinent (5-7 days) MA users 14 non-MA using, tobacco using controls 29 non-MA using, non-tobacco using controls All groups equal on education and IQ</td>
<td>Stop-Signal Paradigm</td>
<td>Stop-signal reaction time (SSRT) scores (a measure of the latency to inhibit an initiated motor response) were significantly longer for MA users than for either control group ($p’s &lt; .01$). In contrast, MA users did not differ from either group on Go trial reaction time (RT) or number of discrimination errors, which reflect motor speed and decision-processes, respectively. This study showed that MA use was specifically associated with a specific deficit in inhibiting a pre-potent response.</td>
</tr>
<tr>
<td>Salo et al. (2005)</td>
<td>34 recently abstinent MA users 20 age matched, non-substance abusing controls</td>
<td>A Letter-Number Naming Task</td>
<td>Compared to control participants, MA users demonstrated a greater number of errors on trials that required inhibition of distracting information.</td>
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<tr>
<td>Reference</td>
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<td>Behavioural Task Used</td>
<td>Results</td>
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<tr>
<td>Fillmore &amp; Rush (2002)</td>
<td>22 chronic cocaine users 22 age matched controls</td>
<td>Stop-Signal Task</td>
<td>Cocaine users exhibited significantly poorer ability to inhibit behavioural responses and required more time to inhibit responses to stop-signals. The groups did not differ in their ability to execute responses as measured by their speed and accuracy of responses to go-signals.</td>
</tr>
<tr>
<td>Salo et al. (2002)</td>
<td>8 male, recently abstinent MA dependent abusers 12 male non-drug abusing controls</td>
<td>Stroop Test</td>
<td>MA dependent males showed significantly greater reaction time interference despite intact priming.</td>
</tr>
<tr>
<td>Paulus et al. (2002)</td>
<td>10 MA dependent subjects 10 age and education matched controls</td>
<td>Two-Choice Prediction Task Two-Choice Response Task fMRI</td>
<td>MA dependent participants exhibited fundamental cognitive deficits during decision-making that are consistent with both orbitofrontal and dorsolateral prefrontal dysfunction.</td>
</tr>
<tr>
<td>Simon et al. (2002)</td>
<td>40 current MA users 40 current cocaine users 80 age matched controls</td>
<td>A Cognitive Battery of tests measuring a range of neurobehavioural domains, including the Stroop Task</td>
<td>MA users and cocaine users were significantly more impaired than non-using controls on several cognitive measures, including the Stroop task.</td>
</tr>
<tr>
<td>Simon et al. (2000)</td>
<td>65 regular MA users 65 controls</td>
<td>A Cognitive Battery of tests measuring a range of neurobehavioural domains, including the Stroop Task</td>
<td>MA users were significantly more impaired on recall tasks, digit symbol, Stroop color words, and Trail Making B, but scores fell within the normal ranges on the other measures.</td>
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</table>
**Self-report studies.** The third body of research supporting a link between MA use, impulsivity and reduced behavioural control is embedded in studies which have found elevated scores on self-report measures that measure constructs related to impulsivity, behavioural control and anti-social traits (Clark et al., 2006; Coffey et al., 2003; Ersche et al., 2008; Moeller et al., 2004; Semple, Zians, et al., 2005). Certainly, theoretical models of substance use have highlighted the potential role of impulsivity as a shared vulnerability to a set of behaviours that have been classified as Antisocial Personality Disorder (*DSM-IV-TR*; APA, 2000). For instance, Verheul (Verheul, 2001; Verheul, Van Den Brink, & Geerlings, 1999) proposed a three-pathway model in explaining the co-morbidity between personality disorders and substance abuse. Of particular interest to the current study are the behavioural disinhibition and the reward sensitivity pathways. According to the model, individuals who score high on behavioural disinhibition (including traits of antisociability and rash impulsiveness), and experience deficient socialisation, are likely to abuse stimulants. The model suggests that these individuals have lower thresholds for deviant behaviours, including substance misuse. This pathway is thought to account for the relationship between antisocial personality traits and substance misuse. In the reward sensitivity pathway, individuals who are more reward sensitive and score higher on measures of novelty seeking are presumably motivated by the positive reinforcing properties of substances. The model proposes that people with a histrionic, antisocial or narcissistic personality disorder, might develop a regular pattern of drug use along this pathway.

The high co-morbidity that is observed between substance use disorders and Axis II disorders from the impulse control spectrum provides considerable support for the above. Prevalence rates are four times greater in individuals with substance abuse than those without, with as many as 73% meeting *DSM-IV-TR* (APA, 2000) criteria for
an Axis II disorder (Bowden-Jones et al., 2004; Brooner, King, Kidorf, Schmidt, & Bigelow, 1997; Dawe & Loxton, 2004; Todd et al., 2004; Verheul, 2001). Conduct Disorder and Antisocial Personality Disorder are most commonly observed and represent a pervasive pattern of disregard for, and violation of, the rights of others that begins in early childhood or early adolescence and continues into adulthood (DSM-IV-TR; APA, 2000). Further, these two antisocial disorders are commonly associated with aggressive and violent behaviour in adulthood (Buitelaar, Montgomery, & van Zwieten-Boot, 2003; Hodgins, Cree, Alderton, & Mak, 2008; Odgers et al., 2007). Indeed, particularly high rates of Antisocial Personality Disorder and comorbid substance use disorders are observed among prison populations. One study found that among 320 recently incarcerated offenders, 113 (35.3%) met diagnostic criteria for Antisocial Personality Disorder (Black, Gunter, Loveless, Allen, & Sieleni, 2010). Further, an astounding 98.2% of those individuals with Antisocial Personality Disorder also had a substance use disorder. Unfortunately the researchers did not specify the type of substance abused and so prevalence rates for MA use are unattainable. Other studies have examined comorbidity rates between Antisocial Personality Disorder and opiate users with prevalence rates of between 25% and 44% being reported (Brooner et al., 1997; Darke, Hall, & Swift, 1994), and almost two thirds for injecting heroin users (Brooner, Bigelow, Strain, & Schmidt, 1990; Mills, Teeson, Darke, Ross, & Lynskey, 2004). A large number of prison inmates dependent on opiates (75%) have also met criteria for Antisocial Personality Disorder (Darke, Kaye, & Finlay-Jones, 1998).

Despite the large body of literature concerning personality disorders and opiate abuse, there has been relatively little investigation of personality disorders in individuals who use MA. Nevertheless, it is highly unlikely that the co-morbidity rates would differ markedly between MA and heroin users (Weatherburn, Jones, Freeman, &
Makkai, 2003. In Australia, demographic characteristics of MA users are similar to previous studies of heroin users as many MA users shifted from heroin to MA in the recent ‘heroin drought’. Whether differences emerge as a consequence of MA use (i.e., greater propensity for violence and impulsiveness) is not yet clear although there is good evidence that psychosis and subclinical symptoms of psychosis are more prevalent in MA users. A recent study by Darke et al. (2010) compared rates of violent crime among regular MA users and opiate users. While there were no significant differences in rates of lifetime violence and offending between the two groups, MA users were significantly more likely than opiate users to have committed violence in the past 12 months. Specifically, 50% of MA users had committed a violent act in the preceding year compared to 30% of opiate users. In one of the few studies focusing specifically on comorbidity between MA use and Antisocial Personality Disorder, Chen et al., (2003) reported a prevalence rate of 21% among a large group of injecting MA users. Further, higher rates of Antisocial Personality Disorder were found among MA users who had experienced MA-induced psychosis (33%) compared with MA users who had not experienced psychosis (13%, Chen et al., 2003).

Thus, it is possible that the hostility observed among substance users may reflect some broader personality dimension in which behaviour tends to be poorly regulated (i.e., high levels of impulsiveness and behavioral disinhibition, poor regulation of negative and hostile feelings and behaviours), and may predate substance use and/or increase as a result of subsequent MA use. One striking finding from the personality literature is that individuals with Antisocial Personality Disorder have not only been shown to score higher on measures of impulsiveness, but also perform on a series of behavioural tasks in a manner that is consistent with a general model of behavioural disinhibition (Carver & White, 1994; MacAndrew & Steele, 1991; Quay, 1988).
Despite the large number of reviewed studies which have investigated the relationship between MA and disinhibition, an important research question that does not appear to have been considered to date is whether those individuals with frontocortical dysfunction associated with chronic MA use are unable to inhibit a range of behavioural tendencies – including aggression and hostility. It is possible that the hostility and aggression observed among MA users may in fact reflect a behavioural tendency to act rashly and without consideration across many different settings, with anger provoking situations being but a single example.

The Relationship between Methamphetamine Use and Psychotic Symptoms

It is well established that MA-induced psychosis may occur following either prolonged use of the stimulant or following binge use (Leamon et al., 2010; Mahoney et al., 2008; McKetin, McLaren, Lubman, et al., 2006). The symptom profile is similar to that found in other non-drug induced psychoses, particularly the paranoid type. The rate of psychosis is higher among psychostimulant users than among the general population, even after adjusting for history of psychotic disorders (Buffenstein, Heaster, & Ko, 1999; D. Harris & Batki, 2000; McKetin, McLaren, Lubman, et al., 2006; Srisurapanont et al., 2003). Further, higher rates of psychosis have also been reported following MA use than after cocaine use (King & Ellinwood, 1992). The prevalence of psychotic symptoms among MA users is reported to be between 13% and 52%, depending of the measure of psychosis (Farrell et al., 2002; Hall et al., 1996; McKetin, McLaren, Lubman, et al., 2006). According to Dean and Whyte (2004), while MA use initially results in hypervigilance and euphoria, these effects gradually give rise to auditory, visual and tactile illusions, hallucinations, paranoia and delusions. Stimulant-induced psychosis generally occurs during use or withdrawal and abates within hours or days after discontinuation of MA use. Nevertheless, in a smaller proportion of individuals,
psychotic symptoms are protracted, especially in those with a predisposition to psychotic symptoms, including schizotypal or schizoid traits and family histories of psychotic disorders (Chen et al., 2003; Chen et al., 2005; Dawe & Mc Ketin, 2004).

The effects of MA have been extensively studied in human clinical populations. Curran, Byrappa, and McBride (2004) conducted a review of 54 studies investigating the relationship between stimulant use among humans and the development of psychotic symptoms. They concluded that there is clear evidence that irrespective of the individual’s mental state, a large enough dose of a stimulant drug can produce a brief psychotic reaction, usually lasting several hours in the majority of individuals.

A study of regular intravenous drug users in Sweden found that 80% of those who had used MA had experienced at least one psychotic episode (Kall, 1997). Another study examined the mental health among 445 MA users who were recruited from a detention centre and a psychiatric hospital in Taipei (Chen et al., 2003). The overall sample was divided into two smaller groups, which consisted of 174 individuals who had experienced MA-induced psychosis and 261 who reported the absence of psychotic symptoms. Of those with MA-induced psychosis, 85% reported auditory hallucinations, 46% reported visual hallucinations and 12% reported tactile hallucinations. Further, common themes of delusions included persecution (71%), delusions of reference (63%) and the ability to mind read (40%). Those who had experienced psychosis began their use of MA at a younger age and had used larger amounts of MA – although it is not entirely clear whether the researchers were referring to larger quantities per occasion or more regular use (Chen et al., 2003).

A recent survey of 310 regular MA users found higher rates of psychosis than typically found in general population studies (Mc Ketin et al., 2005). Twenty-two percent of users, irrespective of mental health history, had experienced a clinically
significant symptom of psychosis in the year prior. While the majority of regular MA users did not seek hospital treatment and experienced brief symptoms of psychosis that dissipated within three hours, 11% did require hospital assistance and tended to experience more severe and persistent symptoms.

Given that a majority of the research on MA use and psychosis has focused on regular or dependent MA users (Chen et al., 2003; Matsumoto et al., 2002; McKetin, McLaren, Lubman, et al., 2006), a recent study sought to determine whether a similar association is observed in recreational MA users. McKetin, Hickey, Devlin, and Lawrence (2010) conducted a cross-sectional survey of 157 individuals who reported using MA for recreational purposes only. Most individuals reported using MA monthly or less often (83%), while a much smaller proportion reported weekly use (12%). Only 5% of the sample reported using MA more often than once a week. Results indicated that increases in the frequency of recreational MA use inflated the risk of individuals experiencing two or more psychotic symptoms from 9% to 21% in the past year. Further, this effect remained constant after removing individuals with a history of psychotic disorder (n = 16) from analyses and adjusting for poly-drug use (McKetin et al., 2010).

**Methamphetamine use, psychosis and behavioural sensitisation.** Animal based studies are again useful in assisting our understanding of an interesting and concerning phenomenon that sometimes accompanies MA-induced psychosis in humans. More specifically, many studies have shown that animals repeatedly exposed to MA become behaviourally sensitized to the drug. Behavioural sensitisation is a reverse tolerance-like phenomenon which involves progressive quantitative and qualitative changes in behaviour in response to repeated doses of MA. Multiple doses of MA increase sensitisation in susceptibility to drug-induced stereotypy. Sensitisation
AGGRESSION IN METHAMPHETAMINE USERS

has been observed among rats following repeated doses of MA. Ujike (2002) reported that psychostimulants, such as MA, induced ambulation and hyperlocomotion in rodents. Repeated stimulant administration led to gradual increases in hyperlocomotion, instead of tolerance developing, and finally resulted in abnormal stereotypy with repetitive head movement, licking and gnawing. Once the animals had developed an increased sensitivity to stimulants, subsequent doses of the drug resulted in intense stereotypy, even following lengthy abstinence periods of up to one year (Ujike, 2002). Indeed, one of the key characteristics of behavioural sensitisation is how long lasting it is. Rats have a brief life span, but once sensitisation has developed, it remains present until death. Another major characteristic of behavioural sensitisation is that other drugs and stressors can trigger stereotypy. Specifically, animals that have been sensitised to MA also show an enhanced response to other classes of drugs such as nicotine, cannabis and opiates, as well as a hyper-response to environmental and physiological stress (Shaham, Erg, & Stewart, 2000; Ujike, 2002). For instance, rats that were either restrained, or given a saline injection and placed into the same cage in which the stimulant had previously been administered, displayed the same abnormal behaviour (Antelman, Eichler, Black, & Kocan, 1980; Shaham et al., 2000). Overall, studies show that animals can become sensitised to MA over time, which can further result in cross sensitisation with other drugs and stressors.

Evidence has suggested that a similar process of sensitisation occurs in humans exposed to repeated doses of MA. Indeed, MA-induced psychosis in humans seems to have shared the same two characteristics of sensitisation in animals (Ujike, 2002). Following chronic use and repeated psychotic episodes, a psychosis may be triggered by lower doses of MA (Sato, Numachi, & Hamamura, 1992). In an early study, Sato, Chen, Akiyama, and Otsuki (1983) found that lower doses of amphetamine than had
been used previously precipitated a psychotic episode in individuals with a history of long lasting psychosis, despite several years of abstinence. Further, sensitisation may occur within a short period of use. In a double-blind, randomised study of non-drug using volunteers, two identical doses of amphetamines were administered 48 hours apart (Strakowski, Sax, Setters, & Keck, 1996). Symptoms (e.g., energy level, mood, rate and amount of speech, eye blink rates) were measured hourly for five hours following drug administration. Both amphetamine doses produced an increase in mood, energy, speech rate and eye-blink rate compared to placebo. Critically, the second dose of amphetamine produced a greater response than the first, a result that was interpreted to reflect behavioural sensitisation (Strakowski et al., 1996). This study demonstrated that similar to animals, human beings might also develop sensitisation following repeated stimulant administration.

Interestingly, some research has shown that occasionally, stress alone, has induced recurring episodes of psychotic symptoms among MA users (Salo et al., 2002; Yui, Goto, Ikemoto, & Ishiguro, 1997, 2000). Sato et al. (1983) found that a male patient involved in their study experienced recurrent paranoid delusions and hallucinations two years after his first MA-induced psychotic episode had resolved and MA use had ceased. This individual reported high levels of psychological stress, which in turn, were thought to induce the recurrent psychotic symptoms. More recently, Yui et al. (1997) examined clinical characteristics and the processes that triggered a recurrence of psychotic symptoms in 28 incarcerated women and also examined peripheral monoamine neurotransmitter function in 12 of these women. To ensure these women had not used MA during the times in which they experienced recurrent psychotic symptoms, their venous plasma was also screened at this time. Results confirmed that MA was not related to the recurrence of symptoms, but rather, mild
psychosocial stressors, particularly having a mild fear of other people, appeared to trigger psychotic symptoms.

These results were replicated in another study by Yui, Ishiguro, Goto, and Ikemoto (1998) which again investigated factors that triggered spontaneous recurrences of psychotic symptoms among individuals (n = 125) with a history of MA-induced psychosis. Just over one-third of the sample (41 participants) was found to have experienced recurrent psychotic symptoms, particularly vivid auditory and visual hallucinations and paranoid delusions. Interestingly, all of the participants in this subsample reported having previously experienced actual threatening events or frightening paranoid hallucinations during previous MA use. The authors suggested that in light of the fact these individuals were exposed to highly stressful events at the time they were using MA, and subsequently experienced MA-induced psychosis, they may have developed an increased sensitivity to stress. Therefore, MA use, combined with threatening experiences, may increase the sensitivity of individuals to psychosocial stressors (Yui et al., 1998). For the individuals in this study, stress alone appeared to provide the necessary conditions to induce recurring psychotic episodes. These results have since been replicated (Yui et al., 2000).

Overall, there is a clear link between MA use and psychosis. High doses and binge use appear to be the patterns of use most strongly associated with drug induced psychosis. Further, with repeated use of MA, sensitisation to the psychosis inducing aspect of the drug occurs across species. This results in an increase in the risk of recurrent episodes of psychosis. In some individuals with a history of MA-induced psychosis, exposure to serious psychosocial stressors alone might be sufficient to induce recurrent episodes of psychosis.
**Methamphetamine use and subclinical symptoms of psychosis.** While a substantial number of individuals who use MA do not develop MA-induced psychosis, many report a range of subclinical symptoms such as suspiciousness, paranoia, and disordered thought processes (Dawe & McKetin, 2004; Leamon et al., 2010; McKetin, McLaren, Lubman, et al., 2006). According to the Australian Institute of Health and Welfare (2008), almost two out of every five individuals who used an illicit drug, including MA, in the past month reported high or very high levels of psychological problems. In the following section, studies in which subclinical symptoms of psychosis have been investigated will be described. However, in most of these studies other psychological symptoms such as mood difficulties, aggression and hostility have also been investigated.

In an Australian study, Hall et al. (1996) found that three quarters of injecting MA users \((n = 301)\) reported symptoms of depression (79%) and anxiety (76%). Half of the sample reported that these symptoms predated MA use (48% reported anxiety, 62% reported depression). A high proportion of users consistently reported hallucinations (61%) and paranoia (59%), the onset of which generally followed MA use. Finally, following first use of MA, individuals reported greater occurrences of violence (44%). Thus, while some symptoms predated MA use, others, including violence, increased following initial MA use (39% versus 44%).

Similar findings were reported in a review of studies investigating the associated harms of regular psychostimulant use (Kamieniecki, Vincent, Allsop, & Lintzeris, 1998). High rates of psychological impairment were reported with rates ranging from 51% to 92% for depression, 60% to 76% for anxiety, 28% to 67% for hallucinations, 33% to 78% for paranoid ideation and 17% to 72% for aggression and violence. For example, Klee, Carnwath, Merrill, and Morris (1995) found that in a sample of
amphetamine users currently in treatment \((n=39)\), 83% of the sample reported irritability, 78% reported paranoid delusion, 72% reported aggression, and 67% reported hallucinations in the three months prior to treatment.

Among a sample of regular MA users in Australia, one in five reported a psychiatric problem (McKetin et al., 2005). Commonly reported problems included aggression, agitation, depression, poor motivation, impaired concentration and memory, as well as symptoms of psychosis. During the past year, 23% of the sample reported a ‘clinically significant’ symptom of psychosis, while 52% experienced sub-clinical symptoms and only 25% reported being symptom free. Twelve percent of regular MA users reported clinically significant levels of suspiciousness, while 47% reported milder symptoms. Delusions were reported by 7% of the sample, with a further 26% reporting sub-clinical delusional beliefs. Seventeen percent of users reported hallucinations, while an additional 30% reported milder symptoms.

Similarly high rates of psychological problems have been reported by Baker and colleagues (Baker et al., 2004). Almost half of regular MA users reported that they had been diagnosed or treated for a mental health problem in their lifetime. Almost two-thirds (62.9%) of individuals reported that they experienced the problem/s after commencing regular MA use. Research clearly indicates that regular MA use induces and exacerbates many psychological problems including sub-clinical symptoms of psychosis.

One study (Sommers et al., 2006) found that out of 106 MA dependent users, 37.7% reported experiencing hallucinations and 62.3% reported high levels of paranoia as a result of their MA use. An interesting and important finding from this study was that the most common form of paranoia reported by MA users related to fear of others wanting to harm or threaten the individual. The authors highlighted the particular
relevance of this symptom to violence since previous research has indicated that the risk of interpersonal violence increases when an individual fears personal harm (Link & Stueve, 1998; Sommers et al., 2006). A large portion of the sample also reported depression (36.8%) and irritability (79.3%). Findings indicated that individuals who reported the greatest number of psychological and social problems also reported the highest levels of MA use. It is worth noting that approximately 69% of participants used MA daily, which indicates that the sample was most likely to be dependent on MA. Similar findings were reported by McKetin, et al. (2005) who found that dependent MA users were three times more likely to experience psychotic symptoms than non-dependent users, after controlling for premorbid schizophrenia. A strong association was found between frequency of use and severity of dependence, such that using MA more than twice a week increased the risk of psychosis. Thirty-one percent of individuals, who were either dependent on MA or using more than twice weekly, reported psychosis in the preceding year (McKetin et al., 2005).

Descriptive studies of the clinical profile of MA users with psychosis have shown a pattern of high scores on measures of positive symptoms and relatively low scores on measures of negative symptoms (Chen et al., 2003; Iwanami et al., 1994). Research with non-MA-induced psychotic patients has shown that this profile may represent an additional risk for increased aggression and violence. For example, Foley et al. (2007) reported that among 29% of first-episode psychosis patients \((n = 157)\), positive symptoms scores were most predictive of violence. Similarly, in a large study of schizophrenics \((n = 1,410)\), Swanson et al. (2006) found that positive symptoms of psychosis increased the risk for minor (defined as assault without injury or use of weapon) and serious (defined as assault resulting in injury and/or involvement of a lethal weapon) acts of violence. Negative symptoms of psychosis, on the other hand,
reduced the risk of serious violence. The six-month prevalence of violence in the total sample was 19.1%. In sum, it is possible that the distinct symptom profile observed among MA users who experience psychosis may be associated with increased aggression (Dawe, Geppert, Occhipinti, & Kingwell, 2010).

Overall, there is a compelling body of evidence linking MA use and increased psychotic symptoms, particularly positive symptoms of psychosis. In particular, paranoid delusions have been strongly associated with MA use. This is of particular concern since paranoid delusions have been identified as a key feature of mental state that is associated with aggression (Junginger, 1996; Nestor, Haycock, & Doiron, 1995). Very simply, individuals who believe they are at risk of being harmed by others are more likely to act on these fears by behaving aggressively.

**The synergistic effects of methamphetamine use and psychosis on aggression.** Studies have demonstrated that a dual diagnosis of substance use disorder and psychotic disorder is associated with a greater risk for aggression and violence (Cuffel, Shumway, Chouljioa, & McDonald, 1994; Ries et al., 2000; Soyka, 2000). For instance, Scott et al. (1998) compared aggressive behaviour in a group of individuals with comorbid substance use and psychotic disorders ($n = 27$) with another group of individuals with psychosis only ($n = 65$). Results showed that individuals with a dual diagnosis were significantly more likely to display aggressive and hostile behaviour (as measured by the Health of the Nation Outcome Scale), have a reported history of criminal offences and a recent history of assault.

Further, Malla and Payne (2005) conducted a review of first-episode psychosis patient characteristics and found that high rates (20% to 30%) of verbal aggression and/or violent behaviour were not only associated with first-episode psychosis, but this was particularly pronounced among individuals with substance misuse.
In a different study which sampled community mental health patients \((n = 233)\), individuals diagnosed with comorbid stimulant, alcohol or cannabis abuse/dependence and a psychotic illness (schizophrenia, schizoaffective disorder, bipolar disorder, or delusional disorder) were compared on illness characteristics, including violence and self-harm (Miles et al., 2003). Violence and self-harm were measured by a purpose designed scale, although definitions were not provided. Nearly one-quarter \((n = 55)\) of the sample were identified as primary stimulant users (including cocaine, amphetamines, khat, and ketamine). Individuals with a comorbid stimulant use disorder and psychotic illness were significantly more likely than those with comorbid alcohol or cannabis use disorders to report a life-time history of violence. Despite the distinct substance related difference pertaining to violence, the groups did not differ on other factors such as utilisation of treatment services and self-harm.

**Summary**

There are several studies in which aggression and hostility have been reported in MA users. It is possible that MA use is associated with aggressive behaviour due to specific toxic effects on neural pathways mediating aggression. Some data from the animal laboratory (Crowley, 1972; Miczek & O’Donnell, 1978; Shintomi, 1975; Sokolov et al., 2004) and at least one study in humans (Sekine et al., 2006) have indicated that chronic use of MA is associated with serotonergic depletion and dysfunction, a neurotransmitter that has been found to influence aggressive behaviour.

Numerous studies have demonstrated a relationship between impulsivity, particularly reduced behavioural control and MA, and also impulsivity and aggression. Further, there are particularly high rates of antisocial behavioural tendencies in people with substance misuse, especially illicit drug use. This raises the possibility that the aggressive behaviour predates MA use and is related to a general tendency to engage in
impulsive and nonconforming behaviour, including interpersonal aggression. In addition, this tendency may in fact be exacerbated by chronic MA use as there is evidence from the animal laboratory that such use is associated with prefrontal cortical disinhibition.

Finally, there is a well established link between MA use and psychosis and subclinical symptoms of psychosis. Given that the predominant phenomenology is one of paranoid symptoms, in which people frequently believe they are being threatened or persecuted, it is reasonable to propose that the hostility and aggression observed among MA users may be related to the level of positive psychotic symptoms that an individual is currently experiencing. Furthermore, higher rates of aggression and violence are observed in psychotic populations compared to other psychiatric populations (Foley et al., 2005; Pearson et al., 1986; Tardiff et al., 1997). In brief, the combination of psychotic symptoms and MA use is likely to further enhance an individual’s aggressive behaviour.

MA use represents a significant and worldwide health problem. The perceived effect of MA on aggression is a major concern. While evidence clearly documents high levels of aggression and violence among MA users, it is unclear whether this phenomenon is a direct result of MA. The specific mechanisms through which this association operates remain unclear. It is noted that other factors (e.g., reduced behavioural inhibition and positive psychotic symptoms) likely contribute to the strength of this relationship. Research has not only demonstrated a link between MA, heightened impulsivity and reduced behavioural control, but also a link between impulsivity and aggression more generally. Similarly, strong evidence attests to a clear link between MA and psychotic symptoms, and also psychotic symptoms and aggression in their own right.
Despite research efforts to explore each of the associations mentioned above, there is a paucity of studies examining how these factors might interact, including whether they operate in synergy (i.e., when two or more variables work together in a way that produces an effect greater than the sum of their individual effects). The purpose of the first study presented in this thesis was to build on previous work which has identified increased aggression, heightened levels of impulsivity and reduced behavioural control, and psychotic symptoms as factors associated with MA use. The aim of Study 1 was to extend previous research by: (i) exploring the independent associations between MA, impulsivity and psychotic symptoms and aggression; and (ii) determining whether impulsivity and positive psychotic symptoms influence the relationship between MA and aggression. In this study, a sample of current MA users completed a range of self-report measures of hostility, impulsivity, psychotic symptoms, and MA dependence. The study thus sought to explore the influence of MA dependence, impulsivity, and positive psychotic symptoms on hostile behaviour in a group of injecting users.
CHAPTER 4

Study 1: Predictors of Aggression in Methamphetamine Users: A Cross Sectional Study

MA has been strongly associated with heightened aggression, hostility and violence in observational and experimental human studies as well as animal based laboratory research. There is, however, considerable variability in the expression of aggression and in the nature of the aggressive acts. As reviewed in the previous chapter, two factors which appear to play an etiological role in the expression of aggressive behaviour are the capacity to exercise impulse control and the presence of positive symptoms of psychosis that are associated, in particular, with reactive aggression. Difficulties with impulse control appear to be both a predisposing factor in substance use that co-occur with a range of other antisocial behaviours (Dawe & Loxton, 2004; McEllistrem, 2004; Sommers et al., 2006) and are also exacerbated by chronic use of psychomotor stimulants (Jentsch & Taylor, 1999). Psychotic-like phenomena are more clearly a consequence of psychomotor stimulant use, although there is considerable individual variability in the experience of such symptoms (Leamon et al., 2010). While a large body of literature exists in which these two factors have been independently investigated in substance use generally, it is notable that there has been no specific attempt to determine what influence each of these factors may play on the expression of aggressive behaviour in MA users. Further, it is also reasonable to propose that these two factors may act synergistically. Thus, an investigation of the relative contributions of impulsivity and positive symptoms of psychosis to hostility and aggression in MA users seems warranted.

This study was therefore undertaken to investigate the relationship between both positive psychotic symptoms and impulsivity in a group of injecting MA users. As
shown by the predicted model in Figure 1, a positive relationship was predicted between exposure to MA (indexed by severity of dependence; SDS) and hostility. It was further hypothesised that this relationship would be mediated by both impulsivity and positive symptoms. Finally, it was predicted that there would be an interaction between these variables, resulting in greater hostility in those with both an impulsive personality style and positive symptoms.
Figure 1. Hypothesised model of the influence of severity of methamphetamine dependence, impulsivity and positive symptoms on hostility in methamphetamine users.
Method

Participants

Two hundred and thirty-seven MA users were recruited from an inner city Needle Syringe Exchange Program in Brisbane, Australia. Eligibility criteria for inclusion in the study included being 18 years or older, currently using MA, identifying MA as ‘drug of choice’, and using injection as route of administration of the drug. Participants were paid $10 for their involvement in the study. Informed consent was obtained from each participant and the study was approved by the relevant ethics committees of both Griffith University and Prince Charles Hospital Health Service District.

Materials

A structured interview provided socio-demographic information, reports of daily living circumstances, family history of mental illness, treatment history of both substance use and mental illness, and health service utilisation (see Appendix A).

Substance use. The Timeline Follow Back (TLFB) technique (Sobell & Sobell, 1992) was used to collect retrospective data on daily substance use. In the current study, this method was used to collect information regarding MA use, cannabis use, and any other substance use (including alcohol, ecstasy, cocaine, and prescribed neuroleptic medications) in the past 30 days. This technique uses a calendar recall process to obtain a detailed description of substance use. Guided by the interviewer, the participant marked significant events and other noteworthy occasions such as national holidays, birthdays, and pay days on a blank calendar and these reminder points were then used to prompt recall of substance use (type of substance, route of administration, frequency, and amount used) in the previous 30 days. This method has high test-retest reliability (co-efficients of ≥ .79), and convergent and discriminant validity with other measures of
drug use (Fals Stewart, O'Farrell, Freitas, McFarlin, & Rutigliano, 2000). A copy of the TLFB technique calendar is presented in Appendix B. The age of first regular use of each drug (defined as ≥ once weekly for a month) was also collected.

Severity of MA dependence in the last 12 months was assessed on the Severity of Dependence Scale (SDS), a 5-item scale designed to measure the degree of dependence on a range of drugs (Gossop et al., 1995). Items are explicitly concerned with psychological components of dependence, including impaired control over drug taking and preoccupation with, and anxieties about, drug use. Total scores range between 0 and 15, with a score of 4 or greater indicating MA dependence (Topp & Mattick, 1997). Higher scores reflect a greater degree of dependence on the drug being examined. The SDS has high internal consistency and demonstrated criterion validity (Gossop et al., 1995). A copy of the SDS is presented in Appendix C.

The advantage of using the SDS is that it provides a measure of dependence and chronicity of MA use over the past 12 month period. The rationale for using this measure in analyses was based on the substantive research evidence based on both animal and human laboratory studies reviewed earlier that it is chronic not acute exposure that is associated with hostility.

Positive symptoms. Psychiatric symptoms were assessed using the BPRS – Expanded Version (4.0) for the previous month (Ventura, Lukoff, et al., 1993). The BPRS has been widely used and evaluated over the past four decades, achieving excellence as a measure of psychiatric symptomatology (Panos, 2004; Velligan et al., 2005). The BPRS has strong validity as a measure of symptomatic change across time (Burlingame et al., 2006; Velligan et al., 2005). It also shows discriminant (Rhoades & Overall, 1988), construct (Ventura, Nuechterlein, Subotnik, Gutkind, & Gilbert, 2000), and concurrent validity (Gur, Mozley, Resnick, & Levick, 1991; Ventura et al., 2000),
in addition to internal consistency (Dingemans, Linszen, Lenior, & Smeets, 1995). The BPRS demonstrates good intra-rater and inter-rater reliability of 0.8 or greater (Burlingame et al., 2006; Earnshaw, Rees, Dunn, Burlingame, & Chen, 2005).

The expanded version of the BPRS used in this study consisted of a semi-structured interview of 24 items each scored on a 7-point continuum (1 = not present to 7 = extremely severe). Fourteen items (e.g., anxiety, suicidality, hostility, hallucinations, bizarre behaviour) were rated using the probe questions provided with the measure. The remaining ten items (e.g., blunted affect, tension, distractibility, motor hyperactivity) were rated according to rater observations during the interview.

A recent factor analysis of the 24-item BPRS by Ventura et al. (2000) identified a four-factor solution in a sample consisting mainly of first episode psychotic patients. The factors consisted of positive symptoms (bizarre behaviour, unusual thought content, disorientation, hallucinations, and suspiciousness), negative symptoms (blunted affect, motor retardation, emotional withdrawal, and self-neglect), mania symptoms (motor hyperactivity, elevated mood, excitement, distractibility, hostility, and grandiosity), and depression-anxiety symptoms (depression, anxiety, suicidality, and guilt). The scoring range for each subscale is positive symptoms: 5 to 35, negative symptoms: 4 to 28, mania: 6 to 42, and depression-anxiety: 4 to 28). A score of $\geq 4$ on any item represents a symptom considered to be of pathological intensity (Lukoff, Nuechterlein, & Ventura, 1986). Although the full version of the BPRS was administered to participants, the positive symptom scale was of central importance.

Research assistants currently undertaking postgraduate training in clinical psychology were trained to rate the BPRS using taped BPRS interviews and conducted several real life BPRS interviews while under supervision. Inter-rater reliability for the BPRS was obtained on 44 (18.6%) of the interviews. The intraclass correlation
coefficient for items comprising the positive symptom scale was 0.95. A copy of the BPRS is presented in Appendix D.

**Hostility/aggression.** The BPRS was also used to assess hostility in the previous month. The standard BPRS Hostility question explores a range of aggressive behaviours within a semi-structured interview format. The interviewer asks (i) How have you been getting along with people such as family, co-workers, etc.? (ii) Have you been irritable or grumpy lately? (iii) How do you show it? (iv) Do you keep it to yourself? (v) Were you ever so irritable that you would shout at people or start fights or arguments? (vi) Have you found yourself yelling at people you don’t know? (vii) Have you hit anyone recently? Answers to these questions are rated by the interviewer on the standard 7-point scale. Inter-rater reliability for the BPRS hostility item was obtained on 45 (19.0%) of interviews in the current study. The intraclass correlation coefficient was 0.94. See Appendix D (p. 197).

**Impulsivity.** Impulsivity was measured using the Impulsive Non Conformity Scale from the Oxford-Liverpool Inventory of Feelings and Experiences (O-LIFE; Mason & Claridge, 2006; Mason, Claridge, & Jackson, 1995). The O-LIFE is a four-scale questionnaire used for measuring psychotic characteristics in healthy individuals, measuring trait rather than symptom features. The Impulsive Non-Conformity subscale describes behaviours also measured on other scales of impulsivity, antisocial behaviour and sensation seeking (Pickering, 2004) and consists of 23 items describing impulsive, anti-social, and eccentric forms of behaviour, typically suggesting a lack of self-control. Overall, the O-LIFE has high internal consistency (Mason et al., 1995; Rawlings & Freeman, 1997), test-retest reliability (Burch, Steel, & Hemsely, 1998) and construct validity (Rawlings & Goldberg, 2001; Sellen, Oaksford, & Gray, 2005). Given this study’s focus on the Impulsive Non-Conformity Scale, it was assessed for reliability
using Cronbach’s Alpha prior to conducting analyses. The scale showed good internal consistency ($\alpha = 0.70$). A copy of the Impulsive Non Conformity scale from the O-LIFE is displayed in Appendix E.

**Procedure**

Recruitment took place over an eight month period. Notices were displayed in prominent places in an inner-city Needle Syringe Exchange service inviting participation in a study of MA use. On approaching a counter to obtain injecting equipment, clients were asked to point to the drug group that they were requesting injecting equipment for, from a list displayed on the counter top (benzodiazepines; speed/ice or base; heroin; morphine; methadone). Those who indicated ‘speed’ as the drug they intended to use were asked if they wished to participate in the current study by either needle exchange workers or the research team. Following an explanation of the study and completion of the informed consent form (see Appendix F), a structured face-to-face interview was conducted by a trained, clinical researcher. The interview took approximately 40 minutes to complete.

**Data Analysis**

Data were analysed using the Statistical Package for the Social Sciences (SPSS) program version 14.1 (SPSS Inc, 2005). A series of correlations were initially run to determine whether there were significant relationships between severity of MA dependence, positive symptoms, impulsivity and hostility. A hierarchical, linear regression was used to simultaneously test for mediation and moderation. This method of analysis allowed one variable to be entered into the model at a time in order to examine how much each variable contributed to explaining the variance. It further allowed for the testing of main effects and interactions within and between levels. Step 1 of the regression allows testing of Baron and Kenny’s (1986) criteria for mediation, as
well as testing main effects for tests of moderation. Steps 2 and 3 of the regression tested for interaction effects.

Tests for mediation were based on the definitions and procedures outlined by Baron and Kenny (1986). In order to demonstrate mediation, four criteria need to be fulfilled. First, there should be a significant relationship between the independent variable (IV; MA dependence) and the dependent variable (DV, hostility). Second, there needs to be significant associations between the IV and the proposed mediating variables (MV, i.e., positive symptoms and impulsivity). Third, there needs to be a significant relationship between the MV and the DV when controlling for the association with the IV. Finally, mediation is demonstrated when the relationship between the IV and the DV becomes non-significant when the MV is entered in the equation. Zero-order correlations and regression analyses are used to test these conditions.

To investigate the predicted interaction between positive symptoms and impulsivity, tests of moderation were used following the procedures outlined by Cohen et al. (2003). In order to test moderation, hierarchical regression analyses are used. Predictor variables (i.e., impulsivity, positive symptoms and MA dependence) are entered in the first step of the equation. Two-way interactions are entered in the second step followed by the three-way interaction in step 3. To prevent problems with multicollinearity between the main effects and the cross-products, interaction predictors were calculated using the product of the mean-centred predictor variables (by computing the mean of each independent variable and then replacing each variable with the difference between it and the mean). Significant change in $F$ at the second or third step indicates significant interaction effects. The significance level was set at .05. In order to examine the nature of the significant interactions, simple slopes analyses using
high and low values of each variable were performed, in accordance with the guidelines proposed by Cohen et al. (2003).

**Results**

**Descriptive Information**

Of the 237 participants, over two-thirds (n = 169, 71.3%) were male. The mean age of the sample was 27.7 years (SD = 6.7). The majority of the sample was unemployed (145, 61.2%), with 71 (30.0%) participants engaged in study, 17 (7.2%) were working permanently or part-time, and 4 (1.7%) were engaged in home duties. Seventy-eight (32.9%) had received $\leq$ 10 years of education; 61 (25.7%) had received 12 years of education, 39 (16.4%) had a specific trade or technical college qualification and 36 (15.2%) had a tertiary qualification.

Using a cut-off score of $\geq$ 4 on the SDS, 116 (48.9%) of the sample were dependent on MA. Mean age of first use of MA was 18.5 years (SD = 5.2), with participants using an average of 10.5 days (SD = 8.9) in the past 30 days. Thirty one participants (13.1%) were daily or almost daily users (> 25 days in last 30 days) of MA. Similar descriptive information was collected for cannabis, alcohol and heroin with age of first regular use being 14.9 years (SD = 3.6), 15 years (SD = 3.2) and 19.5 years (SD = 4.9), respectively. Daily or almost daily use of cannabis was reported by 24% (n = 57) of the sample and mean number of days use in the last 30 days was 10.8 (SD = 11.86). There was relatively less alcohol use, with only 4.2% (n = 10) reporting daily drinking and mean number of days use per month being 3 (SD = 6.8). Heroin use was even less common: 3.8% (n = 9) reported daily use and the mean number of days use per month was 2.2 (6.4). There was almost no cocaine use, with only 1 person reporting daily use and 2 people reporting 11 and 12 days of use in the past month. A further 6 people reported that they had used cocaine for three days or less in the past 30 days.
The mean positive symptom score from the BPRS was 6.8 ($SD = 2.8$; range 5 to 19), with 37 participants (15.6%) having a rating of pathological intensity (> 4) on at least one or more symptoms (Lukoff et al., 1986). Table 2 provides a breakdown of scores/ratings for each of the items on the positive symptom scale. Pearson product moment correlations were conducted on key variables including recent use of cannabis, heroin and alcohol on positive symptoms, impulsivity and hostility. All were non-significant. Significant correlations were found between MA dependence and hostility, impulsivity and positive symptoms (see Table 3).
Table 2.

*BPRS Scores on Positive Psychotic Symptom Items and Hostility (N = 237)*

<table>
<thead>
<tr>
<th>Variable</th>
<th>M (SD)</th>
<th>Absent – Mild (1 - 3)</th>
<th>Moderate – Extremely Severe (4 - 7)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unusual thought content</td>
<td>1.38 (0.97)</td>
<td>224 (94.5%)</td>
<td>13 (5.5%)</td>
</tr>
<tr>
<td>Hallucinations</td>
<td>1.52 (1.08)</td>
<td>221 (93.3%)</td>
<td>16 (6.7%)</td>
</tr>
<tr>
<td>Conceptual disorganisation</td>
<td>1.35 (0.74)</td>
<td>230 (97.0%)</td>
<td>7 (3.0%)</td>
</tr>
<tr>
<td>Suspiciousness</td>
<td>1.60 (1.04)</td>
<td>211 (89.1%)</td>
<td>26 (10.9%)</td>
</tr>
<tr>
<td>Bizarre Behaviour</td>
<td>1.27 (0.74)</td>
<td>230 (97.1%)</td>
<td>7 (2.9%)</td>
</tr>
<tr>
<td>Hostility</td>
<td>2.13 (1.67)</td>
<td>188 (79.3%)</td>
<td>49 (20.7%)</td>
</tr>
</tbody>
</table>

Table 3.

*Intercorrelations Between Severity of Methamphetamine Dependence, Impulsivity, Positive Symptoms and Hostility (N = 237)*

<table>
<thead>
<tr>
<th>Variables</th>
<th>BPRS hostility</th>
<th>Impulsive non-conformity</th>
<th>Severity of MA dependence</th>
<th>Mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>BPRS hostility</td>
<td></td>
<td></td>
<td></td>
<td>2.13 (1.67)</td>
</tr>
<tr>
<td>Impulsive non-conformity</td>
<td>.41**</td>
<td></td>
<td></td>
<td>10.98 (4.24)</td>
</tr>
<tr>
<td>Severity of MA dependence</td>
<td>.20**</td>
<td>.28**</td>
<td></td>
<td>4.54 (3.94)</td>
</tr>
<tr>
<td>BPRS – positive symptoms</td>
<td>.40**</td>
<td>.34**</td>
<td>.31**</td>
<td>6.82 (2.79)</td>
</tr>
</tbody>
</table>

*Note.* ** p < .01
Prediction of Hostility: Tests of Mediation and Moderation

(Baron & Kenny, 1986) define mediation as the extent to which a given variable accounts for the causal relationship between two variables, the predictor/independent variable and the criterion/dependent variable. The general test for mediation is to examine the relationship between the independent and the dependent variables, the relationship between the independent and the mediator variables, and the relationship between the mediator and dependent variables. All of these correlations should be significant. The relationship between the independent and dependent variables should be reduced (to zero in the case of total mediation) after controlling for the relationship between the mediator and dependent variables. Moderation, on the other hand, refers to the extent to which a given variable (either qualitative or quantitative) “affects the direction and/or strength of the relation between an independent or predictor variable and a dependent or criterion variable. Specifically within a correlational analysis framework, a moderator is a third variable that affects the zero-order correlation between two other variables. In the more familiar analysis of variance (ANOVA) terms, a basic moderator effect can be represented as an interaction between a focal independent variable and a factor that specifies the appropriate conditions for its operation” (Baron and Kenny, 1986, p. 1174). Thus, a moderator variable is one that influences the strength of a relationship between two other variables, whereas a mediator variable is one that explains the relationship between the two other variables.

As can be seen in Table 3, there was a significant relationship between MA dependence and hostility thereby meeting the first criterion for testing mediation. As shown in Table 4, when all variables were entered simultaneously, total variance accounted for was 25%, with impulsivity accounting for 8% unique variance and
AGGRESSION IN METHAMPHETAMINE USERS

positive symptoms accounting for an additional 7% unique variance. MA dependence contributed little unique variance.

**Mediation.** As shown in Table 3, the first criterion of a significant association between MA dependence and hostility was met ($r = .20, p < .01$). The second criterion (i.e., significant associations between the IV and putative mediators) was also met ($r_{MA \text{ dependence impulsivity}} = .28; r_{MA \text{ dependence positive sym}} = .31$). As shown at step 1 in Table 4, the third criterion (that the putative MVs are significantly associated with hostility after controlling for MA dependence) was also met ($β_{\text{impulsivity}} = .31; β_{\text{positive sym}} = .23$). Finally, the fourth criterion that the association between MA dependence and hostility dropped to non-significance after controlling for the MVs was met, ($β = .05$). Thus, impulsivity and positive symptoms mediated the relationship between MA dependence and hostility.

**Moderation.** Table 4 shows at the second step, a significant change in $R^2$ after the addition of two-way interactions. Specifically, there was a significant interaction between impulsivity and positive symptoms that accounted for 4% unique variance. There was no significant change in $R^2$ at the third step, indicating a nonsignificant 3-way interaction.
Table 4.

*Hierarchical Regression Predicting Hostility (N = 237)*

<table>
<thead>
<tr>
<th>Variables</th>
<th>R²</th>
<th>Δ R²</th>
<th>Δ F</th>
<th>Final Equation after Step 3</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Step 1: Main effects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Impulsivity</td>
<td>.25</td>
<td>.25</td>
<td>25.82***</td>
<td>.120</td>
</tr>
<tr>
<td>Positive Symptoms</td>
<td>.29</td>
<td>.04</td>
<td>3.81*</td>
<td>.140</td>
</tr>
<tr>
<td>MA dependence</td>
<td>.29</td>
<td>.00</td>
<td>.15</td>
<td>.020</td>
</tr>
<tr>
<td><strong>Step 2: 2-way interaction effects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MA x Impulsivity</td>
<td></td>
<td></td>
<td></td>
<td>-.007</td>
</tr>
<tr>
<td>MA x Pos Symptoms</td>
<td></td>
<td></td>
<td></td>
<td>-.002</td>
</tr>
<tr>
<td>Impulsivity x Pos symptoms</td>
<td></td>
<td></td>
<td></td>
<td>.033</td>
</tr>
<tr>
<td><strong>Step 3: 3-way interaction effects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Symptoms x Impulsivity x MA</td>
<td></td>
<td></td>
<td></td>
<td>-.001</td>
</tr>
<tr>
<td>Constant</td>
<td></td>
<td></td>
<td></td>
<td>-.329</td>
</tr>
</tbody>
</table>

Note. *p < 0.05; **p < 0.01; ***p < .001.
In order to examine the nature of the significant interaction between impulsivity and positive symptoms, a simple slopes analysis for hostility and positive symptoms was performed for high and low impulsive groups (based on median split), respectively. As shown in Figure 2, there was a relationship between positive symptoms and hostility in highly impulsive individuals. Those with high positive symptoms and high impulsivity scores had the highest ratings on hostility, $t(230) = 4.93, p < 0.001$. Those with low impulsivity ratings did not differ on hostility, regardless of their level of positive psychotic symptoms, $t(230) = 0.22, ns$.

**Examination of gender.** It is worth noting that the analyses were also performed across gender. While the results for men replicated the effects obtained for the entire sample, the results obtained for women were slightly different. While there was still a significant main effect of impulsivity and positive symptoms in Step 1 for women, this interaction in Step 2 was not significant. The most obvious and plausible explanation for this discrepant finding was the small sample size ($n = 68$) and relative lack of power associated with this. For this reason, only the total group results have been reported.
Figure 2. Simple slopes analysis: Influence of levels of impulsivity and positive symptoms on hostility in methamphetamine users (N = 237).
AGGRESSION IN METHAMPHETAMINE USERS

Discussion

Study 1 was designed to investigate the relationship between dependence on MA and levels of hostility and aggression in a large sample of injecting MA users. The study firstly examined whether a positive relationship existed between MA dependence and increased aggression. The study then explored whether two factors known to be associated with both MA use and aggression – impulsivity and psychotic symptoms – influenced the relationship between MA and aggression.

An Integration of Current Findings

Several theoretical models have been presented and reviewed in an attempt to better understand the factors underlying the relationship between MA use and aggression. First, there is a growing literature base which demonstrates a direct pharmacological effect of MA on aggressive behaviour (Sekine et al., 2006; Sokolov & Cadet, 2006; Sokolov et al., 2004). Second, there is a body of evidence which shows that high levels of impulsivity are commonly observed in individuals who engage in a range of antisocial behaviours, including acts of hostility and aggression, and substance misuse (Carver & White, 1994; Chen et al., 2003; Dawe & Loxton, 2004; Moeller et al., 1997; Todd et al., 2004). Third, compelling evidence has not only demonstrated a strong link between MA and psychotic symptoms (Curran et al., 2004; Leamon et al., 2010; Mahoney et al., 2008; McKetin, McLaren, Lubman, et al., 2006; McKetin, McLaren, Riddell, et al., 2006; Srisurapanont et al., 2003; Zweben et al., 2004), but also a link between psychosis and aggression (Foley et al., 2007; Swanson et al., 2006) that is potentially intensified when it is accompanied by substance misuse (Malla & Payne, 2005; Miles et al., 2003; Ries et al., 2000; Soyka, 2000).

Taken together, the above theories and evidence lead to the suggestion that aggressive individuals possibly already possess high levels of impulsivity – a precipitant
of substance use – and so not only have a greater propensity to experience reduced behavioural control, but are also more likely to abuse substances, such as MA. Once MA is added into the mix, these individuals are also more likely to experience positive psychotic symptoms, thus becoming increasingly paranoid, which subsequently give rise to even more aggressive behaviour.

Consistent with the theoretical underpinnings above, results of the current study revealed that, as predicted, higher levels of MA dependence were associated with increased hostility among users. The study then went on to examine how impulsivity and positive psychotic symptoms might influence this relationship. As expected, the relationship between MA use and aggression/hostility was mediated by both impulsivity and positive symptoms of psychosis. More specifically, increased dependence on MA was related to higher levels of impulsivity and the presence of more positive symptoms of psychosis. Both of these, in turn, were associated with increased hostility. Reviewed research has demonstrated that a prominent psychotic symptom in many MA users is severe paranoia, which has been identified as one of the key elements of mental state that are associated with violence (Junginger, 1996; Nestor et al., 1995). The finding that positive symptoms mediated the relationship between MA and aggression fits well with the existing body of literature which has shown increased levels of aggression in psychotic individuals (Foley et al., 2007; Swanson et al., 2006). Of particular interest, synergistic effects of impulsivity and positive psychotic symptoms on hostility were also apparent in this study, with substantially higher levels of hostility being associated with the presence of positive symptoms in conjunction with heightened impulsivity. Each of these findings will be discussed in turn.

Previous research involving both animals and humans has clearly demonstrated a relationship between chronic MA use and increased aggressive behaviour (Crowley,
1972; Miczek & O’Donnell, 1978; Sekine et al., 2006; Sokolov et al., 2004). In light of this, the current study used a measure of chronic use (i.e., dependence in the last 12 months rather than relying only on days of use in the last 30 days). The present finding that individuals with a greater dependence on MA also reported greater levels of hostility is consistent with existing literature. However, this research extends the findings of previous research by demonstrating that the effects of MA dependence on hostile behaviour are mediated by an increase in impulsivity and an increase in positive symptoms of psychosis. Importantly, when the effects of these two variables are statistically taken into account, the relationship between dependence and hostility/aggression disappears. As previously mentioned, the increased hostility associated with impulsivity presumably reflects increased disinhibition and a reduced ability to regulate negative, hostile feelings and behaviours. In contrast, the association between positive symptoms and heightened levels of hostility presumably reflects an increased propensity to perceive situations as threatening and hence respond to them in a hostile and aggressive manner.

Findings from the current study demonstrated that both impulsivity and positive psychotic symptoms not only contribute independently to the hostile and aggressive behaviour observed in MA users, but their effects are amplified when they occur conjointly. Specifically, it was found that the highest levels of hostility are reported by MA users with greater scores on both positive symptoms and impulsivity. Thus, while the present research showed that MA users report high levels of hostility and that at the univariate level, MA dependence and hostility are significantly correlated, it also demonstrated the mediating and moderating roles of impulsivity and positive symptoms of psychosis on this relationship.
What remains to be systematically proven is whether people who inject MA are highly impulsive before they commence using the drug, and if so, whether MA further increases this impulsivity. Alternatively, MA use may itself result in certain individuals becoming highly impulsive or more so. Both of these hypotheses are likely to be true, with impulsive behaviour being associated with the initiation of drug use (Dawe & Loxton, 2004) and chronic MA use resulting in greater impulsivity and aggression (Homer et al., 2008; Jentsch & Taylor, 1999). The need for caution in determining causality is always necessary in such research designs.

Nevertheless, the General Aggression Model (Anderson & Bushman, 2002) can be utilised to help make sense of the above findings. In accordance with the model, an individual’s likelihood of behaving aggressively is directly related to his or her ability to successfully engage two important faculties – cognitive processes which allow one to appraise and evaluate both the situation at hand, and one’s own internal state, in order to make rational decisions. Research has shown that MA use has a detrimental effect on these appraisal and decision making cognitive processes, including deficits in response inhibition, difficulties with facial affect regulation and in identifying feelings (Henry et al., 2009; Y. T. Kim et al., 2011; Payer et al., 2011; Payer et al., 2008). These difficulties clearly impact on the individual’s ability to identify internal states, as well as the ability to accurately evaluate the intentions of others and make sense of their social environment.

This can be understood in light of the small number of very recent and intriguing studies which have shown that MA results in neurological damage to the prefrontal cortex, which in turn, leads to impaired social-cognitive functioning (Henry et al., 2009; Y. T. Kim et al., 2011; Payer et al., 2011; Payer et al., 2008). The finding in the current study that the highest rates of hostility were reported by those MA users who also
reported high levels of both impulsivity (typically expressed as reduced behavioural inhibition and poor decision making) and positive psychotic symptoms – which are both also associated with disruption in the prefrontal cortex – suggests a mechanism by which some MA users react impulsively with aggression and hostility. Thus, this finding compliments emerging research that the behavioural disturbances, including aggression, which are observed among MA users, may be attributable to neurobiological factors that are associated with impairments in social cognition.

Social Context as a Factor in Methamphetamine Related Aggression

Despite the widely held perception that there is a clear link between MA use and aggression, both earlier studies and the present data indicate that not all MA users engage in aggressive behaviour. The current research clearly highlights two factors (psychotic symptoms and impulsivity) that influence the MA-aggression relationship. Another factor that may be relevant is the social context of the MA user (Asnis & Smith, 1978). Goldstein’s (1985, 1989) three-factor model of the relation between substance use and violence draws attention to the critical role social context may play in increasing the expression of violence among substance abusers in general. The model considers: (i) the psychoactive impact of the substance, (ii) the involvement in crime in order to fund ongoing substance use, and (iii) the type of lifestyle associated with dealing and trafficking drugs, as crucial factors underpinning the relationship between substance use and violence.

With regard to MA, the psychoactive impact of use refers to the ‘toxic’ blend of common MA-induced symptoms such as reduced inhibition, heightened activity levels and energy, a sense of increased self-importance and paranoid thoughts, leading to a higher risk of violence during intoxication and withdrawal (Wright & Klee, 2001). Intoxicated users and those experiencing withdrawal are typically more suspicious and
hypersensitive to external threats, increasing their potential to react aggressively (Sommers & Baskin, 2006). While information about involvement in crime was not collected from MA users in the current study, it seems reasonable to assume that the high rates of unemployment noted, coupled with high levels and frequent use of MA would result in the need for users to find an alternative means to fund and maintain their ongoing drug use. Thus, MA users might use threatening, aggressive or violent behaviour to obtain the funds necessary to sustain their drug use. This model is supported by research which has shown that involvement in the illicit drug market as a result of using MA, coupled with situational factors that accompany a drug dependent lifestyle, leads to a universal and increased risk of both being a victim and perpetrator of violence (Torok et al., 2008). Thus, it is possible that hostility, aggression and violence are prevalent in substance abusing populations.

Other Factors that May Play a Role in Methamphetamine Related Aggression

Everyday aspects of physiological functioning, such as sleep deprivation and reduced food intake (leading to a reduction in insulin), have an adverse impact on the ability to inhibit aggressive responses and to behave in a pro-social manner. Sleep deprivation in particular may play a significant role in the expression of increased behavioural impulsivity, irritability and aggressive behaviour (Anderson & Platten, 2011; McCann, Wilson, Sgambati, & Ricaurte, 2009; Pilcher & Huffcutt, 1996). Research has shown that sleep loss has a significant impact on the prefrontal cortex, which results in a range of deficits in human behaviour including reduced behavioural inhibition (Anderson & Platten, 2011; Drummond, Paulus, & Tapert, 2009), and slower and less accurate processing of emotional facial expressions (Pallesen et al., 2004; van der Helm, Gujar, & Walker, 2010).
The findings from these sleep deprivation studies share remarkable similarity with the studies mentioned previously that have focused on facial affect regulation and identification of feelings (e.g., Henry et al., 2009; Y. T. Kim et al., 2011; Payer et al., 2011; Payer et al., 2008), and reported similar behavioural and cognitive disturbances among MA users. This information is useful in interpreting the finding in the current study that the highest levels of hostility were reported by MA users with the highest levels of impulsivity and positive psychotic symptoms. More specifically, sleep deprivation is a known consequence of acute MA intoxication, with longer binge periods resulting in extreme sleep deprivation (Semple, Patterson, & Grant, 2004).

While the neuropsychological mechanisms underpinning sleep deprivation in MA use remains to be clearly elucidated, preliminary evidence suggests that not only is the prefrontal cortex particularly vulnerable to sleep loss (Chee & Choo, 2004; Drummond et al., 2000), but it also appears to be equally vulnerable to the effects of MA use (Chee & Choo, 2004; Drummond et al., 2000). Evidence shows that reduced metabolic activity and dysfunction in the prefrontal regions of the brains of MA users plays an important role in the expression of personality, affect, and inhibitory behaviour and warrants further investigation (Kahn-Greene, Lipizzi, Conrad, Kamimori, & Killgore, 2006).

**Contributions and Limitations of the Current Study and Future Directions**

In contrast to many other studies investigating the association between MA use and aggression, this study has attempted to examine two of the key mechanisms through which this association operates. This has allowed a deeper understanding of how these factors interact to influence the relationship between MA and aggression. Further, the current study has sampled a large number of injecting MA users and has utilised reliable and valid standardised measures. In addition, the sample of participants recruited for
this study identified MA as being the primary drug they used, with few reporting other illicit drug or alcohol use. This is clearly an important factor when trying to examine the specific effects of MA on behaviour, since other substances, particularly alcohol, have been associated with increased aggression (Giancola et al., 2009).

As with all research, this study also has several limitations that require consideration. First, Study 1 was both cross-sectional in nature and based on self-report data. While it was considered beyond the scope of the current study, it is acknowledged that a prospective study investigating the course of the relationship between MA use and aggression would contribute to a level of understanding beyond that which a cross-sectional study is able to achieve. Further, while one of the key advantages of using self-report measures is that a large sample is able to be recruited and tested in a relatively brief period of time, the use of this method in isolation presents some concerns when considering the constructs of impulsivity, aggression and hostility. More specifically, while self-report questionnaires are the most widely used form of assessment of personality constructs, with over 95% of personality research reports using this method (Kagan, 2007; Vazire, 2006), there are limitations associated with self-report measures including subjective and inaccurate reporting, response biases, and the influence of demand characteristics (Fiske & Taylor, 2008; Leite & Cooper, 2010; Paulhus, 1991). Although self-report has been identified as a highly valid measure of substance use (McPhillips et al., 1997; Selten et al., 2002; Weiss et al., 1998; Wolford et al., 1999), the use of urine drug screening in a more systematic manner would have enhanced the methodological rigour of Study 1. Further, recent reviews of measurement issues in the aggression literature also conclude that non-laboratory based assessment measures alone do not assess aggression with appropriate conceptual clarity (Eckhardt, Norlander, & Deffenbacher, 2004; Parrott & Giancola, 2007; Suris et al.,
Thus, future research should ideally include the use of both self-report measures and experimental tasks in examining the relationship between MA use and aggression (Tyner & Fremouw, 2008).

Second, while the BPRS (Ventura, Lukoff, et al., 1993) has been shown to be a valid and reliable measure of psychological functioning, the sole reliance on the hostility item from the questionnaire poses some concern. It is acknowledged that this study would have benefited from the inclusion of an additional standardised measure of aggression. While the BPRS is not a comprehensive measure of aggression, it was chosen based on its use and convenience for the assessment of psychotic symptoms and hostility in MA users. Given the time involved in sampling such a large number of participants, this measure seemed to offer the benefit of assessing a range of relevant behaviours in a time efficient manner. Furthermore, assessment using the BPRS did incorporate responses from the participants themselves and interviewer observations.

Third, the current study was not able to systematically test for gender differences. This is an important aspect of any study on aggression as there is evidence of gender differences in both the expression of aggression, hostility (e.g., Bettencourt & Miller, 1996) and in impulsiveness (Cross et al., 2011; Mason & Claridge, 2006). As mentioned previously, although the regression model was run separately for men and women, there were problems with power that may have rendered the findings inconclusive. While the results remained the same for men, and there was still a significant main effect of impulsivity and positive symptoms in Step 1 for women, this interaction in Step 2 was not significant, which most likely reflects small sample size and reduced power. Clearly, future research with a larger sample of women is needed to further explore possible gender differences.
Finally, route of drug administration may also play a key role in the development of hostile behaviour. All participants reported that they injected MA. Past research reveals that this route, compared for example with smoking, increases the risk of psychotic and other psychopathological symptoms, since injecting may be associated with higher brain concentrations (Domier, Simon, Rawson, Huber, & Ling, 2000; Matsumoto et al., 2002; Sekine et al., 2006). Longitudinal research that begins before the onset of MA use is required to answer questions regarding the role played by route of MA administration.

In conclusion, this study found evidence that higher levels of MA dependence are associated with increased hostility among users and clearly demonstrated that this relationship is mediated by both impulsivity and positive symptoms of psychosis. More specifically, increased dependence on MA was associated with higher levels of impulsivity and the presence of more positive symptoms of psychosis. Both of these factors, in turn, were associated with increased hostility. Of particular interest, synergistic effects of impulsivity and positive psychotic symptoms on hostility were also apparent, with substantially higher levels of hostility being associated with the presence of positive symptoms in conjunction with heightened impulsivity. Based on the literature available, the current study appears to be the first to examine and report such findings. The findings of this study, coupled with an extensive literature base supporting a link between MA dependence and aggression, highlights the need for additional research which is rigorous in design, and which employs both self-report and behavioural measures of hostility, aggression and impulsivity.
CHAPTER 5

Study 2: A Behavioural Investigation of Aggression and Impulsivity in MA Users

Study 1 yielded several findings of note – positive relationships between severity of MA use and impulsivity, psychotic symptoms, and aggression; synergistic or interactive effects of impulsivity and psychotic symptoms on levels of aggression; and evidence that impulsiveness and symptoms of psychosis mediate the MA-aggression relationship. These findings both replicate and extend previous research on the effects of MA use. They are, however, based on self-report measures and, as such, may be influenced by response biases. Confirmation of at least some of these findings using behavioural measures would be of value.

More specifically, Study 1 was both cross sectional in nature and based on self-report data. Although the use of self-report measures allowed for the testing of a large number of participants in the first study, as previously discussed, there are potential limitations associated with self-report measures and concerns about the overreliance on such measures (Fiske & Taylor, 2008; Leite & Cooper, 2010; Paulhus, 1991). Issues have been raised regarding the impact of social desirability on reporting and the extent to which individuals are able to accurately report on their own behaviour (Fiske & Taylor, 2008; Leite & Cooper, 2010; Nisbett & Wilson, 1977; Paulhus, 1991). Similarly, the exclusive use of behavioural tasks is also considered insufficient in completely capturing the construct of aggression (Eckhardt et al., 2004; Parrott & Giancola, 2007; Suris et al., 2004), despite Furr and Funder’s (2007, p. 273) suggestion that, “The most seemingly obvious and necessary way to gauge an individual’s personality is to see how they act”. Instead, emerging opinions strongly support the inclusion of a range of these data collection methods, particularly in examining the MA-aggression link (Tyner & Fremouw, 2008).
Given that MA is an illicit drug, and chronic MA use has been shown to be associated with increased aggression, it would clearly be unethical to administer doses of this drug to human participants in order to directly observe and study the effects of it on their behaviour. It is possible, however, to observe the behavioural responses of individuals who use MA themselves by having them complete laboratory tasks and comparing their performance with a non-MA using control group.

A second study was therefore designed to examine the relationship between MA use and impulsivity and aggression using behavioural as well as self-report measures of the latter two constructs. Psychotic symptoms, especially positive symptoms, are difficult to assess behaviourally and hence behavioural measures of psychotic symptoms were not included. Instead, considerable care was taken to select participants without active psychotic symptoms or a prior history of hospitalisation or treatment for psychosis.

Study 2 employed a between-subjects design in which the responses of MA users were compared with those of non-MA users. As previously mentioned (see Chapter 3), other researchers have used behavioural measures to explore the effects of MA on impulsivity and inhibitory control, but these studies have typically compared MA users to poorly matched control participants, paying little heed to the marked differences in education, background, and sociodemographic factors between the two populations. It is thus difficult to know whether the observed group differences in impulsivity and aggression reported in earlier studies are due to MA use or to one or more of the many other factors that differentiate the groups. To overcome this problem, the current study paid great attention to recruiting a control group of non-MA using participants who did not differ on sex, age, intelligence, education or socioeconomic status and were also residing in the same geographic location (i.e., same post code).
At the time Study 2 was designed and conducted, no previous research had examined the effects of MA use on a behavioural measure of aggression. Recently, however, Payer et al. (2011) have reported an extremely interesting and important study on the neurobiological effects of MA using a behavioural task of aggression. Although this research has been outlined in Chapter 2, in light of its relevance to the current study, the key features of the Payer et al. (2011) research will be summarised below.

In their research, Payer et al. (2011) were the first to combine a self-report measure of aggression, a behavioural task measuring aggression and neuroimaging techniques to examine the effects of MA on aggressive behaviour in users. The key focus of the study was an investigation of the neurobiological correlates of affect processing and aggression in 39 MA dependent individuals (16 females, 23 males) who had remained abstinent for 7 to 10 days and 37 non-drug using controls (18 females, 19 males). As mentioned previously, although all participants completed self-report measures of aggression and alexithymia, only 12 participants in the MA group and 15 controls completed the behavioural measure of aggression, and fMRI was only performed on 25 MA dependent individuals and 23 control participants. A revised version of the AQ (Buss & Warren, 2000), containing 34 items, provided the self-report measure of aggression. Participants also completed the Toronto Alexithymia Scale (Bagby, Parker, & Taylor, 1994) and two affect processing tasks (affect matching and affect labelling). The Competitive Reaction Time Task was used to elicit aggression among participants. This task was delivered over 4 blocks, with the first block consisting of a single practice trial, and the remaining 3 blocks consisting of 8 trials each. Level of provocation was gradually increased as the blocks progressed, such that the final block represented peak provocation. Finally, fMRI was used to examine links
between brain function, aggression and affect regulation in MA users, with a particular focus on examining the roles of the amygdala and prefrontal cortex in this process.

Key findings included higher levels of self-reported aggression and increased displays of elevated aggressive responding on the Competitive Reaction Time Task among MA users compared to controls. This effect only occurred during the fourth testing block (i.e., peak provocation), however, suggesting that MA users were especially reactive and aggressive when confronted with high levels of provocation. Performance on the affect matching task revealed that no significant differences between the groups in activation of the amygdala, a region of the brain implicated in emotion regulation. In contrast to controls, MA users demonstrated lower activation in the bilateral ventral inferior frontal gyrus, which is implicated in emotional insight. Further, decreased amygdala activity was associated with elevated displays of aggression on the behaviour task in both MA users and controls, but was only related to increased self-reported aggression in controls. Compared to controls, MA users reported higher levels on one of the three scales of the Toronto Alexithymia Scale (Bagby et al., 1994). Specifically, they reported greater difficulty in identifying feelings.

In sum, although there was no evidence of deficits in emotion regulation among MA users, findings were suggestive of poor emotional insight, as evidenced by distinct patterns of activation in specific regions of the brain (particularly the frontal gyrus) and higher self-reported levels of alexithymia than controls. The authors concluded that despite finding a positive relationship between MA and both self-reported aggression and perpetrated aggression this relationship is clearly mediated by other factors, including personality variables (Payer et al., 2011).
The findings and conclusions revealed by Payer et al. (2011) are both directly relevant and complimentary to the current study. In order to extend the findings from Study 1, the current study investigated the relationship between MA use and aggression using a behavioural measure of aggression, as well as two self-report measures. Research has clearly shown that in order to behave in a controlled and pro-social manner, an individual must have the cognitive resources/abilities to assimilate new information, meaningfully integrate the information with previously known information, and form a plan for behavioural response (Frank, 2005). Importantly, the current study sought to conduct a rigorous investigation using a combination of the aforementioned measures in a group of MA users and a group of non-MA users from the same community. Further, participants in the MA group were current/active users who were not asked to abstain from taking MA for the purpose of conducting this investigation.

**Behavioural Measures of Aggression – Establishing the Context for Study 2**

Although there is a paucity of behavioural studies investigating aggression in MA users, a number of studies have used laboratory tasks to measure the effects of alcohol on aggressive behaviour (for meta-analyses see Bushman & Cooper, 1990; Ito, Miller, & Pollock, 1996). In these studies, alcohol is given within a laboratory setting and aggressive responding is measured using behavioural tasks. The two most commonly used behavioural tasks to measure aggression in alcohol users are the Taylor Aggression Paradigm (S. Taylor, 1976) and the Point Subtraction Aggression Paradigm (Cherek, 1992). The TAP typically involves participants engaging in a competitive reaction time task with a fictitious opponent wherein they both administer and receive mild electric shocks or blasts of white noise. This task is similar to that used by Payer et al. (2011). The Point Subtraction Aggression Paradigm typically involves
participants engaging in a task with a fictitious opponent wherein they are both given the opportunity to earn and subtract points from each other, which are redeemable for money. In the following section, two recent studies which have behaviourally measured the relationship between aggression and alcohol using the TAP specifically will be presented to exemplify recent research in the area.

**Behavioural studies examining the link between alcohol and aggression.** A recent study used a modified version of the TAP to examine the acute effects of alcohol on aggressive behaviour in men and women (Giancola et al., 2009). Participants were led to believe they were competing against an opponent of the same gender on a competitive reaction time task. Results revealed that alcohol significantly increased aggressive responding among both men and women, with a stronger effect for men. Interestingly, this is one of the first studies to demonstrate in a controlled laboratory setting that alcohol increases aggression in women.

Another study used a modified version of the TAP to examine whether irritability mediates the relationship between executive functioning and alcohol related aggression in a group of 313 healthy social drinkers (Godlaski & Giancola, 2009). Past research indicates that two factors which increase the risk of becoming aggressive when intoxicated are lower levels of executive functioning and higher levels of irritability (Giancola, 2002, 2004; Godlaski & Giancola, 2009). Participants consumed either a placebo or alcoholic beverage before engaging in the competitive reaction time task. Aggression was operationalised as the level of electric shock intensities administered to a fictitious opponent under conditions of high and low provocation. The authors found that for intoxicated men, irritability mediated the association between executive functioning and aggression. There were no effects found for sober men or women, or intoxicated women.
In sum, behavioural studies have successfully demonstrated that participants who consume alcohol prior to performing such tasks typically respond more aggressively than participants who receive either a placebo or non-alcoholic beverage (Bushman & Cooper, 1990; Chermack & Giancola, 1997). Interestingly, a dose-response effect is often observed whereby an increase in alcohol leads to an increase in aggressive responding (Giancola, 2002; Hoaken & Pihl, 2000).

**Behavioural studies examining the links between stimulant use, alcohol use and aggression.** A recent study of particular interest, focused on stimulants, alcohol and aggression. More specifically, Giancola and Parrott (2005) investigated the moderating effects of past-year stimulant and sedative use on alcohol related aggression in a group of 330 healthy social drinkers. The authors were particularly interested in determining whether stimulant users possessed higher levels of behavioural disinhibition that in turn heightened alcohol-related aggression in comparison to sedative users. The authors suggested that individuals with high impulsivity and behavioural disinhibition were more likely to have used stimulants rather than sedatives in the past 12 months. Further, they predicted that alcohol would more likely increase aggression in participants with higher levels of past year stimulant use than those with lower levels and this effect would be accounted for by individual differences in the ability to exercise behavioural control. Finally, it was expected that there would be no relationship between past year sedative use and increased aggressive behaviour.

Participants completed a range of self-report questionnaires measuring substance use and behavioural inhibition and also participated in a behavioural task measuring aggression. Following consumption of either an alcoholic beverage or a placebo, participants were tested on a modified version of the TAP which involved them administering and receiving mild electric shocks from a fictitious opponent under
conditions of both low and high provocation (S. Taylor, 1976). It was found that alcohol significantly increased the relationship between past year stimulant use and aggressive responding in men only. Moreover, this effect was particularly pronounced in men with higher levels of past year stimulant use. Contrary to expectations, behavioural disinhibition (i.e., high levels of impulsivity and sensation seeking) did not account for the association between stimulant use and aggression. Finally, consistent with much of the research regarding gender differences in aggression, men were found to respond more aggressively than women on the TAP (Giancola & Parrot, 2005; Verona & Curtin, 2006).

In another study which examined a broad range of substances, aggressive responding was compared between 29 subjects with a history of substance dependence (but no current use) and 24 subjects with no history of drug abuse or dependence (T. J. Allen, Moeller, Rhoades, & Cherek, 1997). Sixty-two percent of the sample reported a history of cocaine dependence and twenty four percent reported a history of amphetamine dependence. The Point Subtraction Aggression Paradigm was used to examine aggressive responding among both groups and involved several 25 minute testing sessions which were conducted over the course of two days. Participants with a substance dependence history were found to emit more aggressive responses per session than controls. Similarly, the substance group also reported more aggression on self-report measures of aggression, including the Buss-Durkee Hostility Inventory, the Overt Aggression Scale and the Brown History of Violence questionnaire. These results were consistent with prior studies which have reported an association between aggression and drug abuse or dependence.
In brief, the behavioural studies above demonstrated that individuals who were either currently using a combination of alcohol and stimulants, or had a history of stimulant use perpetrated greater levels of aggression than controls.

**Summary**

Despite the existing studies which have examined the relationship between MA use and self-reported aggression, the relationship between MA use and self-reported impulsivity, and also the studies which have behaviourally examined the relationship between MA use and disinhibition, only one behavioural study examining aggression in MA users appears to have been conducted to date. Importantly, no single study has employed a combination of both self-report and behavioural measures of aggression and impulsivity to explore important elements of behavioural and social functioning in MA users. Study 2 was designed to address this gap in the literature.

**Aims of Study 2**

Study 2 aimed to use both self-report measures and behavioural measures of aggression and behavioural inhibition in MA using and non-MA using participants recruited from the same geographic area/post code. The study sought to compare the performance of MA users with controls on (i) a laboratory task that measured aggression, and (ii) a laboratory task that measured behavioural disinhibition. Participants also completed the same self-report measures of substance use, aggression/hostility and impulsivity used in Study 1, as well an additional self-report measure of both aggression and impulsivity.

Based on the findings from Study 1 and the extensive literature base confirming the MA-aggression link, it was predicted that MA users would report higher levels of aggression than controls. It was further predicted that MA users would perpetrate higher levels of aggression compared to controls. Similarly, in light of results from
Study 1 and a large body of research demonstrating high levels of impulsivity and disinhibition in MA users, it was hypothesised that MA users would report higher levels of impulsivity than control participants and would perform worse than controls on a behavioural measure of disinhibition, demonstrating a reduced ability to inhibit a pre-potent response. Finally, the potential influence of MA use was investigated by testing whether level of chronic use (measured as number of years of dependent use) was related to either aggression or impulsivity.

Method

Participants

A total of 42 participants were recruited for this study. The MA group consisted of 11 males (55%) and 9 females (45%) who were regular, intravenous users of MA. Participants in the MA group were recruited through notices placed at the Needle Syringe Exchange Service at Logan Alcohol Tobacco and Other Drugs Service (ATODS) in Queensland, Australia. Of 51 participants screened at the Needle Syringe Exchange Service, 21 MA users met eligibility criteria (see next paragraph for criteria). 22 control participants were screened of which 21 met eligibility criteria (see next paragraph). The control group comprised of 10 male (47.6%) and 11 female (52.4%) non-MA using healthy individuals. While control participants were not matched to MA participants, they were deliberately recruited from the same community and geographical post code as the Needle Syringe Exchange Service which is located in a low socioeconomic area characterised by high rates of unemployment and substantial social and economic disadvantage (Australian Bureau of Statistics, 2010).

All individuals who expressed an interest in the research were asked a series of screening questions. These included: (i) How old are you? (ii) Do you use alcohol? If yes, how often do you drink and how many standard drinks would you usually consume
on a single occasion?  (iii) Do you use any illicit or prescription drugs?  If yes, which
ones and how often do you use this/these drugs?  What is your drug of choice, as in
which drug do you like to use most often?  (iv) Do you currently use
methamphetamine?  If no, have you ever used methamphetamine in your life?  (v) Have
you ever acquired brain damage or been classified as having an intellectual impairment?
(vi) Have you ever been diagnosed with a psychotic illness, such as schizophrenia or
drug-induced psychosis?  (vii) Have you ever been prescribed anti-psychotic
medication?  (viii) In the past two weeks have you experienced any of the following
symptoms including – feeling overly suspicious or concerned that someone might be
overly concerned about you or watching you; having hallucinations where you thought
you saw or heard things even though no-one appeared to be around; behaving in a way
that attracted the attention of other people or in a way that other people might have
found odd or unusual; feeling like other people were able to read your mind or someone
or some force was trying to control you?

Eligibility criteria for inclusion in the experimental group of the study were
current use of MA, not currently dependent on any other substances except MA,
cannabis, or nicotine, 18 years or older, not currently psychotic, and the absence of an
acquired brain injury or intellectual impairment.  The decision was made to not include
cannabis dependence in the exclusion criteria on the basis that approximately one
quarter (24%) of MA users in Study 1 also met criteria for cannabis dependence.

Eligibility criteria for inclusion in the control group were no current or lifetime
dependence on any illicit substances, alcohol and prescription drugs, an absence of
lifetime MA use, aged 18 years or older, no current psychotic symptoms, no self-
reported history of treatment or hospitalisation for psychosis, and no prior brain injury
or intellectual impairment.  All participants were offered $20 for their participation.
Informed consent was obtained from each participant and the study was approved by the relevant human research ethics committees of both Griffith University and Metro South Health Service District.

**Materials**

**Demographic information.** A semi-structured interview was used to collect information regarding age, gender, education, employment status, living situation, marital status and ethnicity. The interview schedule also contained questions about the participant’s psychiatric history and use of psychotropic medication (see Appendix G).

**Psychosis.** The Psychosis Screener (Jablensky et al., 2000) was used to screen participants for characteristic psychotic symptoms in the past year. This screening instrument was used in the 1998 Australian National Survey of Mental Health and Well-Being to measure the prevalence of psychosis among the Australian general population (Degenhardt, Hall, Korten, Morgan, & Jablensky, 2005) and has been successfully used in several studies since this time (e.g., McKetin, McLaren, Lubman, et al., 2006). The Psychosis Screener comprises 7 items, 3 of which (items 1a, 2a, 3a) are asked only if the participant endorses a previous question. The first 6 items cover the following features of psychotic disorders: delusions of control, thought interference and passivity (Questions 1 and 1a); delusions of reference or persecution (Questions 2 and 2a), and grandiose delusions (Question 3 and 3a). The final item (Question 4) records whether a participant has ever been diagnosed with a psychotic illness including schizophrenia. The psychosis screener was scored according to the procedure used by Degenhardt et al. (2005) in which a score of 1 was assigned to each endorsed item, except for one item that identified whether grandiose beliefs were shared by a group of people, which was scored as minus 1. Possible scores range from 0 to 6. A cumulative score of 3 or greater was used to identify cases of psychosis, yielding a sensitivity of 82% and a
specificity of 57% against a DSM-III-R or an *International Classification of Diseases* version 10 (ICD-10) diagnosis of schizophrenia. The screener is a valid (Jablensky et al., 2000; McKetin, McLaren, Lubman, et al., 2006) and reliable instrument that demonstrates good internal consistency, with an alpha reliability coefficient (α) of 0.74 (Degenhardt et al., 2005; Jablensky et al., 2000). A copy of the Psychosis Screener is presented in Appendix H.

**Substance use.** Consistent with Study 1, frequency and quantity of MA use in the last 30 days was collected using the TLFB technique (Sobell & Sobell, 1992). This method was also used to collect information regarding any other illicit substance use, alcohol use, and prescription drug use within the same period of time. The age of first regular use of each drug (defined as ≥ once weekly for a month) was also collected. A detailed description of the TLFB technique was provided in Chapter 4 (p. 77). A copy of the TLFB technique calendar is presented in Appendix B.

The Psychiatric Research Interview for Substance and Mental Disorders (PRISM; Hasin, Trautman, & Endicott, 1998) was used to confirm eligibility in the study, to provide a confirmed diagnosis of MA dependence and to collect information regarding number of years of dependence. The PRISM is a semi-structured diagnostic interview designed specifically to assess and diagnose substance abuse and dependence, in accordance with *DSM-IV-TR* (American Psychiatric Association, 2000) criteria. Section 2 of the PRISM was used to screen for substance use in both groups and Section 3 was used in the MA group to assess participants’ current and/or lifetime abuse or dependence on any substances that passed screening. The PRISM interview has demonstrated good psychometric properties in terms of test–retest reliability (Hasin et al., 2006), inter-rater reliability (Morgello et al., 2006) and validity (Torrens, Serrano, Astals, Pérez-Domínguez, & Martín-Santos, 2004) in diagnosing psychiatric disorders.
among substance using populations. The researcher had received formal training in the use of this instrument and had three years prior experience in using it as part of a large, longitudinal study. The information obtained from the PRISM was used to determine which substances would be further assessed for dependence using the SDS (described below) and checks were performed to ensure dependence was confirmed across both measures. A copy of the PRISM can be found at http://www.columbia.edu/~dsh2/prism/.

As in Study 1, the SDS was also used to provide a measure of dependence and chronicity of MA use over the past 12 month period (Gossop et al., 1995). Thus, fitting with research evidence that chronic rather than acute exposure to MA is associated with hostility in both animals and humans. A description of the SDS and its psychometric properties was provided in Chapter 4 (p. 78). Total scores range between 0 and 15, with a score of 4 or greater indicating MA dependence (Topp & Mattick, 1997). Higher scores reflect a greater degree of dependence on the drug being examined. This measure was also used to assess dependence on cannabis. A copy of the SDS is presented in Appendix C.

**Impulsivity.** Two self-report measure of impulsivity were used. Consistent with Study 1, impulsivity was measured using the Impulsive Non Conformity Scale from the Oxford-Liverpool Inventory of Feelings and Experiences (O-LIFE; Mason & Claridge, 2006; Mason et al., 1995). This measure has been described in Chapter 4 (p. 80). A copy of the Impulsive Non-Conformity Scale from the O-LIFE is presented in Appendix E.

The Barratt Impulsiveness Scale - 11 (BIS-11; Patton, Stanford, & Barratt, 1995) was also used to measure impulsiveness. The BIS-11 is a 30-item self-report measure containing 6 first-order factors and 3 second-order factors. First-order factors include:
1) attention – focus on the present task; 2) motor impulsiveness – acting on the spur of the moment; 3) self-control – thinking and planning carefully; 4) cognitive complexity – enjoyment from mentally challenging tasks; 5) perseverance – living a stable/consistent lifestyle; and 6) cognitive instability – racing thoughts and thought insertion. Taken together, scores on the attention and cognitive instability items form the Attentional Impulsiveness factor; the perseverance and motor impulsiveness items form the Motor Impulsiveness factor; and the self-control and cognitive complexity items form the Non-planning Impulsiveness factor. The BIS-11 has been found to have good internal reliability with alpha coefficients ranging from 0.79 to 0.83 (Fossati, Di Ceglie, Acquarini, & Barratt, 2001). Test-retest reliability of 0.89 has been reported after 11 months (Fossati et al., 2001). An alpha coefficient for total BIS-11 score for the current study was 0.82. A copy of the BIS-11 is presented in Appendix I.

**Aggression/hostility.** The AQ is a full revision of the Buss-Durkee Hostility Inventory, a widely-used measure assessing hostility and aggression (Buss & Perry, 1992). The measure contains 29 items, which are scored on the following four subscales: physical aggression, verbal aggression, anger and hostility. A total score is also obtained with possible scores ranging from 29 to 145, with higher scores representing greater aggression. Participants rate their responses using a 5 point Likert scale ranging from 1 – ‘very uncharacteristic of me’ to 5 – ‘very characteristic of me’. Internal consistency for the four subscales and total score range from 0.72 (Verbal Aggression) to 0.89 (Total AQ score). An alpha coefficient of 0.94 (Total AQ score) was obtained in the current study. Retest reliability for the AQ after 9 weeks is good with reported correlations ranging from 0.72 for anger to 0.80 for physical aggression and for the total score (Buss & Perry, 1992). The AQ has good reported construct
validity (M. B. Harris, 1996; M. B. Harris & Knight-Bohnhoff, 1996). A copy of the AQ is presented in Appendix J.

As in Study 1, the hostility items from The BPRS was also used to provide a self-report measure of hostility (Ventura, Green, Shaner, & Liberman, 1993). The item questions explore a range of hostile behaviours (How have you been getting along with people such as family, co-workers, etc.? Have you been irritable or grumpy lately? How do you show it? Do you keep it to yourself? Were you ever so irritable that you would shout at people or start fights or arguments? Have you found yourself yelling at people you don’t know? Have you hit anyone recently?). Answers to these questions were rated by the interviewer on the standard 7-point scale. The psychometric properties of the BPRS have been detailed in Chapter 4 (p. 78). A copy of the BPRS hostility item is presented in Appendix D.

**Intelligence.** The Quick Test (Ammons & Ammons, 1962) was included for two reasons: (i) to exclude any participants who were intellectually impaired, and (ii) to obtain an estimate of participants’ overall intellectual functioning to ensure parity across the MA and control group. The Quick Test is a brief intelligence test consisting of 3 forms containing 50 items each. Each form takes approximately 2 minutes to complete. Although the test is designed to allow the administration of a single form under conditions where time is limited, the authors recommend administering a combination of all 3 forms to increase the accuracy and reliability of the test (Ammons & Ammons, 1962). The Quick Test is a passive response picture-vocabulary test in which the participant is shown an A4 size card containing line drawings/pictures. The participant is asked to point to the picture that best illustrates the meaning of a word read aloud by the researcher. Participants are awarded one point for each correct answer. The range
of possible raw scores on each form is 0 to 50, giving a total possible score ranging from 0 to 150. Raw scores are then converted to an intelligence quotient score.

The Quick Test correlates highly with other cognitive measures including the Wechsler Adult Intelligence Scale (WAIS) Full Scale IQ (Hogan, 1969; Seitz & Braucht, 1971). A raw score of either 46 on a single form of the Quick Test or a combined raw score of 135 across the three forms, translates to a Full Scale IQ score of 110, which is in the high average range of functioning. Normative data are available in the test manual for samples of non-impaired individuals and clinical populations. The test has good concurrent (Ammons & Ammons, 1962) and construct validity (Acker & Davis, 1989), and reported reliability coefficients ranging from 0.89 (Husband & DeCato, 1982) to 0.91 (Levine, 1971). Participants were required to complete all 3 forms of the Quick Test as part of the current study.

**Behavioural tasks.** Two computer-based behavioural tasks were used to directly measure aggression and disinhibition. These laboratory tasks are described below.

**Aggression.** A modified version of the TAP (S. Taylor, 1976) was used to measure aggressive behaviour. The TAP is a commonly used, safe and valid task which has been shown to elicit aggressive behaviour in men and women (Anderson & Bushman, 1997; Giancola & Parrott, 2008; Hoaken & Pihl, 2000; Richardson, Bernstein, & Taylor, 1979; Richardson, Vinsel, & Taylor, 1980). The original version of the TAP places participants in a situation in which electric shocks are received from, and administered to, a fictitious opponent during a supposed competitive reaction-time task (S. Taylor, 1976). In this version of the task, physical aggression is operationalised as the shock intensities selected by the participants. Since its original conception, however, a modified version of the task has been developed which involves
administering uncomfortable white noise for a selected duration of time, instead of
electric shocks. Research has demonstrated that noise delivered out of the participant’s
direct control serves as a highly potent noxious stimulus (Lundberg & Frankenhaeuser, 1978). The paradigm measures two aspects of aggression – noise level is generally
considered an indicator of perpetrated overt aggression, whereas noise duration is
considered an indicator of more covert aggression (since participants receive feedback
after each trial advising them of the noise level, but not the duration, their opponent had
selected). The task captures the true essence of aggressive behaviour, since participants
can intentionally and deliberately choose to subject another person to pain or harm. The
adapted noise version of the TAP has been used successfully in other aggression
research with a range of populations (Böhnke, Bertsch, Kruk, & Naumann, 2010; Bond

The task was run on a laptop computer with sound delivered via headphones. Participants were told that they were competing against another person of the same sex in a reaction time task that required them to click their ‘mouse’ button as quickly as possible when a red square appeared on their computer screen. They were advised that whoever lost the trial (i.e., the slowest competitor) would receive a burst of noise, preset by the winning opponent, and prior to each trial. Various noise durations could be selected by the participant and ranged between 0 seconds (level 0) and 5 seconds (level 10) in 0.5 second increments. Similarly, the participant was able to select a range of noise volumes or intensities between 60 dB (level 1) and 105 dB (level 10) in 5 dB increments. Participants were able to choose to not punish their opponent at all by selecting the level 0, which corresponded to no sound at all.

Unknown to the participants, there was obviously no actual ‘competitor’, but rather the number of winning and losing trials, as well as the opponent noise levels and
durations were preset by the researcher and consisted of varying levels of provocation including low (intensity noise levels 1 to 3 and short durations of 1 to 2 seconds), moderate (intensity noise levels 4 to 6 and intermediate durations of 2 to 3.5 seconds), and high (intensity noise levels 7 to 10 and long durations of 3.5 to 5 seconds). A combination of low, medium and high intensity noises were distributed throughout the 25 trials in accord with the sequence outlined in Table 5. The task took approximately 10 minutes to complete.

The task contained a single block, consisting of 25 trials of which the participants won 12. The same sequence of events applied to each trial and consisted of the following 4 steps. First, participants were required to set the noise level of the burst (if any) their opponent would receive if he or she was the slowest to respond. Second, participants selected the duration of the noise their opponent would receive in the event of losing, by clicking on a specified duration button on the computer screen. Third, participants competed in a single trial of the task and received feedback on their computer screen indicating whether they had won or lost the trial. It is important to note that irrespective of whether the participant won or lost a trial, they were informed of the noise level (but not the duration) their opponent set during each trial via a feedback metre which appeared on their computer screen. Fourth, participants either received a burst of noise in their headphones on trials they lost (i.e., slower reaction time than fictitious opponent), or did not receive any burst of noise, but instead, assumed their opponent was hearing the noise they had previously set. This process was repeated for each trial. Participants received a written series of steps to follow for each trial (see Figure 3).
Participants were led to believe that their opponent was seated in front of a laptop computer in another room in the same building. In order to maximise the credibility of the task, once the participant was ready to commence the task, the
Table 5.

_TAP – Schedule Displaying Levels of Provocation Used_

<table>
<thead>
<tr>
<th>Trial</th>
<th>Intensity (Level 1 to 10)</th>
<th>Duration (1 to 5 seconds)</th>
<th>Level of Provocation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2</td>
<td>2</td>
<td>Low</td>
</tr>
<tr>
<td>2</td>
<td>1</td>
<td>1</td>
<td>Low</td>
</tr>
<tr>
<td>3</td>
<td>4</td>
<td>3</td>
<td>Medium</td>
</tr>
<tr>
<td>4</td>
<td>1</td>
<td>2</td>
<td>Low</td>
</tr>
<tr>
<td>5</td>
<td>2</td>
<td>1</td>
<td>Low</td>
</tr>
<tr>
<td>6</td>
<td>3</td>
<td>3.5</td>
<td>Medium</td>
</tr>
<tr>
<td>7</td>
<td>4</td>
<td>3.5</td>
<td>Medium</td>
</tr>
<tr>
<td>8</td>
<td>1</td>
<td>1</td>
<td>Low</td>
</tr>
<tr>
<td>9</td>
<td>7</td>
<td>4</td>
<td>High</td>
</tr>
<tr>
<td>10</td>
<td>7</td>
<td>5</td>
<td>High</td>
</tr>
<tr>
<td>11</td>
<td>3</td>
<td>1</td>
<td>Low</td>
</tr>
<tr>
<td>12</td>
<td>8</td>
<td>5</td>
<td>High</td>
</tr>
<tr>
<td>13</td>
<td>7</td>
<td>5</td>
<td>High</td>
</tr>
<tr>
<td>14</td>
<td>9</td>
<td>5</td>
<td>High</td>
</tr>
<tr>
<td>15</td>
<td>7</td>
<td>3.5</td>
<td>High</td>
</tr>
<tr>
<td>16</td>
<td>4</td>
<td>2</td>
<td>Medium</td>
</tr>
<tr>
<td>17</td>
<td>2</td>
<td>1</td>
<td>Low</td>
</tr>
<tr>
<td>18</td>
<td>3</td>
<td>1</td>
<td>Low</td>
</tr>
<tr>
<td>19</td>
<td>7</td>
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<td>High</td>
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<td>20</td>
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<td>1</td>
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<td>21</td>
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<td>Medium</td>
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<tr>
<td>22</td>
<td>9</td>
<td>3.5</td>
<td>High</td>
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<tr>
<td>23</td>
<td>1</td>
<td>1</td>
<td>Low</td>
</tr>
<tr>
<td>24</td>
<td>2</td>
<td>1</td>
<td>Low</td>
</tr>
<tr>
<td>25</td>
<td>1</td>
<td>1</td>
<td>Low</td>
</tr>
</tbody>
</table>
**SEQUENCE TO FOLLOW FOR EACH TRIAL**

1. When a green square appears on your screen, this is your signal to set the level of noise and duration of noise that your opponent will hear if he/she loses on this trial.

To select the level of noise you want your opponent to hear, use your mouse to click once on any level from 0 to 10.

To select the duration of noise or how long you want your opponent to hear the noise, use your mouse to click on the specified duration button ranging from 0 seconds to 5 seconds.

You can choose to not blast your opponent with any noise by selecting 0 for both level of noise and duration.

2. Press the spacebar once you have finished step 1 and are ready to proceed to the trial.

3. When a yellow square appears on your screen, this is your cue to get ready.

4. When a red square appears on your screen, press your spacebar as quickly as possible to try to win the trial.

If you **win** the trial (by being the fastest to respond), your opponent will be blasted with the level and duration of noise you have already selected.

If you **lose** the trial (by being the slowest to respond), you will be blasted with the level and duration of noise your opponent has preselected.

**Important Note** - A feedback meter will appear on the left side of your screen. If you won, this feedback tells you what level of noise your opponent has just blasted you with. If you lost, this feedback tells you what level of noise he/she was planning to blast you with.

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*Figure 3.* List of sequences for participant to follow in completing each trial on the Taylor Aggression Paradigm

researcher advised the participant that she was leaving the room to go and check that the other opponent was ready to begin. The researcher returned after 60 seconds to confirm that the other opponent was ready to start. The researcher then pressed a button on the
The computer which activated a message on the screen which confirmed that both players were now connected.

The TAP provides measures of several aspects of aggression. The duration and volume settings of the participants were recorded for each trial on the scales ranging from 0 to 10. Five outcome measures were calculated including: (i) mean noise level selected across 25 trials; (ii) mean noise duration across 25 trials; (iii) noise level chosen on the first trial; (iv) noise duration selected on the first trial; and (v) mean number of times maximum level of noise was used. Table 6 provides a detailed description of the five outcome variables and the specific aspect of aggression each one measured.

The task has been shown to be a reliable measure of aggression and has good convergent, discriminant and construct validity (Anderson & Bushman, 1997; Bernstein, Richardson, & Hammock, 1987; Giancola & Zeichner, 1995). Participants were debriefed following the completion of the study and advised that their supposed opponent was fictitious.
Table 6.

*Description of Outcome Measures Derived from the Taylor Aggression Paradigm.*

<table>
<thead>
<tr>
<th>Description</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Mean noise level</td>
<td>The mean noise level selected by the participant and calculated across the 25 trials. Provides a measure of general, direct aggression.</td>
</tr>
<tr>
<td>2. Mean noise duration</td>
<td>The mean noise duration selected by the participant and calculated across the 25 trials. Provides a measure of more covert aggression.</td>
</tr>
<tr>
<td>3. 1st noise level</td>
<td>The noise level selected by the participant in the first trial. Provides a measure of unprovoked, direct, initial aggression, given that the participant has not been blasted by noise or received any feedback regarding his/her opponent’s intentions.</td>
</tr>
<tr>
<td>4. 1st noise duration</td>
<td>The noise duration selected by the participant in the first trial. Provides a measure of covert, unprovoked aggression.</td>
</tr>
<tr>
<td>5. N level 10</td>
<td>The number of times a participant selected the maximum noise level (level 10 which corresponded to 105 dB). Provides a measure of the participant’s tendency to use extreme aggression against their opponent.</td>
</tr>
</tbody>
</table>

Note: Outcome measures adapted from Kuepper and Hennig (2007).
Disinhibition. A stop signal task called STOP-IT was used to investigate response inhibition (Verbruggen et al., 2008). STOP-IT is a computerised task in which participants are instructed to respond as quickly and accurately as possible to a primary task stimulus (i.e., the GO stimulus). On a subset of trials, the GO stimulus is followed by a STOP signal, at which point, participants are required to inhibit their response to the GO stimulus. Performance on this task is likened to a race-model, which suggests that whether or not a particular response will be inhibited depends on the outcome of a race between two independent processes – the GO process and the STOP process (Schachar & Logan, 1990). If the STOP process finishes first, the response will be inhibited (referred to as signal-inhibit trials). In contrast, if the GO process finishes first, the response will most likely be executed (signal-respond trials) and participants will find it much harder to inhibit the automatic response. As the time delay between the GO stimulus and the STOP signal delay increases, the probability of participants’ responding on STOP signal trials increases (Verbruggen et al., 2008). Thus, the task measures the ability to cancel an ongoing speeded motor response, rather than response restraint (Lipszyc & Schachar, 2010). Response inhibition is dependent upon the relative finishing time of these two processes. The task measures the time that is required to inhibit the response once it has already commenced and a score is calculated – the SSRT score – which provides an outcome measure of inhibition. Longer SSRT scores are associated with greater disinhibition.

In the current study, the STOP-IT task was presented on a laptop computer which was equipped with sound through which auditory signals were presented. There were two GO stimuli used – a square and a circle. On the GO trials, participants were required to respond to the shape of the stimulus. Specifically, when the GO stimulus was a square, participants were instructed to press the left response key (the letter ‘Z’ on
the keyboard of the computer). When the GO stimulus was a circle, participants were instructed to press the right response key (the ‘?’ button on the keyboard). On 25% of the trials, an auditory tone (i.e., a loud beep – the STOP signal) was presented after a variable delay (STOP-signal delay). STOP-signal delay was initially set at 250 milliseconds and was continuously adjusted by the computer program using a tracking procedure to obtain a probability of stopping of .50. STOP-signal delay was decreased by 50ms after unsuccessful stopping and was increased by 50 milliseconds after successful stopping.

The task commenced with a practice block of 32 trials. The practice block was then followed by 3 experimental blocks each containing 64 trials. After each block, the participant was provided with feedback displayed on the computer screen about their performance in the previous block. They were then required to wait for 10 seconds before moving on to the next block. The task took approximately 9 to 11 minutes to complete. A copy of the instructions given to participants is presented in Appendix K.

**Procedure**

Participants who formed the experimental group (MA users) were recruited through notices placed at the Needle Syringe Exchange Service at Logan ATODS. The notice was placed on the counter top and participants’ attention was drawn to this notice as they approached the counter. Individuals in the MA group who expressed an interest in participating in the study were provided with an information sheet (see Appendix L) and encouraged to ask any questions about the study. Potential participants were asked a series of screening questions to ensure they met inclusion criteria. Participants who met inclusion criteria and gave their verbal consent to participate in the study made an appointment time to complete the battery of assessments. Testing of MA users took place in an office at Logan ATODS.
Participants who formed the control group were recruited through posters displayed in local community settings, including neighbourhood centres, community health centres, churches, and childcare centres. These community settings were all located in the same post code region as Logan ATODS. Individuals who expressed an interest in participating in the study were also given a written information sheet (see Appendix L) and invited to raise any questions about the study. Control participants were then asked a series of screening questions to ensure they met inclusion criteria. Eligible participants who wanted to take part in the study were then given an appointment time to complete the test battery. Testing of controls took place in an office at the community organisation.

At the commencement of the testing session, the researcher verbally reiterated the purpose of the study and the nature of the participant’s involvement, and subsequent written consent was obtained (see Appendix M). Participants then completed several self-report measures, followed by the two computer-based behavioural tasks. The total duration of the testing session was approximately 1 hour and 15 minutes.

Results

Data Screening and Assumptions

Prior to analysis, all variables were examined through SPSS (version 17.0) for accuracy of data entry, missing values, and fit between their distributions and the assumptions of parametric and non-parametric tests. Results of these examinations were satisfactory. Data ranges were checked for each variable and were found to lie within valid parameters.

Missing data. Examination of the data showed that one participant was missing data on the STOP-IT task. Closer examination of this participant’s response data indicated long periods of non-response suggesting the participant may not have been
responding to the task appropriately. This participant was excluded from all analyses, leaving 41 participants for further analysis ($n = 20$ in the MA group and $n = 21$ in the control group).

**Normality.** Six variables were found to be significantly positively skewed and showed kurtosis. The extent of skewness ranged from mild to moderate with the greatest skewness found on the TAP task – specifically, the mean number of times a participant punished their fictitious opponent using the maximum noise intensity. This type of skewness is expected on this scale since most participants typically choose to punish using less intense noise levels rather than higher intensity noise levels.

**Transformations.** Square-root transformations improved skewness on the TAP scale (mean duration score, number of times a participant punished their opponent using the maximum noise level score and number of times a participant punished their opponent using the minimum noise level score), and the STOP-IT task (mean percentage of correct responses on no signal trials (see Appendix N). Statistical analyses were performed using both transformed and non-transformed variables to test the influence of skewness in the raw data. Examination of the results showed no influence of the transformed variables on the results. Consequently, analyses are reported using non-transformed raw data.

**Outliers.** The data were screened for outliers and this analysis revealed the presence of four univariate outliers. Two of the univariate outliers were extreme scores on the STOP-IT behavioural task. The other two univariate outliers were extreme scores on the TAP behavioural task. These outliers had no significant effect on the results and were thus retained for further analysis.
Data Analysis

Analyses (chi-square for categorical variables and independent sample t-tests for continuous variables) were performed to determine if there were any significant differences between the MA users and controls. An independent t-test was used to determine whether there were any significant group differences on self-report measures and on the TAP and the STOP-IT task.

Descriptive Statistics

The sample submitted for analysis consisted of 41 participants. It is noted that all of these participants met inclusion criteria for the study, as assessed by the initial screening questions and information derived from the PRISM. There was no significant difference in age between the control group ($M_{\text{years}} = 32.81, SD = 8.89$) and the MA using group ($M_{\text{years}} = 37.25, SD = 7.88$), $t(39) = -1.69, p = .099$. See Table 7 for a description of demographics and sample characteristics. As demonstrated, with the exception of a single variable (employment), there were no significant differences in defining characteristics of individuals in the MA group and those in the control group. Pearson correlations were conducted on self-report measures, behavioural tasks, and key demographic characteristics to confirm the groups were comparable on all major demographic variables, which they were (see Results section).
Table 7.

Demographics and Sample Characteristics (N = 41) % in Parentheses

<table>
<thead>
<tr>
<th></th>
<th>Total Sample (n = 41)</th>
<th>Controls (n = 21)</th>
<th>MA Users (n = 20)</th>
<th>Statistic</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Gender</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>21 (51.2)</td>
<td>10 (47.6)</td>
<td>11 (55.0)</td>
<td>(\chi^2 = 0.22)</td>
</tr>
<tr>
<td>Female</td>
<td>20 (48.8)</td>
<td>11 (52.4)</td>
<td>9 (45.0)</td>
<td></td>
</tr>
<tr>
<td><strong>Living Arrangements</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alone</td>
<td>5 (12.2)</td>
<td>4 (19.0)</td>
<td>1 (5.0)</td>
<td>(\chi^2 = 7.44)</td>
</tr>
<tr>
<td>With Partner</td>
<td>11 (26.8)</td>
<td>4 (19.0)</td>
<td>7 (35.0)</td>
<td></td>
</tr>
<tr>
<td>With Partner &amp; Children</td>
<td>14 (34.1)</td>
<td>10 (47.6)</td>
<td>4 (20.0)</td>
<td></td>
</tr>
<tr>
<td>Sharing with Others</td>
<td>11 (26.8)</td>
<td>3 (14.3)</td>
<td>8 (40.0)</td>
<td></td>
</tr>
<tr>
<td><strong>Marital Status</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single</td>
<td>16 (39.0)</td>
<td>8 (38.1)</td>
<td>8 (40.0)</td>
<td>(\chi^2 = 7.91)</td>
</tr>
<tr>
<td>Defacto/married</td>
<td>20 (48.8)</td>
<td>11 (52.4)</td>
<td>9 (45.0)</td>
<td></td>
</tr>
<tr>
<td>Separated /divorced</td>
<td>5 (12.2)</td>
<td>2 (9.5)</td>
<td>3 (15.0)</td>
<td></td>
</tr>
<tr>
<td><strong>Education</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High School</td>
<td>23 (56.1)</td>
<td>9 (42.9)</td>
<td>14 (70.0)</td>
<td>(\chi^2 = 3.35)</td>
</tr>
<tr>
<td>Trade Qualification</td>
<td>7 (17.1)</td>
<td>5 (23.8)</td>
<td>2 (10.0)</td>
<td></td>
</tr>
<tr>
<td>TAFE</td>
<td>9 (22.0)</td>
<td>6 (28.6)</td>
<td>3 (15.0)</td>
<td></td>
</tr>
<tr>
<td>Undergraduate Degree</td>
<td>2 (4.9)</td>
<td>1 (4.8)</td>
<td>1 (5.0)</td>
<td></td>
</tr>
<tr>
<td><strong>Employment</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unemployed</td>
<td>16 (39.0)</td>
<td>0 (0)</td>
<td>16 (80.0)</td>
<td>(\chi^2 = 30.76^{***})</td>
</tr>
<tr>
<td>Full-Time Employment</td>
<td>13 (31.7)</td>
<td>12 (57.1)</td>
<td>1 (5.0)</td>
<td></td>
</tr>
<tr>
<td>Part-Time Employment</td>
<td>11 (26.8)</td>
<td>9 (42.9)</td>
<td>2 (10.0)</td>
<td></td>
</tr>
<tr>
<td>Home Duties</td>
<td>1 (2.4)</td>
<td>0 (0.0)</td>
<td>1 (5.0)</td>
<td></td>
</tr>
<tr>
<td><strong>Ethnicity</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>34 (83.0)</td>
<td>18 (85.7)</td>
<td>16 (80.0)</td>
<td>(\chi^2 = 2.29)</td>
</tr>
<tr>
<td>Maori</td>
<td>5 (12.0)</td>
<td>3 (14.3)</td>
<td>2 (10.0)</td>
<td></td>
</tr>
<tr>
<td>Aboriginal or TSI</td>
<td>2 (0.50)</td>
<td>0 (0.0)</td>
<td>2 (10.0)</td>
<td></td>
</tr>
<tr>
<td>Estimated IQ</td>
<td>92.5 (100)</td>
<td>92.9 (100)</td>
<td>92.0 (100)</td>
<td>(t = 0.30)</td>
</tr>
</tbody>
</table>

Note. *p < .05. **p < .01. ***p < .001.
Using the diagnostic algorithms provided with the PRISM, 16 MA users (80%) met DSM-IV criteria for MA dependence, 2 MA users (10%) met DSM-IV criteria for MA use, and another 2 MA users (10%) did not meet either criterion. Mean age of first use of MA was 21 years ($SD = 7.65$), with participants using an average of 13.7 days ($SD = 4.54$) in the past 30 days. Eight participants (40%) had used MA at least 15 days in the past month. Similar descriptive information was collected for cannabis and alcohol with age of first regular use being 17.1 years ($SD = 6.72$) and 17.8 years ($SD = 4.21$), respectively. Daily or almost daily use of cannabis was reported by 35% ($n = 7$) of the sample and mean number of days use in the last 30 days was 16.4 ($SD = 12.08$). There was considerably less alcohol use, with no-one reporting daily drinking and 75% ($n = 15$) of the sample reporting nil alcohol consumption in the past 30 days. The mean number of days use per month was 1 ($SD = 2.22$).

Table 8 shows bivariate correlations between self-report measures of impulsivity, hostility, aggression, and substance use in the MA using group. The SDS (MA) was not significantly correlated with self-report measures of impulsivity, aggression, or hostility, although there was a positive trend for hostility and impulsivity ($p < .10$). Similarly, the SDS (MA) was not significantly associated with either behavioural measure. Number of years of dependent MA use, as measured by the PRISM, was positively correlated with mean noise level across 25 trials of the TAP.

As expected, there was a positive association between self-reported aggression (AQ) and self-reported hostility (BPRS). There was also a significant positive correlation between self-reported aggression (AQ) and mean noise level across 25 trials of the TAP. Self-reported hostility (BPRS) was not significantly correlated with performance on the TAP, although there was a trend for mean noise level ($p < .10$).
Impulsive non-conformity was positively associated with self-reported impulsivity (BIS-11), aggression (AQ) and hostility (BPRS). The BIS-11 was also positively related to self-reported aggression (AQ) and hostility (BPRS).

Mean noise level on the TAP correlated positively with the mean number of times maximum noise level was used. Mean noise duration was also positively associated with mean number of times maximum noise level was selected. Finally, noise level selected on the first trial was positively correlated with noise duration selected on the first trial.
Table 8.

*Bivariate Correlations of Self-Report Measures of Impulsivity, Aggression, Personality and Substance Use in MA Users (N = 20)*

<table>
<thead>
<tr>
<th>Scale</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. SDS MA</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. PRISM – MA Years of Dependence</td>
<td>.08</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. SDS Cannabis</td>
<td>.30</td>
<td>-.14</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. BIS-11 Total Score</td>
<td>.30</td>
<td>-.03</td>
<td>.36</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. O-LIFE Imp Non-Conformity</td>
<td>.40</td>
<td>-.08</td>
<td>.48</td>
<td>.66</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. AQ – Total Score</td>
<td>.30</td>
<td>-.23</td>
<td>.35</td>
<td>.80</td>
<td>.80</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. BPRS Hostility</td>
<td>.40</td>
<td>-.17</td>
<td>.49</td>
<td>.67</td>
<td>.62</td>
<td>.76</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. TAP – Mean Noise Level</td>
<td>-.05</td>
<td>.46</td>
<td>.31</td>
<td>.27</td>
<td>.18</td>
<td>.45</td>
<td>.38</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. TAP – Mean Duration Level</td>
<td>-.01</td>
<td>-.28</td>
<td>.44</td>
<td>.10</td>
<td>.11</td>
<td>.29</td>
<td>.30</td>
<td>.82</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. TAP – 1st Noise Level (Unprovoked Agg)</td>
<td>.21</td>
<td>-.22</td>
<td>.18</td>
<td>.06</td>
<td>.23</td>
<td>.22</td>
<td>.27</td>
<td>.31</td>
<td>-.05</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11. TAP – 1st Noise Duration (Unprovoked Agg)</td>
<td>.06</td>
<td>.20</td>
<td>.42</td>
<td>.12</td>
<td>.16</td>
<td>.06</td>
<td>.19</td>
<td>.03</td>
<td>-.01</td>
<td>.53</td>
<td></td>
<td></td>
</tr>
<tr>
<td>12. TAP – Mean No. of Times Noise Level 10 Used</td>
<td>-.07</td>
<td>-.38</td>
<td>.36</td>
<td>.13</td>
<td>-.04</td>
<td>.28</td>
<td>.30</td>
<td>.87</td>
<td>.81</td>
<td>.15</td>
<td>.11</td>
<td></td>
</tr>
<tr>
<td>13. STOP-IT Task – Mean SSRT</td>
<td>.04</td>
<td>.20</td>
<td>.01</td>
<td>.23</td>
<td>.21</td>
<td>.17</td>
<td>-.20</td>
<td>-.13</td>
<td>.01</td>
<td>-.14</td>
<td>.01</td>
<td>-.17</td>
</tr>
</tbody>
</table>

Table 9 displays bivariate correlations between self-report measures of impulsivity, aggression, hostility, the TAP, and the STOP-IT task for the entire sample. As shown in Table 9, there was convergence between self-report measures of aggression (i.e., BPRS hostility, AQ) and the behavioural measure of aggression (i.e., The TAP) across the total sample. Also of note, there was a trend between one of the self-report measures of impulsivity (i.e., BIS-11) and the behavioural measure of disinhibition (STOP-IT task).
Table 9.

*Bivariate Correlations of Self-Report Measures of Impulsivity, Aggression and the TAP in the Total Sample (N = 41)*

<table>
<thead>
<tr>
<th>Scale</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. O-LIFE Imp Non-Conformity</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. BIS-11 Total score</td>
<td>.68**</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. BPRS Hostility</td>
<td>.46**</td>
<td>.54**</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. AQ – Total score</td>
<td>.66**</td>
<td>.70**</td>
<td>.72**</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. TAP Mean noise level</td>
<td>.29</td>
<td>.31*</td>
<td>.31*</td>
<td>.39*</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. TAP Mean noise duration</td>
<td>.12</td>
<td>.13</td>
<td>.42**</td>
<td>.30</td>
<td>.58**</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. TAP 1st noise level</td>
<td>.30</td>
<td>.17</td>
<td>.05</td>
<td>.17</td>
<td>.48**</td>
<td>-.14</td>
<td>-</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. TAP 1st noise duration</td>
<td>.19</td>
<td>.20</td>
<td>.28</td>
<td>.29</td>
<td>.29</td>
<td>.08</td>
<td>.46**</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>9. TAP No. of times selected level 10 (max noise)</td>
<td>.15</td>
<td>.21</td>
<td>.37*</td>
<td>.34*</td>
<td>.75**</td>
<td>.68**</td>
<td>.25</td>
<td>.23</td>
<td>-</td>
</tr>
<tr>
<td>10. STOP-IT Mean SSRT</td>
<td>.17</td>
<td>.30*</td>
<td>-.12</td>
<td>.20</td>
<td>-.01</td>
<td>-.14</td>
<td>.10</td>
<td>-.08</td>
<td>-.11</td>
</tr>
</tbody>
</table>

*Note.* O-LIFE Imp Non Conformity = Oxford Liverpool Inventory of Feelings and Experiences – Impulsive Non-Conformity Subscale, BIS-11 = Barrett Impulsiveness Scale Version 11, BPRS Hostility = TheBrief Psychotic Rating Scale – Hostility Item, AQ Total Score = The Aggression Questionnaire, TAP = The Taylor Aggression Paradigm.*p < .05; **p < .01, *p < .10.
Comparison between groups on behavioural and self-report measures

An independent samples t-test was conducted to compare controls with MA users on a range of self-report measures of impulsivity, aggression, hostility, and behavioural measures of aggression and disinhibition. The results are presented in Table 10 and Table 11, along with means, standard deviations and t values. As hypothesised, MA users reported higher rates of hostility and aggression as measured by both the BPRS and the AQ (see Table 10 for values) than controls. There were no significant differences between MA users and controls on self-reported impulsivity. As can be seen in Table 11, there was a significant difference between MA users and controls on the TAP mean noise duration, the TAP first noise duration, and the TAP number of times participants punished their opponents using the maximum noise level. Notably, MA users responded more aggressively than controls on these measures.
Table 10.

Mean Scores +/- SD and t-values on Self-Report Measures of Impulsivity, Aggression and Inhibition by Group (df = 39)

<table>
<thead>
<tr>
<th>Measure</th>
<th>Control Group</th>
<th>MA Users</th>
<th>( t )</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(n = 21)</td>
<td>(n = 20)</td>
<td></td>
</tr>
<tr>
<td>M (SD)</td>
<td>M (SD)</td>
<td>M (SD)</td>
<td></td>
</tr>
<tr>
<td>BPRS Hostility Item (1-7)</td>
<td>1.43 (0.68)</td>
<td>3.35 (1.90)</td>
<td>-4.27**</td>
</tr>
<tr>
<td>AQ – Physical Aggression</td>
<td>22.00 (6.09)</td>
<td>27.95 (8.22)</td>
<td>-2.64**</td>
</tr>
<tr>
<td>AQ – Verbal Aggression</td>
<td>13.52 (3.41)</td>
<td>17.35 (4.39)</td>
<td>-3.12**</td>
</tr>
<tr>
<td>AQ – Anger</td>
<td>16.00 (4.83)</td>
<td>20.20 (6.73)</td>
<td>-2.30*</td>
</tr>
<tr>
<td>AQ – Hostility</td>
<td>18.05 (4.63)</td>
<td>24.65 (7.72)</td>
<td>-3.30**</td>
</tr>
<tr>
<td>AQ – Total</td>
<td>69.57 (14.35)</td>
<td>90.15 (23.27)</td>
<td>-3.39***</td>
</tr>
<tr>
<td>BIS-11 Total</td>
<td>68.33 (9.50)</td>
<td>71.95 (12.60)</td>
<td>-1.04</td>
</tr>
<tr>
<td>BIS-11 Attentional Impulsiveness</td>
<td>17.10 (3.48)</td>
<td>17.60 (5.34)</td>
<td>-0.36</td>
</tr>
<tr>
<td>BIS-11 Motor Impulsiveness</td>
<td>24.71 (3.99)</td>
<td>27.00 (5.07)</td>
<td>-1.61</td>
</tr>
<tr>
<td>BIS-11 Unplanned Impulsiveness</td>
<td>26.52 (3.99)</td>
<td>27.35 (5.19)</td>
<td>-0.57</td>
</tr>
<tr>
<td>O-LIFE Impulsive Non-Conformity</td>
<td>7.52 (3.60)</td>
<td>8.85 (4.29)</td>
<td>-1.07</td>
</tr>
</tbody>
</table>

Note. BPRS Hostility = The Brief Psychotic Rating Scale – Hostility Item, AQ = The Aggression Questionnaire, BIS-11 = Barrett Impulsiveness Scale Version 11, O-LIFE Imp Non-Conformity = Oxford Liverpool Inventory of Feelings and Experiences – Impulsive Non-Conformity Subscale. *\( p < .05 \); **\( p < .01 \); ***\( p < .001 \).
Table 11.

**Mean Scores +/- SD and T-scores on TAP and STOP-IT by Group (df = 39)**

<table>
<thead>
<tr>
<th>SCALE</th>
<th>Control Group (n = 21)</th>
<th>MA Users (n = 20)</th>
<th>t</th>
</tr>
</thead>
<tbody>
<tr>
<td>TAP – Mean noise level</td>
<td>3.49 (1.52)</td>
<td>4.14 (1.84)</td>
<td>-1.24</td>
</tr>
<tr>
<td>TAP – Mean noise duration</td>
<td>0.95 (0.67)</td>
<td>1.70 (1.48)</td>
<td>-2.07*</td>
</tr>
<tr>
<td>TAP – 1st noise level</td>
<td>4.00 (2.47)</td>
<td>4.05 (1.79)</td>
<td>-.07</td>
</tr>
<tr>
<td>TAP – 1st noise duration</td>
<td>1.53 (0.85)</td>
<td>2.24 (1.22)</td>
<td>-2.18*</td>
</tr>
<tr>
<td>TAP – No. of times level 10 noise used</td>
<td>0.38 (1.32)</td>
<td>1.95 (3.14)</td>
<td>-2.07*</td>
</tr>
<tr>
<td>STOP IT – Mean SSRT</td>
<td>352.15 (134.49)</td>
<td>335.43 (129.90)</td>
<td>.43</td>
</tr>
</tbody>
</table>

*Note.* TAP = The Taylor Aggression Paradigm, SSRT – Stop Signal Reaction Time.

*p < .05.*
Discussion

The primary purpose of Study 2 was to extend previous research by examining the relationship between MA use and aggression, using both a behavioural measure as well as self-report measures of aggression. Results revealed that, as predicted, MA users not only reported high levels of aggression, but they also behaved more aggressively when competing with a person they believed to be their opponent. This is an important finding, given that self-report and behavioural measures frequently do not converge and little prior research has examined the effects of MA use on actual aggressive behaviour. Study 2 also investigated the relationship between MA use and impulsivity, again using both self-report and behavioural measures of impulsivity. Unexpectedly, the prediction that levels of impulsivity would be higher in MA users than in the non-MA using control group was not upheld for any of the impulsivity measures used. This finding is somewhat unexpected in light of the findings from Study 1 and a considerable body of previous research demonstrating increased impulsivity and behavioural disinhibition associated with MA use. These findings will be discussed in greater detail in the sections to follow.

Aggression in Methamphetamine Users

To summarise, findings revealed that MA users reported higher rates of hostility and aggression as measured by both the BPRS and the AQ than controls, and they responded more aggressively than controls with significant differences noted across a range of TAP measures including mean noise duration (considered a measure of covert aggression), first noise duration (considered a measure of unprovoked, covert aggression), and the number of times participants punished their opponents using the maximum noise level (considered a measure of extreme aggression).
Study 2 extends upon the work of Sekine et al. (2006) who demonstrated significant reductions in the density of human brain serotonin transporters in 12 MA users. Notably, this reduction was both significantly correlated with duration of use and with scores on the AQ. This led the authors to conclude that “the present findings indicate that methamphetamine induced serotonergic disturbances are responsible for the elevated aggressiveness that is frequently observed in abstinent methamphetamine users” (Sekine et al., 2006, p. 95). While this is highly plausible, it is not necessarily the case that changes observed in brain function and structure translate to changes at a behavioural level. Hence, using a well validated behavioural task of aggression in a population of MA users was an important contribution to the existing literature and the findings were certainly consistent with the conclusion drawn by Sekine et al (2006).

Results of the current study are also broadly consistent with those of Payer et al. (2011) who found a difference between recently abstinent MA users and non-MA users on a behavioural measure of aggression. Interestingly, however, there are differences across the studies in terms of the specific behavioural effects observed. Both studies used a similar competitive reaction time task involving the delivery of an aversive stimulus (noise) to a fictitious opponent, but the level of provocation was systematically varied across 4 testing blocks by Payer and colleagues, whereas in the current study a mix of low, medium and high provocation conditions were spread throughout a single block which took approximately 10 minutes to complete. The significant effect observed by Payer et al. (2011) was limited to the high provocation condition, with no group differences between MA users and non-users evident during the low and moderate provocation trials. In the current research, on the other hand, the duration of aversive noise displayed by the MA users was greater, on average, across all trials of varying levels of provocation, though there were no group differences in the level
(intensity) of noise demonstrated. Information (i.e., computer feedback) regarding duration was not provided to participants whereas information regarding noise level was made explicit, an important difference that is conceptualised as reflecting the distinction between covert and overt aggression. The pattern of results in Study 2 thus suggests that MA users display a higher level of covert aggression across differing levels of provocation. Also, the finding that MA users punished their opponents with longer noise durations on the first trial of the task suggests higher levels of covert aggression, prior to any provocation occurring at all. Interestingly, the finding that MA users punished their opponents using the maximum noise level more often than controls highlights a greater tendency to display more punitive and extreme aggression. Despite these interesting findings and in light of the differences in the tasks employed in the current research and that of Payer et al. (2011), additional research using the same behavioural measures is needed to further examine these findings. In the meantime, however, the converging evidence across the two studies using somewhat similar behavioural measures of aggression is very encouraging.

As mentioned earlier, in the current study MA users scored significantly higher than controls on the AQ (Buss & Perry, 1992 version; $90 \pm 23$ and $69 \pm 14$ respectively). This finding was also obtained by Sekine et al. (2006) using the same version of the AQ although interestingly, both their MA users and controls scored somewhat lower than the current sample ($75 \pm 13$ and $30.2 \pm 1.7$ for MA users and controls). Direct comparison with Payer et al.’s (2011) sample is more difficult as they used a slightly different version of the AQ (Buss & Warren, 2000), although once again, MA users reported higher scores than controls, as did ecstasy users in Gerra et al.’s (2001) study. Thus, there are now at least three studies using a version of the AQ that have shown elevated scores on self-report measures of aggression in this population.
A particularly interesting finding from the current study was the demonstrated relationship between duration of dependent MA use and performance on one of the outcome measures of the TAP, namely the mean noise level across the 25 trials (a measure of overt aggression). This result is consistent with and adds to the findings of Sekine et al. (2006) who found that severity of self-reported aggression on the AQ paralleled decreases in serotonin transporter density in several key regions in the brain, which in turn, were associated with duration of MA use. Further, the authors argued that based on their deliberate attempts to recruit individuals with no history of ‘abnormal aggression’ prior to the MA-based interviews, that is was unlikely that the increased aggression observed among MA users reflected a broader or pre-existing disposition or personality trait. While the current study did not examine the role of serotonin in the MA-aggression relationship, it did suggest an effect between duration of dependent MA use and actual aggressive responding, suggesting a specific role or action of MA in increasing aggression among users.

**Impulsivity in Methamphetamine Users**

The prediction that MA users would respond more impulsively than non-MA using controls on a behavioural task measuring response inhibition was not supported. Furthermore, no group differences were apparent on the self-report measures of impulsivity either. It is worth noting, however, that although the correlation between severity of MA dependence (SDS) and the same measure of impulsivity (i.e., the O-LIFE) used in Study 1 failed to find conventional levels of significance in Study 2, it did approach significance ($p < .10$).

Given the considerable body of evidence documenting a relationship between MA use and both self-report measures (Perry & Carroll, 2008) and behavioural measures of impulsivity (Monterosso et al., 2005; Salo, Nordahl, Buonocore, et al.,
AGGRESSION IN METHAMPHETAMINE USERS

2009; Salo, Nordahl, Galloway, et al., 2009; Salo et al., 2002; Salo, Ursu, et al., 2009; Simon et al., 2010) and the results of Study 1, the finding of no significant relationship between MA and impulsivity is both puzzling and unexpected. In light of this, careful consideration of both procedural and design issues are warranted.

In the first instance, it is worth considering whether the self-report measures of impulsivity and behavioural task measuring disinhibition employed in the current study truly measured the constructs they purport to. In regards to self-report measures, both the BIS-11 and the O-LIFE have good demonstrated reliability and validity and have been used extensively in prior research, including with stimulant users (Dawe & Loxton, 2004; Duva, Silerstien, & Spiga, 2011; Kjome et al., 2010). Further, the O-LIFE was used in Study 1 and was clearly able to detect varying levels of impulsiveness in a large cohort of MA users. It therefore seems unlikely that the failure to find group differences in impulsivity can be attributed to the particular self-report measures used in Study 2.

In relation to the behavioural measurement of inhibition, it is worth noting that the vast majority of studies investigating behavioural control in MA users have employed the Stroop Test (e.g., Salo, Nordahl, Buonocore, et al., 2009; Salo, Nordahl, Galloway, et al., 2009; Salo, Ursu, et al., 2009) as opposed to the STOP-IT. Despite this, a decision was made to use the STOP-IT task in the current study for several reasons. First, some researchers have argued that compared to all other behavioural measures of response inhibition, stop-signal paradigms have a unique and greater ability to directly assess the ability to inhibit an automatic behavioural response (Monterosso et al., 2005; Quay, 1997). Second, numerous studies have demonstrated that the Stop-Signal task is sensitive to specific elements of behavioural inhibition that are implicated in a range of self-control disorders (Lipszyc & Schachar, 2010; Oosterlaan & Sergeant,
Third, the Stop-Signal task has been used extensively to examine and demonstrate deficits in behavioural inhibition in stimulant abusers, particularly cocaine (Fillmore & Rush, 2002; Fillmore et al., 2002; Li et al., 2006). Finally, one previous study has used the STOP-IT to examine behavioural inhibition in MA users specifically (Monterosso et al., 2005), and clearly found deficits in inhibitory control among MA users compared with non-MA users. In brief, a substantial body of evidence demonstrates that the Stop-Signal Task is a valid and reliable measure of response inhibition that is more than capable of demonstrating differences in performance when they exist.

Taken together, the information presented above provides a persuasive argument that the finding of no significant differences in levels of impulsivity among MA users and non-MA users in the current study is not the result of poorly selected measures. Another possibility to consider, concerns the nature of the control group used in MA research. In many studies, MA users and non-users are similar in age and sex, but little heed has been paid to such factors as education and intelligence. Furthermore, there is some evidence that controlling for differences in education eliminates the difference between MA users and non-users in behavioural disinhibition (Simon et al., 2010). In the current study, considerable care was taken to ensure that the control group was similar in education and intelligence to the MA group which raises the possibility that the use of a well-matched control group attenuates group differences in impulsivity. Unfortunately, this explanation is not viable. In the only other study to compare MA users and non-users on the STOP-IT, Monterosso et al. (2005) found significantly longer reaction times (indicative of greater disinhibition/impulsivity) in the MA users than in the control group – and in this study, the MA users and non-users in the control group were equivalent in education and estimated intelligence.
Another potential explanation for the finding of a non-significant relationship between MA use and impulsivity on the two self-report measures and the behavioural task is the possibility that participants in neither group were particularly impulsive. While a large body of evidence attests to a link between drug use and heightened impulsivity it simply was not observed in the current sample of MA users. Interestingly, in comparing the mean scores obtained on the Impulsive-Non Conformity Scale by both MA users ($M = 8.85, SD = 4.29$) and controls ($M = 7.52, SD = 3.6$) to those obtained from similar aged females ($M = 7.62, SD = 3.86$) and males ($M = 8.58, SD = 3.91$) in the general population, it is apparent that individuals in the MA and non-MA groups were not highly impulsive individuals.

In brief, a consideration of several possible explanations for the failure to find group differences in impulsivity reveals no compelling reason that can satisfactorily account for this finding. In their recent meta-analysis of studies using the STOP-IT, Lipszyc and Schachar (2010) found a small to medium effect size for SSRT deficits associated with substance dependence (primarily cocaine and alcohol dependence). Furthermore, although most studies have found differences in impulsivity between MA users and non-users, several have not (e.g., Chang et al., 2002; Kalechstein, Newton, & Green, 2003). Discrepant findings across studies, including the current study, may simply reflect “variability given an underlying modest effect” (Monterosso et al., 2005, p. 274).

**Contributions and Limitations of the Current Study**

This study has made a substantial contribution to the existing literature base concerning the relationship between MA use and aggression in so far as it is one, of only two studies conducted to date, to behaviourally examine aggression in MA users using a controlled laboratory task.
Another significant strength of Study 2 relates to the use of a carefully and purposefully selected control group who, with the exception of being employed, did not differ on any important demographic characteristics which could confound findings, and have in fact been found to (e.g., Simon et al., 2010). As mentioned previously, control participants were recruited from the same community and geographical post code as the Needle Syringe Exchange Service which is located in a low socioeconomic area characterised by high rates of unemployment and substantial social and economic disadvantage (Australian Bureau of Statistics, 2010).

Finally, another important strength of this research relates to the use of the PRISM, a gold standard instrument designed to specifically increase the accuracy of diagnosis in substance using individuals, which allowed the accurate identification of number of years of dependent MA use. This information was then able to be used to demonstrate a direct effect between greater years of dependent MA use on higher levels of perpetrated aggression.

As with all research, this study also has several limitations that require consideration. The most obvious limitation relates to the small sample size. It is noted that the sample size in the current research (20 MA users and 21 non-MA using controls) is comparable, and in some cases, greater than samples used in other behavioural studies. For example, Payer et al. (2011) behaviourally assessed aggression in a sample of 12 MA users and 15 control participants, while Gerra et al. (2001) performed their behavioural investigation with a sample of 12 ecstasy users and 20 non-using controls. Nevertheless, it is acknowledged that this sample size is still relatively small. Consequently, post hoc power analyses were conducted to determine the effect sizes of the behavioural measures of aggression. Using TAP duration as the variable of interest, a large effect size was found (Hedge’s g = 0.63; Cohen 1988). Similarly, a
large effect size was found for TAP first noise level (Hedge’s g = 0.67) and an even larger effect size was found for TAP mean duration (Hedge’s g = 1.91). The finding that effect sizes were large indicates that the sample size was adequate to detect group differences in aggression. Nevertheless, it is acknowledged that Study 2 failed to find the expected significant group differences on both self-report and behavioural measures of impulsivity/behavioural inhibition. Given the large body of reviewed research attesting to problems of impulse control in MA users compared to non-users, it is possible that such effects might have been found in a larger sample and raises the possibility of Type II error (falsely accepting the null hypothesis and failing to find a significant effect that actually exists). A common reason for Type II error is an inadequate sample size resulting in reduced power to detect significant effects. Thus, a clear goal of future research is to replicate the present study with a larger sample size, thereby substantially increasing power and reducing the possibility of Type II error.

A second limitation relates to the reliance on self-report measures of substance use as opposed to more reliable methods, such as urine screening. Although the sample of participants recruited for this study identified MA as being the primary drug they used, with relatively few individuals reporting other current illicit drug or alcohol use, it is possible that these reports were inaccurate. Indeed, a large problem in conducting research into the specific effects of MA relates to the common problem of poly drug use. Nevertheless, participants were not told they would be excluded from this study if they were dependent on any other drugs, except for MA, cannabis and/or nicotine, which hopefully encouraged accurate reporting of drug use. Given that 8 participants met dependence for cannabis, it is worth questioning whether cannabis may have affected aggressive behaviour. There is however, no evidence linking cannabis use to aggressive behaviour (Macleod et al., 2004). For example, cannabis use is not
associated with aggression in either student sample (Schaub, Boesch, & Stohler, 2006), or in psychiatric patients (Dhossche, 1999). Further, animal studies have predominantly found that cannabis administration induces passive and subservient behaviours, and curbs attacking behaviour (Hoaken & Stewart, 2003). While this remains to be tested empirically by using cannabis as a covariate in future studies, it is reasonable to argue that this did not affect results in the current study.

The difference between the behavioural task of aggression employed in the present research and that employed by Payer et al. (2011) also warrants further mention. As discussed above, the TAP design used in the current study consisted of 25 low, medium and high provocation (i.e., the duration and intensity of the noise delivered by the “opponent”) trials which were spread throughout a single block which took approximately 10 minutes to complete. While this design allowed examination of aggressive responding across trials of varying provocation, it was not possible to ascertain whether a particular level of intensity or provocation was more likely to elicit aggressive behaviour. Payer and her colleagues, on the other hand, were able to conduct this type of investigation by using a different task design which involved gradually increasing the level of provocation across subsequent blocks. Such design was used to identify higher levels of aggressive responding in MA users than controls on trials which involved peak provocation. Nonetheless, it is acknowledged that even this design could be associated with potential limitations. For instance, the peak provocation trials were included in the final block of the task, which raises the possibility that given participants had already completed three prior blocks (the first block containing a single practice trial, the second block containing 8 trials of low provocation, and the third block containing 8 trials of moderate provocation), they may have already been feeling quite frustrated by the time they reached block 4. Thus, it is
difficult to ascertain whether the increased levels of aggression MA users displayed on the final block were more accurately explained by ongoing and persistent provocation or whether they were in fact directly associated with peak provocation per se. Nevertheless, taken together, findings from both behavioural tasks shed some light on the possible processes underlying aggressive behaviour in MA users.

**Concluding Comments and Future Research**

Previous research on the relationship between MA and aggression has relied on self-report measures. The present study therefore makes an important contribution to current literature by clearly demonstrating the effects of MA on aggressive behaviour itself. Furthermore, this finding converges nicely with the recently reported Payer et al. (2011) research, indicating that the increased aggression expressed in the *actual behaviour* of MA users, appears to be a robust phenomenon.

While the possibility of Type I error needs to be considered as five t-tests were conducted on the TAP data, it is notable that three of these met significance at the .05 level. In each instance, the MA users were more aggressive than participants in the control group. The consistency in this pattern of results substantially reduces the possibility of Type I errors in reporting the aggression results in the current study.

The finding that significant group differences in aggression emerged despite there being no significant differences in impulsivity suggests the presence of at least another factor that may have exerted an influence on the level of aggression observed. The first contender may be the presence of subclinical or clinical symptoms of psychosis – that in turn influence suspiciousness, paranoia and ultimately defensive aggressive behaviour. However, considerable care was taken to exclude participants with current psychotic symptoms or a history of psychosis. In accordance with emerging research, it is possible that impaired social cognitive functioning may have
been present in the sample of MA users included in this study, which in turn, may have contributed to the heightened levels of aggression observed. While the current study did not focus on social-cognitive deficits in MA users, but rather focused on other equally important variables, emerging evidence in this area is indeed interesting.

Clearly a direction for future research is to further explore the relationship between MA use and aggression, by taking into account the complex interplay between three sets of factors that have already been identified as possible mechanisms influencing the effects of MA use on aggression. Two of these factors have been considered in the current thesis – impulsivity or impaired ability to inhibit behaviour and positive symptoms of psychosis. The third factor, social cognitive deficits, has been considered in a small number of very recent studies. An examination of how these three mechanisms influence aggression in MA users would require a large study which would undoubtedly explore the complex interplay among these factors.

Clinical Implications

The findings of the present research have important clinical implications for treatment interventions designed to reduce the adverse consequences of MA use. Firstly, findings have highlighted the important role of proper assessment of comorbid psychopathology so that MA users receive appropriate and optimal intervention. Accurate screening and assessment of key clinical indicators, including positive psychotic symptoms, would facilitate identification of MA users for whom psychiatric intervention may be warranted. Previous research has demonstrated that early treatment of subclinical symptoms of psychosis results in delayed onset of psychosis and possible reductions in the incidence of psychosis (McGorry, 2002). Further, early interventions that focus on paranoid symptoms may also reduce the possibility of benign situations being misinterpreted as threatening, and thereby reduce levels of hostility and
aggression. The addition of an intervention designed to enhance behavioural regulation and control, and thereby increase the ability to inhibit or control hostile feelings and actions would have further beneficial effects on the high levels of hostility displayed by some MA users.

The issues of MA users presenting at emergency departments of hospitals in an agitated and aggressive manner has been highlighted as an ongoing public concern. There is a clear need for development of management strategies to quickly dissipate the likelihood of aggressive outbursts in these environments. Taking this problem into consideration and in applying the findings of the present research, a key focus of such an intervention involves improving behavioural control. Recent research by (Gailliot et al., 2007) suggests the form that such an intervention could take. Based on evidence that acts of self-control impair subsequent attempts at self-control, (Gailliot et al., 2007) argued that self-control relied on a limited energy source that is readily depleted. They further proposed that glucose might be a vital source of energy for self-control. In a series of experiments designed to investigate this proposition, it was found that blood glucose levels dropped significantly following acts of self-control and that low levels of glucose following a self-control task were associated with poor self-regulation on a subsequent task. Finally, and of particular importance, they demonstrated that increasing blood glucose (by providing a glucose beverage) led to improved self control. Thus, a short term, practical intervention that might serve to reduce physical aggression directed at front-line hospital workers involves providing MA users with a highly concentrated glucose drink. Such an intervention could reduce the risk of aggressive outbursts in clinic and hospital settings.

The current research also has important implications for the wider health policy regarding treating MA users. Given the apparent limited success of treatment
approaches to MA abuse (e.g. Knapp, Soares, Farrell, & Silva de Lima, 2007), it seems timely to target specific features or psychological symptoms that are associated with MA use, drawing from both the clinical and experimental literature. In Australia, substance issues and psychological issues are currently dealt with by two separate departments – one aimed at treating addictions and the other aimed at treating other psychological disorders. The knowledge that MA use is associated with a range of psychological problems, including psychotic symptoms and aggression, highlights the need for a unified service that works to address the multiple consequences of MA use. Individually tailored treatments could be developed from a suite of clinical modules, focusing on diet, affect regulation, subclinical positive symptoms and daily living skills.

Another important implication of the current research is the knowledge that a large, prospective study investigating the course of the relationship between MA use and aggression is firmly warranted. A longitudinal study, with rigorous design, incorporating a combination of self-report measures, experimental tasks and possibly even biological markers, would clearly contribute to a level of understanding of this complex relationship beyond that which currently exists. Given the magnitude of the problem for not only individual users, but also those who they encounter in the wider community, including front line workers and members of the general public, the implementation of these recommendations through well designed studies would provide additional information with which to move forward in the task of preventing harm caused by MA use. Exploring the long-term outcomes of MA use on the behaviour of users would provide valuable insights into understanding why some users display high levels of aggression, while others do not.

The present research has further highlighted the need to systematically examine gender differences in the expression of aggression in MA users. As discussed, this is an
important aspect of any study on aggression as there is evidence of gender differences in both the expression of aggression, hostility (e.g. Bettencourt & Miller, 1996) and in impulsiveness (Cross et al., 2011; Mason & Claridge, 2006). A large sample size, ensuring adequate power and containing equal numbers of men and women, would allow for the exploration of possible gender differences, which could then in turn, drive tailored interventions.
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Salo, R., Nordahl, T. E., Possin, K., Leamon, M., Gibson, D. R., Galloway, G. P.,... S.


Appendix A
Demographic Data Collection Sheet – Study 1

1. Age............... years

2. Sex: Male......0 Female......1

3. Present Occupation: _________________________
   Not employed ........................................... 0
   Full time ................................................. 1
   Part time/casual ........................................ 2
   Student .................................................... 3
   Home duties .............................................. 4

4. Marital Status:
   Single................................. 0
   Defacto ................................................. 1
   Married ............................................... 2
   Separated ............................................. 3
   Divorced ............................................. 4
   Widowed ............................................. 5

5. What are your current living arrangements?
   Alone .................................................... 0
   With partner .......................................... 1
   With family ........................................... 2
   Share accommodation ......................... 3
   Other .................................................... 4
   Specify:____________________

6. What is the highest level of formal education you have obtained?
   High school................................. 0 (Specify grade___________)
   Trade qualification ....................... 1
   TAFE .................................................. 2
   Undergraduate ......................... 3
   Postgraduate .................................. 4

7. What is the main language you speak at home (or first language if participant lives alone)?
   English................................. 1
   Other................................. 2 (Specify____________________)
8. What is your country of birth:

Australia..................................1
Other.......................................2 (Specify_________________)  

9. What is the country of birth of your biological mother and father?

Mother __________________
Father __________________

10. What ethnic group do you identify with?
____________________________________________________________________________

11. Have you ever been diagnosed with a psychiatric problem?

No 0........................................
Yes 1......................................

Specify:
____________________________________________________________________________

12. Have you ever taken medication for a psychiatric problem?

No 0........................................
Yes 1......................................

<table>
<thead>
<tr>
<th>Specify name and circle all that apply below:</th>
<th>Specify what participant was medicated for:</th>
</tr>
</thead>
<tbody>
<tr>
<td>None ........................................</td>
<td></td>
</tr>
<tr>
<td>Antipsychotic 1 (e.g. Zyprexa, Olanzapine, Chlorpromazine, Clozapine, Risperidone, Aripiprazole)</td>
<td></td>
</tr>
<tr>
<td>Benzodiazepines 2 (e.g. Valium, Rohys, Serepax, Temazepam)</td>
<td></td>
</tr>
<tr>
<td>Antidepressant 3 (e.g. Tryptanol, Prozac, Prothiaden, Sinequan, Zoloft)</td>
<td></td>
</tr>
<tr>
<td>Other ......................................</td>
<td>Specify:________________</td>
</tr>
</tbody>
</table>
13. Have you taken any medication in the past month for a psychiatric problem?

No 0........................................
Yes 1........................................

If Yes:

<table>
<thead>
<tr>
<th>(a) Name?</th>
<th>(b) Taken for how long? (days)</th>
<th>(c) Dose?</th>
</tr>
</thead>
<tbody>
<tr>
<td>i)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ii)</td>
<td></td>
<td></td>
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<td>iii)</td>
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<td>iv)</td>
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<td>v)</td>
<td></td>
<td></td>
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<tr>
<td>vi)</td>
<td></td>
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</tbody>
</table>

14. Have you ever gone to a psychiatric facility, emergency department, hospital or health service to seek help with a psychiatric problem?

No 0........................................
Yes 1........................................

If Yes:

<table>
<thead>
<tr>
<th>(a) Where?</th>
<th>(b) Details of Problem</th>
<th>(c) For how long? (days)</th>
</tr>
</thead>
<tbody>
<tr>
<td>i)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ii)</td>
<td></td>
<td></td>
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<td>iii)</td>
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<td></td>
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<td>v)</td>
<td></td>
<td></td>
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<tr>
<td>vi)</td>
<td></td>
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</tr>
</tbody>
</table>

Now I would like to ask some questions about your family

15. Do you know if anyone in your family has had a history of schizophrenia, bipolar disorder, anxiety or depression?

No 0........................................
Yes 1

If yes which ones?

Who?
Appendix B
Timeline Follow Back Method Calendar

INSTRUCTIONS: Try to recall the drugs (show list) you have used during the last month. Record any events (birthdays, parties, pay days etc.) on the calendar to help you remember. **List whether or not substance was used on that day, the number of times and the total amount of drug used during the day.**

<table>
<thead>
<tr>
<th>Monday</th>
<th>Tuesday</th>
<th>Wednesday</th>
<th>Thursday</th>
<th>Friday</th>
<th>Saturday</th>
<th>Sunday</th>
</tr>
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<tbody>
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</table>

Total MA Use: _____ past 7 days  
_____ past 14 days  
_____ past 30 days  

Total Cannabis Use: _____ past 7 days  
_____ past 14 days  
_____ past 30 days  

Total _______ Use: _____ past 7 days  
_____ past 14 days  
_____ past 30 days  

Total _______ Use: _____ past 7 days  
_____ past 14 day  
_____ past 30 day
Severity of Dependence Scale

**METHAMPHETAMINE**

The following questions are about your methamphetamine use in the past 12 months, including all the different forms of methamphetamine that we have been discussing like speed powder, ice and base.

**Over the past 12 months:**

1. Did you think your speed/meth use was out of control?
   - 0 never or almost never
   - 1 sometimes
   - 2 often
   - 3 always or nearly always

2. Did the prospect of missing a hit/dose of speed/meth make you anxious or worried?
   - 0 never or almost never
   - 1 sometimes
   - 2 often
   - 3 always or nearly always

3. Did you worry about your use of speed/meth?
   - 0 never or almost never
   - 1 sometimes
   - 2 often
   - 3 always or nearly always

4. Did you wish you could stop using speed/meth?
   - 0 never or almost never
   - 1 sometimes
   - 2 often
   - 3 always or nearly always

5. How difficult would you find it to stop, or to go without speed/meth?
   - 0 not difficult
   - 1 quite difficult
   - 2 very difficult
   - 3 impossible

**SCORE**  _____/15
Appendix D
Brief Psychiatric Rating Scale

INSTRUCTIONS:
Rate items 1 – 10 and 19-22, on the basis of patient’s self-report.
Rate questions 11-18, 23-24 based on observations of patient during the interview.
Ratings of 2-3 are given when symptoms are of a non-pathological intensity, whereas ratings of 4-7 indicate variation in pathological intensities.

Mark X to the left of the item that best characterises the patient now

1. Somatic concern

Degree of concern over present bodily health. Rate the degree to which physical health is perceived as problem by the participant, whether complaints have a realistic basis or not. Somatic delusions should be rated in the severe range with or without somatic concern. Note: Be sure to assess the degree of impairment due to somatic concerns only and not other symptoms, e.g., depression. In addition, if the participant rates a ‘6’ or a ‘7’ due to somatic delusions, then you must rate Unusual Thought Content at least a ‘4’ or above.

Have you been concerned about your physical health?

Have you been concerned about your physical health?
Have you had any physical illness or seen a medical doctor?

**OPTIONAL – ADDITIONAL PROBES

–“What does the doctor say is wrong?”
–“Have your concerns interfered with your ability to perform your usual activities or work?”
–“Has anything changed in regards to your appearance?”

<table>
<thead>
<tr>
<th>1 = Not present</th>
<th>Occasional somatic concerns that tend to be kept to self.</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 = Very Mild</td>
<td>Occasional somatic concerns that tend to be voiced to others (e.g., family, physician).</td>
</tr>
<tr>
<td>3 = Mild</td>
<td>Frequent expressions of somatic concern or exaggerations of existing ills OR some preoccupation and moderate impairment of functioning. Not delusional.</td>
</tr>
<tr>
<td>4 = Moderate</td>
<td>Frequent expressions of somatic concern or exaggeration of existing ills OR some preoccupation and moderate impairment of functioning. Not delusional.</td>
</tr>
<tr>
<td>5 = Moderately Severe</td>
<td>Frequent expressions of somatic concern or exaggeration of existing ills OR some preoccupation and moderate impairment of functioning. Not delusional.</td>
</tr>
<tr>
<td>6 = Severe</td>
<td>Preoccupation with somatic complaints with much impairment in functioning OR somatic delusions without acting on them or disclosing to others.</td>
</tr>
<tr>
<td>7 = Extremely Severe</td>
<td>Preoccupation with somatic complaints with much impairment in functioning OR somatic delusions that tend to be acted on or disclosed to others.</td>
</tr>
</tbody>
</table>
2. Anxiety

Reported apprehension, tension, fear, panic or worry. Rate only participant’s statements, not observed anxiety which is rated under Tension.

Have you been worried a lot during [time frame]?
Have you been nervous or apprehensive? (What are you worried about?)
Are you concerned about anything? How about finances or the future?
When you are feeling nervous, do your palms sweat or does your heart beat fast (or shortness of breath, trembling, choking)?

[If participant reports anxiety or autonomic accompaniment, ask the following]:

How much of the time have you been [use participant’s description]?
Has it interfered with your ability to perform your usual activities/work?

<table>
<thead>
<tr>
<th>1 = Not present</th>
<th>Reports some discomfort due to worry OR infrequent worries that occur more than usual for most normal individuals.</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 = Very Mild</td>
<td>Worried frequently but can readily turn attention to other things.</td>
</tr>
<tr>
<td>3 = Mild</td>
<td>Worried most of the time and cannot turn attention to other things easily but no impairment in functioning OR occasional anxiety with autonomic accompaniment but no impairment in functioning.</td>
</tr>
<tr>
<td>4 = Moderate</td>
<td>Frequent, but not daily, periods of anxiety with autonomic accompaniment OR some areas of functioning are disrupted by anxiety or worry.</td>
</tr>
<tr>
<td>5 = Moderately Severe</td>
<td>Anxiety with autonomic accompaniment daily but not persisting throughout the day OR many areas of functioning are disrupted by anxiety or constant worry.</td>
</tr>
<tr>
<td>6 = Severe</td>
<td>Anxiety with autonomic accompaniment persisting throughout the day OR most areas of functioning are disrupted by anxiety or constant worry.</td>
</tr>
<tr>
<td>7 = Extremely Severe</td>
<td>Anxiety with autonomic accompaniment persisting throughout the day OR most areas of functioning are disrupted by anxiety or constant worry.</td>
</tr>
</tbody>
</table>
3. Depression

Include sadness, unhappiness, anhedonia, and preoccupation with depressing topics (can’t switch attention to TV or conversations due to depression), hopelessness, loss of self-esteem (dissatisfied or disgusted with self or feelings of worthlessness). Do not include vegetative symptoms, e.g., motor retardation, early waking, or a lack of motivation that accompanies the deficit syndrome.

*How has your mood been lately?*

*Have you felt depressed (sad, down, unhappy, as if you didn’t care)?*

*Are you able to switch your attention to more pleasant topics when you want to?*

*Do you find that you have lost interest in or get less pleasure from things you used to enjoy, like family, friends, hobbies, watching TV, eating?*

*Have you lost interest in things you used to enjoy?*

*[If participant reports feelings of depression, ask the following]:*

*How long do these feelings last? Has it interfered with your ability to perform your usual activities/work?*

<table>
<thead>
<tr>
<th>1 = Not present</th>
<th>Occasionally feels sad, unhappy or depressed.</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 = Very Mild</td>
<td>Frequently feels sad or unhappy but can readily turn attention to other things.</td>
</tr>
<tr>
<td>3 = Mild</td>
<td>Frequent periods of feeling very sad, unhappy, moderately depressed, but able to function with extra effort</td>
</tr>
<tr>
<td>4 = Moderate</td>
<td>Frequent, but not daily, periods of deep depression OR some areas of functioning are disrupted by depression.</td>
</tr>
<tr>
<td>5 = Moderately Severe</td>
<td>Deeply depressed daily but not persisting throughout the day OR many areas of functioning are disrupted by depression.</td>
</tr>
<tr>
<td>6 = Severe</td>
<td>Deeply depressed daily OR most areas of functioning are disrupted by depression.</td>
</tr>
<tr>
<td>7 = Extremely Severe</td>
<td>Deeply depressed daily OR most areas of functioning are disrupted by depression.</td>
</tr>
</tbody>
</table>
4. **Suicidality**

Expressed desire, intent or actions to harm or kill self.

*Have you felt that life wasn’t worth living?*
*Have you thought about harming or killing yourself?*
*Have you felt tired of living or as thought you would be better off dead?*
*Have you ever felt like ending it all?*

**[If participant reports suicidal ideation, ask the following]:**

*How often have you thought about [use participant’s description]?*
*Did you (Do you) have a specific plan?*

<table>
<thead>
<tr>
<th>1 = Not present</th>
<th>2 = Very Mild</th>
<th>3 = Mild</th>
<th>4 = Moderate</th>
<th>5 = Moderately Severe</th>
<th>6 = Severe</th>
<th>7 = Extremely Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Occasional feelings of being tired of living. No overt suicidal thoughts.</td>
<td>Occasional suicidal thoughts without intent or specific plan OR he/she feels they would be better off dead.</td>
<td>Suicidal thoughts frequent without intent or plan.</td>
<td>Many fantasies of suicide by various methods. May seriously consider making an attempt with specific time and plan OR impulsive suicide attempt using non-lethal method or in full view of potential saviours.</td>
<td>Clearly wants to kill self. Searches for appropriate means and time, OR potentially serious suicide attempt with patient knowledge of possible rescue.</td>
<td>Specific suicidal plan and intent (e.g., “as soon as ______ I will do it by doing X”), OR suicide attempt characterised by plan patient thought was lethal or attempt in secluded environment.</td>
</tr>
</tbody>
</table>
5. Guilt

Over concern or remorse for past behaviour. Rate only participant’s statements, do not infer guilt feelings from depression, anxiety or neurotic defences. Note: If the participant rates a ‘6’ or ‘7’ due to delusions of guilt, then you must rate Unusual Thought Content at least a ‘4’ or above depending on level of preoccupation and impairment.

Is there anything you feel guilty about?
Have you been thinking about past problems?
How bad does it make you feel?
Do you tend to blame yourself for things that have happened?
Have you done anything you’re still ashamed of?

[If participant reports guilt/remorse/delusions, as the following]:

How often have you been thinking about [use participant’s description]?
Have you disclosed your feelings of guilt to others?

<table>
<thead>
<tr>
<th>Rating</th>
<th>Description</th>
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<tbody>
<tr>
<td>1 = Not present</td>
<td>Concerned about having failed someone or at something but not preoccupied. Can shift thoughts to other matters easily.</td>
</tr>
<tr>
<td>2 = Very Mild</td>
<td>Concerned about having failed someone or at something but not preoccupied. Can shift thoughts to other matters easily.</td>
</tr>
<tr>
<td>3 = Mild</td>
<td>Concerned about having failed someone or at something with some preoccupation. Tends to voice guilt to others.</td>
</tr>
<tr>
<td>4 = Moderate</td>
<td>Disproportionate preoccupation with guilt, having done wrong, injured others by doing or failing to do something, but can readily turn attention to other things.</td>
</tr>
<tr>
<td>5 = Moderately Severe</td>
<td>Preoccupation with guilt, having failed someone or at something, can turn attention to other things, but only with great effort. Not delusional.</td>
</tr>
<tr>
<td>6 = Severe</td>
<td>Delusional guilt OR unreasonable self-reproach very out of proportion to circumstances. Moderate preoccupation present.</td>
</tr>
<tr>
<td>7 = Extremely Severe</td>
<td>Delusional guilt OR unreasonable self-reproach grossly out of proportion to circumstances. Participant is very preoccupied with guilt and is likely to disclose to others or act on delusions.</td>
</tr>
</tbody>
</table>
6. **Hostility**

Animosity, contempt, belligerence, threats, arguments, tantrums, property destruction, fights, and any other expression of hostile attitudes or actions. Do not infer hostility from neurotic defences, anxiety or somatic complaints. Do not include incidents of appropriate anger or obvious self-defence.

*How have you been getting along with people (family, co-workers etc.)*?

*How have you been getting along with others?*

*Have you been irritable or grumpy lately? (How do you show it? Do you keep it to yourself?)*

*Were you ever so irritable that you would shout at people or start fights or arguments? (Have you found yourself yelling at people you didn’t know?)*

*Have you hit anyone recently?*

*Have you destroyed any property?*

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<tr>
<td>1</td>
<td>Not present</td>
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<td>2</td>
<td>Very Mild</td>
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<td>3</td>
<td>Mild</td>
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<tr>
<td>4</td>
<td>Moderate</td>
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<td>5</td>
<td>Moderately Severe</td>
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<td>6</td>
<td>Severe</td>
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<td>7</td>
<td>Extremely Severe</td>
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</table>
7. **Elevated Mood**

A pervasive, sustained and exaggerated feeling of well-being, cheerfulness, euphoria (implying a pathological mood), optimism that is out of proportion to the circumstances. Do not infer elation from increased activity or from grandiose statements alone.

*Have you felt so good or high that other people thought that you were not your normal self?*  
*Have you been feeling cheerful and ‘on top of the world’ without any reason?*

**[If participant reports elevated mood/euphoria, ask the following]:**

*Did it seem like more than just feeling good? How long did that last?*

*Have you been feeling cheerful and on top of the world without any reason?*

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<th>1 = Not present</th>
<th>2 = Very Mild</th>
<th>3 = Mild</th>
<th>4 = Moderate</th>
<th>5 = Moderately Severe</th>
<th>6 = Severe</th>
<th>7 = Extremely Severe</th>
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<tr>
<td></td>
<td>Seems to be very happy, cheerful without much reason.</td>
<td>Some unaccountable feelings of well-being that persist.</td>
<td>Reports excessive or unrealistic feelings of well-being, cheerfulness, confidence or optimism inappropriate to circumstances, some of the time. May frequently joke, smile, be giddy or overly enthusiastic OR few instances of marked elevated mood with euphoria.</td>
<td>Reports excessive or unrealistic feelings of well-being, confidence or optimism inappropriate to circumstances much of the time. May describe feeling &quot;on top of the world&quot;, “like everything is falling into place”, or “better than ever before”, OR several instances of marked elevated mood with euphoria.</td>
<td>Reports many instances of marked elevated mood with euphoria OR mood definitely elevated almost constantly throughout interview and inappropriate to content.</td>
<td>Patient reports being elated or appears almost intoxicated, laughing, joking, giggling, constantly euphoric, feeling invulnerable, all inappropriate to immediate circumstances.</td>
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8. Grandiosity

Exaggerated self-opinion, self-enhancing conviction of special abilities or powers or identity as someone rich or famous. Rate only participant’s statements about himself, not their demeanour. **Note:** If the participant rates a ‘6’ or ‘7’ due to grandiose delusions, you must rate Unusual Thought Content at least a ‘4’ or above.

*Do you have talents or abilities that most people don’t have?*

*Is there anything special about you?*

*Do you have any special abilities or powers?*

*Have you thought that you might be somebody rich or famous?*

If the participant reports any grandiose ideas/delusions, ask the following:

*How often have you been thinking about [use participant’s description]?*

*Have you told anyone about what you have been thinking?*

*Have you acted on any of these ideas?*

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<tr>
<th>1 = Not present</th>
<th>Feels great and denies obvious problems, but not unrealistic.</th>
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<tr>
<td>2 = Very Mild</td>
<td>Exaggerated self-opinion beyond abilities and training.</td>
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<td>3 = Mild</td>
<td>Inappropriate boastfulness, claims to be brilliant, insightful, or gifted beyond realistic proportions, but rarely self-discloses or acts on these inflated self-concepts. Does not claim that grandiose accomplishments have actually occurred.</td>
</tr>
<tr>
<td>4 = Moderate</td>
<td>Same as 4 but often self-discloses and acts on these grandiose ideas. May have doubts about the reality of the grandiose ideas. Not delusional.</td>
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<tr>
<td>5 = Moderately Severe</td>
<td>Delusional – claims to have special powers like ESP, to have millions of dollars, invented new machines, worked at jobs when it is known that they were never employed in these capacities, be Jesus Christ, or the President. Participants may not be very preoccupied.</td>
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<tr>
<td>6 = Severe</td>
<td>Delusional – same as 6 but participant seems very preoccupied and tends to disclose or act on grandiose delusions.</td>
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<tr>
<td>7 = Extremely Severe</td>
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</table>

[If the participant reports any grandiose ideas/delusions, ask the following]:

*How often have you been thinking about [use participant’s description]?*

*Have you told anyone about what you have been thinking?*

*Have you acted on any of these ideas?*
9. Suspiciousness

Expressed or apparent belief that other persons have acted maliciously or with discriminatory intent. Include persecution by supernatural or other non-human agencies (e.g. the devil). Note: ratings of ‘3’ or above should also be rated under Unusual Thought Content.

Have you been getting along with others?
Do you ever feel uncomfortable as if other people are watching you?
Have you been spending time with people? If not why?
Do you ever feel uncomfortable in public?
Does it seem as though others are watching you?
Are you concerned about anyone’s intentions toward you?
Is anyone going out of their way to give you a hard time, or trying to hurt you?
Do you feel in any danger?

[If patient reports any persecutory ideas/delusions, ask the following]:

How often have you been concerned that [use participant’s description]?
Have you told anyone about these experiences?

**IF RATED 5 OR ABOVE, ASK THE FOLLOWING QUESTIONS:**
1. Were you using drugs or alcohol at the time you started experiencing/feeling this?
2. Did you think this symptom was related to your drug/alcohol use?
3. Did you experience this when you weren’t using any drugs/alcohol or using less than 4 days per week?

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<td>1</td>
<td>Not present</td>
</tr>
<tr>
<td>2</td>
<td>Very Mild</td>
</tr>
<tr>
<td>3</td>
<td>Mild</td>
</tr>
<tr>
<td>4</td>
<td>Moderate</td>
</tr>
<tr>
<td>5</td>
<td>Moderately Severe</td>
</tr>
<tr>
<td>6</td>
<td>Severe</td>
</tr>
<tr>
<td>7</td>
<td>Extremely Severe</td>
</tr>
</tbody>
</table>

1 = Not present
2 = Very Mild
3 = Mild
4 = Moderate
5 = Moderately Severe
6 = Severe
7 = Extremely Severe


Describes incidents where other persons have harmed or wanted to harm him/her that sound plausible. Feels as if others are watching, laughing at or criticizing him/her in public, but this occurs only occasionally or rarely. Little or no preoccupation.

Says other persons are talking about him/her maliciously or says others intent to harm him/her. Beyond likelihood of plausibility but not delusional. Incidents of suspected persecution occur occasionally with some preoccupation.

Same as 4, but incidents occur frequently, such as more than once in a week. Moderately preoccupied with ideas of persecution OR reports persecutory delusions expressed with much doubt (e.g., partial delusion).

Delusional. Speaks of Mafia plots, the FBI, or others poisoning food, persecution by supernatural forces.

Same as 6, but the beliefs are bizarre or more preoccupying. Tends to disclose or act on persecutory delusion.
10. **Hallucinations**

Reports of perceptual experiences in the absence of relevant external stimuli. When rating degree to which functioning is disrupted by hallucinations, include preoccupation with the content and experience of the hallucinations, as well as the functioning disrupted by acting out on the hallucinatory content (e.g., engaging in deviant behaviour due to command hallucinations). Include thoughts aloud, or pseudo-hallucinations (e.g., hears voice inside head) if a voice quality is present.

*Have you had any strange experiences? Like hearing or seeing things that others don’t?*
*Do you ever seem to hear your name being called?*
*Have you heard any sounds or people talking to you or about you when there has been nobody around?*
*Have you heard people taking to you, or about you, when there’s nobody around?*

[If hears voices]: *What does the voice/voices say? Did it have a voice quality?*

*Do you ever have visions or see things that others do not see? What about smell odours that others do not smell?*

[If the patient reports hallucinations, ask the following]:

*Have these experiences interfered with your ability to perform your usual activities/work?*
*How do you explain them?*
*How often do they occur?*

**IF RATED 5 OR ABOVE, ASK THE FOLLOWING QUESTIONS:**
1. *Were you using drugs or alcohol at the time you started experiencing/feeling this?*
2. *Did you think this symptom was related to your drug/alcohol use?*
3. *Did you experience this when you weren’t using any drugs/alcohol or using less than 4 days per week?*

<table>
<thead>
<tr>
<th>Rating</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 = Not present</td>
<td>While resting or going to sleep, sees visions, smells odours or hears voices, sounds or whispers in absence of external stimulation, but no impairment in functioning OR hallucinatory quality of the report doubtful.</td>
</tr>
<tr>
<td>2 = Very Mild</td>
<td>While in a clear state of consciousness, hears a voice calling the subject’s name, experiences nonverbal auditory hallucinations (e.g. sounds or whispers), formless visual hallucinations or has sensory experiences in the presence of a modality-relevant stimulus (e.g., visual illusions) infrequently (e.g., 1-2 times per week) and with no functional impairment.</td>
</tr>
<tr>
<td>3 = Mild</td>
<td>Occasional verbal, visual, olfactory, tactile or gustatory hallucinations but no impairment in functioning OR nonverbal auditory hallucinations/visual illusions occurring more than infrequently or with impairment.</td>
</tr>
<tr>
<td>4 = Moderate</td>
<td>Experiences daily hallucinations OR some areas of functioning are disrupted by hallucinations</td>
</tr>
<tr>
<td>5 = Moderately Severe</td>
<td>Experiences verbal or visual hallucinations several times a day OR many areas of functioning are disrupted by hallucinations.</td>
</tr>
<tr>
<td>6 = Severe</td>
<td>Persistent throughout the day or most areas of functioning are disrupted by hallucinations.</td>
</tr>
</tbody>
</table>
11. Unusual Thought Content

Unusual, odd, strange, or bizarre thought content. Rate the degree of unusualness, not the degree of disorganisation of speech. Delusions are patently absurd, clearly false or bizarre ideas that are expressed with full conviction. Note: Full conviction = if he/she has acted as though the delusional belief was true. Ideas of reference/persecution can be differentiated from delusions in that ideas are expressed with much doubt and contain more elements of reality. Include thought insertion, withdrawal, and broadcasting. Include grandiose, somatic, and persecutory delusions even if rated elsewhere. **Note:** If Somatic Concern, Guilt, Suspiciousness, or Grandiosity are rated ‘6’ or ‘7’ due to delusions, then Unusual Thought Content must be rated a ‘4’ or above.

*Have you ever had any strange thoughts that you couldn’t explain?*
*When you are by yourself what do you think about?*
*Have you been receiving special messages?*
*Have you seen any references to yourself on the TV or in the newspapers?*
*Can anyone read your mind? Do you have a special relationship with God?*
*Is anything like electricity, X-rays, or radio waves affecting you?*
*Are thoughts put into your head that are not your own?*
*Have you felt that you were under the control of another person or force?*
*Do you feel as though your thoughts and actions are under your control?*

**IF RATED 5 OR ABOVE, ASK THE FOLLOWING QUESTIONS:**
1. *Were you using drugs or alcohol at the time you started experiencing/feeling this?*
2. *Did you think this symptom was related to your drug/alcohol use?*
3. *Did you experience this when you weren’t using any drugs/alcohol or using less than 4 days per week?*

<table>
<thead>
<tr>
<th>Rating</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 = Not present</td>
<td>Ideas of reference (people stare/laugh at him/her). Ideas of persecution (people mistreat him/her). Unusual beliefs in psychic powers, spirits, UFO’s, or unrealistic beliefs in one’s own abilities. Not strongly held. Some doubt.</td>
</tr>
<tr>
<td>2 = Very Mild</td>
<td>Same as 2, but degree of reality distortion is more severe as indicated by highly unusual ideas or greater conviction. Content may be typical of delusions (even bizarre), but without full conviction. The delusion does not seem to have fully formed, but is considered as one possible explanation for an usual experience.</td>
</tr>
<tr>
<td>3 = Mild</td>
<td>Delusion present but no preoccupation or functional impairment. May be an encapsulated delusion or a firmly endorsed absurd belief about past delusional circumstances.</td>
</tr>
<tr>
<td>4 = Moderate</td>
<td>Full delusion(s) present with some preoccupation or some areas of Severe functioning disrupted by delusional thinking.</td>
</tr>
<tr>
<td>5 = Moderately Severe</td>
<td>Full delusion(s) present with much preoccupation OR many areas of functioning disrupted by delusional thinking.</td>
</tr>
<tr>
<td>6 = Severe</td>
<td>Full delusion(s) present with almost total preoccupation OR most areas Severe of functioning disrupted by delusional thinking.</td>
</tr>
</tbody>
</table>
Rate items 12-13 on the basis of participant’s self-report and observed behaviour.

12. **Bizarre Behaviour**

Reports of behaviours which are odd, unusual, or psychotically criminal. Not limited to interview period. Include inappropriate sexual behaviour and inappropriate affect.

*Have you done anything that has attracted the attention of others?*
*Have you done anything that could have gotten you into trouble with the police?*
*Have you done anything that seemed unusual or disturbing to others?*

**IF RATED 5 OR ABOVE, ASK THE FOLLOWING QUESTIONS:**
1. *Were you using drugs or alcohol at the time you started experiencing/feeling this?*
2. *Did you think this symptom was related to your drug/alcohol use?*
3. *Did you experience this when you weren’t using any drugs/alcohol or using less than 4 days per week?*

<table>
<thead>
<tr>
<th>Rating</th>
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</tr>
</thead>
<tbody>
<tr>
<td>1 = Not present</td>
<td>Slightly odd or eccentric public behaviour, e.g., occasionally giggles to self, fails to make appropriate eye contact, that does not seem to attract the attention of others OR unusual behaviour conducted in private, e.g., innocuous rituals that would not attract the attention of others.</td>
</tr>
<tr>
<td>2 = Very Mild</td>
<td>Noticeably peculiar public behaviour, e.g., inappropriately loud talking, makes inappropriate eye contact, OR private behaviour that occasionally, but not always, attracts the attention of others, e.g., hoards food, conducts unusual rituals, wears gloves indoors.</td>
</tr>
<tr>
<td>3 = Mild</td>
<td>Clearly bizarre behaviour that attracts or would attract (if done privately) the attention or concern of others, but with no corrective intervention necessary. Behaviour occurs occasionally, e.g., fixated staring into space for several minutes, talks back to voices once, inappropriate giggling/laughter on 1-2 occasions, talking loudly to self.</td>
</tr>
<tr>
<td>4 = Moderate</td>
<td>Clearly bizarre behaviour that attracts or would attract (if done privately) the attention of others or the authorities, e.g., fixated staring in socially disruptive way, frequent inappropriate giggling/laughter, occasionally responds to voices, or eats non-foods.</td>
</tr>
<tr>
<td>5 = Moderately Severe</td>
<td>Bizarre behaviour that attracts attention of others and intervention by authorities, e.g., directing traffic, public nudity, starting into space for long periods, carrying on a conversation with hallucinations, frequent inappropriate giggling/laughter.</td>
</tr>
<tr>
<td>6 = Severe</td>
<td>Serious crimes committed in a bizarre way that attracts the attention of others and the control of authorities, e.g., sets fires and stares at flames OR almost constant bizarre behaviour, e.g., inappropriate giggling/laughter responds only to hallucinations and cannot be engaged in interaction.</td>
</tr>
</tbody>
</table>
13. **Self-Neglect**

Hygiene, appearance, or eating behaviour below usual expectations, below socially acceptable standards, or life-threatening.

*How has your grooming been lately?*
*How often do you change your clothes?*
*How often do you take showers?*
*Has anyone (parents/staff) complained about your grooming or dress?*
*Do you eat regular meals?*

<table>
<thead>
<tr>
<th>1 = Not present</th>
<th>2 = Very Mild</th>
<th>3 = Mild</th>
<th>4 = Moderate</th>
<th>5 = Moderately Severe</th>
<th>6 = Severe</th>
<th>7 = Extremely Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hygiene/appearance slightly below usual community standards, e.g., shirt out of pants, buttons unbuttoned, shoe laces untied, but no social or medical consequences.</td>
<td>Hygiene/appearance occasionally below usual community standards, e.g., irregular bathing, clothing is stained, hair uncombed, occasionally skips an important meal. No social or medical consequences.</td>
<td>Hygiene/appearance is noticeably below usual community standards, e.g., fails to bathe or change clothes, clothing very soiled, hair unkempt, needs prompting, noticeable by others OR irregular eating and drinking with minimal medical concerns and consequences.</td>
<td>Several areas of hygiene/appearance are below usual community standards OR poor grooming draws criticism by others, and requires regular prompting. Eating or hydration are irregular and poor, causing some medical problems.</td>
<td>Many areas of hygiene/appearance are below usual community standards, does not always bathe or change clothes even if prompted. Poor grooming has caused social ostracism at school/residence/work, or required intervention. Eating erratic and poor, may require medical intervention.</td>
<td>Many areas of hygiene/appearance/nutrition are extremely poor and easily noticed as below usual community standards OR hygiene/appearance/nutrition requires urgent and immediate medical intervention.</td>
<td></td>
</tr>
</tbody>
</table>
14. **Disorientation**

Does not comprehend situations or communications, such as questions asked during the entire BPRS interview. Confusion regarding person, place or time. Do not rate if incorrect responses are due to delusions.

*May I ask you one or two standard questions we ask everybody?*
*How old are you?*
*What is the date? [Allow ± 2 days]*
*What is this place called?*
*What year were you born?*
*Who is the Prime Minister?*

**IF RATED 5 OR ABOVE, ASK THE FOLLOWING QUESTIONS:**
1. *Were you using drugs or alcohol at the time you started experiencing/feeling this?*
2. *Did you think this symptom was related to your drug/alcohol use?*
3. *Did you experience this when you weren’t using any drugs/alcohol or using less than 4 days per week?*

<table>
<thead>
<tr>
<th>Rating</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 = Not present</td>
<td>Seems muddled or mildly confused 1-2 times during interview. Oriented to person, time and place.</td>
</tr>
<tr>
<td>2 = Very Mild</td>
<td>Occasionally muddled or mildly confused 3-4 times during interview. Minor inaccuracies in person, place, or time, e.g., date off by more than ± or - 2 days, or gives wrong location.</td>
</tr>
<tr>
<td>3 = Mild</td>
<td>Frequently confused during interview. Minor inaccuracies in person, place or time are noted, as in ‘3’ above. In addition, may have difficulty remembering general information, e.g., name of Prime Minister.</td>
</tr>
<tr>
<td>4 = Moderate</td>
<td>Markedly confused during interview, or to person, place, or time. Significant inaccuracies are noted, e.g., data off by more than one week, or cannot give correct name of location. Has difficulty remembering personal information, e.g., where he/she was born, or recognising familiar people.</td>
</tr>
<tr>
<td>5 = Moderately Severe</td>
<td>Disoriented to person, place, or time, e.g., cannot give correct month and year. Disoriented in 2 out of 3 spheres.</td>
</tr>
<tr>
<td>6 = Severe</td>
<td>Grossly disoriented to person, place, or time, e.g., cannot give name or age. Disoriented in all 3 spheres.</td>
</tr>
</tbody>
</table>
Rate items 15-24 on the basis of observed behaviour and speech.

15. Conceptual Disorganisation

Degree to which speech is confused, disconnected, or disorganised. Rate tangentiality, circumstantiality, sudden topic shifts, incoherence, derailment, blocking, neologisms, and other speech disorders. Do not rate content of speech.

*Have people seemed to misunderstand you or not get the point of what you’re saying?*

*Have you been feeling confused?*

<table>
<thead>
<tr>
<th>Rating</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 = Not present</td>
<td></td>
</tr>
<tr>
<td>2 = Very Mild</td>
<td>Peculiar use of words or rambling but speech is comprehensible.</td>
</tr>
<tr>
<td>3 = Mild</td>
<td>Speech a bit hard to understand or make sense of due to tangentiality, circumstantiality, or sudden topic shifts.</td>
</tr>
<tr>
<td>4 = Moderate</td>
<td>Speech difficult to understand due to tangentiality, circumstantiality, idiosyncratic speech, or topic shifts on many occasions OR 1-2 instances of incoherent phrases.</td>
</tr>
<tr>
<td>5 = Moderately Severe</td>
<td>Speech difficult to understand due to circumstantiality, tangentiality, neologisms, blocking, or topic shifts most of the time OR 3-5 instances of incoherent phrases.</td>
</tr>
<tr>
<td>6 = Severe</td>
<td>Speech is incomprehensible due to severe impairments most of the time. Many BPRS items cannot be rated by self-report alone.</td>
</tr>
<tr>
<td>7 = Extremely Severe</td>
<td>Speech is incomprehensible throughout interview.</td>
</tr>
</tbody>
</table>
16. **Blunted Affect**

Restricted range in emotional expressiveness of face, voice and gestures. Marked indifference or flatness even when discussing distressing topics. In the case of euphoric or dysphoric participants, rate Blunted Affect is a flat quality is also present. Use the following probes at the end of interview to assess emotional responsivity:

*Have you heard any good jokes lately? Would you like to hear a joke?*

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1 = Not present</td>
<td></td>
</tr>
<tr>
<td>2 = Very Mild</td>
<td>Emotional range is slightly subdued or reserved but displays appropriate facial expressions and tone of voice that are within the normal limits.</td>
</tr>
<tr>
<td>3 = Mild</td>
<td>Emotional range is diminished, subdued, or reserved, without many spontaneous and appropriate emotional responses. Voice tone is slightly monotonous.</td>
</tr>
<tr>
<td>4 = Moderate</td>
<td>Emotional range is noticeably diminished, participant doesn’t show emotion, smile or react to distressing topics except infrequently. Voice tone is monotonous or there is noticeable decrease in spontaneous movements. Displays of emotion or gestures are usually followed by a return to flattened affect.</td>
</tr>
<tr>
<td>5 = Moderately Severe</td>
<td>Emotional range very diminished, participant doesn’t show emotions, smiles or react to distressing topics except minimally, few gestures, facial expression does not change very often. Voice tone is monotonous much of the time.</td>
</tr>
<tr>
<td>6 = Severe</td>
<td>Very little emotional range or expression. Mechanical in speech and gestures most of the time. Unchanging facial expression. Voice tone is monotonous most of the time.</td>
</tr>
<tr>
<td>7 = Extremely Severe</td>
<td>Virtually no emotional range or expressiveness, stiff movements. Voice tone is monotonous all of the time.</td>
</tr>
</tbody>
</table>
17. **Emotional Withdrawal**

Deficiency in participant’s ability to relate emotionally during the interview situation. Use your own feelings as to the presence of an “invisible barrier” between participant and interviewer. Include withdrawal apparently due to psychotic processes.

<table>
<thead>
<tr>
<th>Rating</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Not present</td>
</tr>
<tr>
<td>2</td>
<td>Very Mild Lack of emotional involvement shown by occasional failure to make reciprocal comments, occasionally appearing preoccupied, or smiling in a stilted manner, but spontaneously engages the interviewer most of the time.</td>
</tr>
<tr>
<td>3</td>
<td>Mild Lack of emotional involvement shown by noticeable failure to make reciprocal comments, appearing preoccupied, or lacking in warmth, but responds to interviewer when approached.</td>
</tr>
<tr>
<td>4</td>
<td>Moderate Emotional contact not present much of the interview because participant does not elaborate responses, fails to make eye contact, doesn’t seem to care if interviewer is listening or may be preoccupied with psychotic material.</td>
</tr>
<tr>
<td>5</td>
<td>Moderately Severe Same as ‘4’ but emotional contact not present most of the interview.</td>
</tr>
<tr>
<td>6</td>
<td>Severe Actively avoids emotional participation. Frequently unresponsive or responds with yes/no answers (not solely due to persecutory delusions). Responds with only minimal affect.</td>
</tr>
<tr>
<td>7</td>
<td>Extremely Severe Consistently avoids emotional participation. Unresponsive or responds with yes/no answers (not solely due to persecutory delusions). May leave during interview or just not respond at all.</td>
</tr>
</tbody>
</table>

18. **Motor Retardation**

Reduction of energy level evidenced by slowed movements and speech, reduced body tone, decreased number of spontaneous body movements. Rate on the basis of observed behaviour of the participant only. Do not rate on the basis of participant’s subjective impression of his/her own energy level. Rate regardless of medication effects.

<table>
<thead>
<tr>
<th>Rating</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Not present</td>
</tr>
<tr>
<td>2</td>
<td>Very Mild Slightly slowed or reduced movements or speech compared to most people.</td>
</tr>
<tr>
<td>3</td>
<td>Mild Noticeably slowed or reduced movements or speech compared to most people.</td>
</tr>
<tr>
<td>4</td>
<td>Moderate Large reduction or slowness in movements or speech.</td>
</tr>
<tr>
<td>5</td>
<td>Moderately Severe Seldom moves or speaks spontaneously OR very mechanical or stiff movements.</td>
</tr>
<tr>
<td>6</td>
<td>Severe Does not move or speak unless prodded or urged.</td>
</tr>
<tr>
<td>7</td>
<td>Extremely Severe Frozen, catatonic.</td>
</tr>
</tbody>
</table>
19. **Tension**

Observable physical and motor manifestations of tension, ‘nervousness’ and agitation. Self-reported experiences of tension should be rated under the item on anxiety. Do not rate is restlessness is solely akathisia, but do rate if akathisia is exacerbated by tension.

<table>
<thead>
<tr>
<th>Rating</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 = Not present</td>
<td>More fidgety than most but within normal range. A few transient signs of tension, e.g., picking at fingernails, foot wagging, scratching scalp several times, or finger tapping.</td>
</tr>
<tr>
<td>2 = Very Mild</td>
<td>Same as ‘2’ but with more frequent or exaggerated signs of tension.</td>
</tr>
<tr>
<td>3 = Mild</td>
<td>Many and frequent signs of motor tension with one or more signs sometimes occurring simultaneously, e.g., wagging one’s foot while wringing hands together. There are times when no signs of tension are present.</td>
</tr>
<tr>
<td>4 = Moderate</td>
<td>Many and frequent signs of motor tension with one or more signs often occurring simultaneously. There are still rare times when no signs of tension are present.</td>
</tr>
<tr>
<td>5 = Moderately Severe</td>
<td>Same as ‘5’ but signs of tension are continuous.</td>
</tr>
<tr>
<td>6 = Severe</td>
<td>Multiple motor manifestations of tension are continuously present, e.g., continuous pacing and hang wringing.</td>
</tr>
</tbody>
</table>

20. **Uncooperativeness**

Resistance and lack of willingness to cooperate with the interview. The uncooperativeness might result from suspiciousness. Rate only uncooperativeness in relation to the interview, not behaviours involving peers and relatives.

<table>
<thead>
<tr>
<th>Rating</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 = Not present</td>
<td>Shows nonverbal signs of reluctance, but does not complain or argue.</td>
</tr>
<tr>
<td>2 = Very Mild</td>
<td>Gripes or tries to avoid complying, but goes ahead without argument.</td>
</tr>
<tr>
<td>3 = Mild</td>
<td>Verbally resists but eventually complies after questions are rephrased or repeated.</td>
</tr>
<tr>
<td>4 = Moderate</td>
<td>Same as 4, but some information necessary for accurate ratings is withheld.</td>
</tr>
<tr>
<td>5 = Moderately Severe</td>
<td>Refuses to cooperate with interview, but remains in interview situation.</td>
</tr>
<tr>
<td>6 = Severe</td>
<td>Same as 6, with active efforts to escape the interview.</td>
</tr>
</tbody>
</table>
21. Excitement

Heightened emotional tone, or increased reactivity to interviewer or topics being discussed, as evidenced by increased intensity of facial expressions, voice tone, expressive gestures or increase in speech quantity and speed.

<table>
<thead>
<tr>
<th>Rating</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 = Not present</td>
<td>Subtle and fleeting or questionable increase in emotional intensity. For example, at times seems keyed-up or overly alert.</td>
</tr>
<tr>
<td>2 = Very Mild</td>
<td>Subtle but persistent increase in emotional intensity. For example, lively use of gestures and variation in voice tone.</td>
</tr>
<tr>
<td>3 = Mild</td>
<td>Definite but occasional increase in emotional intensity. For example, reacts to interviewer or topics that are discussed with noticeable emotional intensity. Some pressured speech.</td>
</tr>
<tr>
<td>4 = Moderate</td>
<td>Definite and persistent increase in emotional intensity. For example, reacts to many stimuli, whether relevant or not, with considerable emotional intensity. Frequent pressured speech.</td>
</tr>
<tr>
<td>5 = Moderately Severe</td>
<td>Marked increase in emotional intensity. For example reacts to most stimuli with inappropriate emotional intensity. Has difficulty settling down or staying on task. Often restless, impulsive, or speech is often pressured.</td>
</tr>
<tr>
<td>6 = Severe</td>
<td>Extremely restless and impulsive most of the time. Constant pressured speech.</td>
</tr>
</tbody>
</table>

22. Distractibility

Degree to which observed sequences of speech and actions are interrupted by stimuli unrelated to the interview. Distractibility is rated when the participant shows a change in the focus of attention or a marked shift in gaze. Participant’s attention may be drawn to noise in adjoining room, books on shelf, interviewer’s clothing, etc. Do not rate circumstantiality, tangentiality, or flight of ideas. Also do not rate rumination with delusional material. Rate even if the distracting stimulus cannot be identified.

<table>
<thead>
<tr>
<th>Rating</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 = Not present</td>
<td>Generally can focus on interviewer’s questions with only 1 distraction or inappropriate shift of attention of brief duration.</td>
</tr>
<tr>
<td>2 = Very Mild</td>
<td>Participant shifts focus of attention to matters unrelated to the interview 2-3 times.</td>
</tr>
<tr>
<td>3 = Mild</td>
<td>Often responsive to irrelevant stimuli in the room, e.g., averts gaze from the interviewer.</td>
</tr>
<tr>
<td>4 = Moderate</td>
<td>Same as above, but now distractibility clearly interferes with the flow of the interview.</td>
</tr>
<tr>
<td>5 = Moderately Severe</td>
<td>Extremely difficult to conduct interview or pursue a topic due to preoccupation with irrelevant stimuli.</td>
</tr>
<tr>
<td>6 = Severe</td>
<td>Impossible to conduct interview due to preoccupation with irrelevant stimuli.</td>
</tr>
</tbody>
</table>
23. **Motor Hyperactivity**

Increase in energy level evidenced by more frequent movement and/or rapid speech. Do not rate if restlessness is due to akathisia.

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Not present</td>
</tr>
<tr>
<td>2</td>
<td>Very Mild</td>
</tr>
<tr>
<td>3</td>
<td>Mild</td>
</tr>
<tr>
<td>4</td>
<td>Moderate</td>
</tr>
<tr>
<td>5</td>
<td>Moderately Severe</td>
</tr>
<tr>
<td>6</td>
<td>Severe</td>
</tr>
<tr>
<td>7</td>
<td>Extremely Severe</td>
</tr>
</tbody>
</table>

24. **Mannerisms and Posturing**

Unusual and bizarre behaviour, stylised movements or acts, or any postures which are clearly uncomfortable or inappropriate. Exclude obvious manifestations of medication side effects. Do not include nervous mannerisms that are not odd or unusual.

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Not present</td>
</tr>
<tr>
<td>2</td>
<td>Very Mild</td>
</tr>
<tr>
<td>3</td>
<td>Mild</td>
</tr>
<tr>
<td>4</td>
<td>Moderate</td>
</tr>
<tr>
<td>5</td>
<td>Moderately Severe</td>
</tr>
<tr>
<td>6</td>
<td>Severe</td>
</tr>
<tr>
<td>7</td>
<td>Extremely Severe</td>
</tr>
</tbody>
</table>
Appendix E

Oxford Liverpool Inventory of Feelings and Experiences – Impulsive Non-Conformity Scale

Instructions: All items are answered YES or NO. There are no right or wrong answers. Remember to answer ALL the questions and to give only ONE response to each question.

<table>
<thead>
<tr>
<th>QUESTION</th>
<th>YES</th>
<th>NO</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Do you often overindulge in alcohol or food?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. When with groups of people, do you usually prefer to let someone else be the centre of attention?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. When you catch a train, do you often arrive at the last minute?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Do you often change between intense liking and disliking of the same person?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Have you ever cheated at a game?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Do you at times have an urge to do something harmful or shocking?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Are you usually in an average sort of mood, not too high and not too low?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Would you take drugs which may have strange or dangerous effects?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. Do you stop to think things over before doing anything?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. Have you ever blamed someone for doing something you know was really your fault?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>11. Would being in debt worry you?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>12. Do you think people spend too much time safeguarding their future with savings and insurance?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>13. Do you ever have the urge to break or smash things?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>14. Have you ever felt the urge to injure yourself?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>15. Would it make you nervous to play the clown in front of other people?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>16. Do you consider yourself to be pretty much an average kind of person?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>17. Have you ever taken advantage of someone?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>18. Would you like other people to be afraid of you?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>19. Do you often have the urge to hit someone?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>20. Do people who drive carefully annoy you?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>21. Do you sometimes talk about things you know nothing about?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>22. Do you often feel like doing the opposite of what other people suggest, even though you know they are right?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>23. Do you often feel like the impulse to spend money, which you know you can’t afford?</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Appendix F
Participant Information and Consent Form – Study 1

(Displayed on Griffith University Letterhead)

**Project Title: An Investigation of Thinking Styles and Beliefs in Methamphetamine Users**

**INFORMATION SHEET**

**Researchers:**
Kely Lapworth, Sharon Dawe, Penelope Davis, Department of Psychology, Sharon Dawe, Department of Psychology, Griffith University.

**The current project:**
This project investigates the relationship between methamphetamine use and thinking. Methamphetamine use often produces changes in the way people think. There is considerable variability but for some people, using methamphetamine makes them feel more suspicious or paranoid. However, this does not occur for everyone.

In this study we would like to ask you about your current use of methamphetamines, other drugs and thinking style.

**What you will be asked to do:**
To participate in this study you will be asked to complete a set of measures. These measures ask about your use of methamphetamine, other drug use and thinking styles and beliefs about events in the last month. This should take approximately 60 minutes.

**Who can take part in the study:**
We are keen to talk to people who use substances, particularly methamphetamine. If you would like to participate in this study, the researcher will ask you a series of questions to ensure you are eligible to participate.

**The expected benefits of the research:**
This study will help us understand the effects that methamphetamine has on a range of different people as well the many different effects of the drug itself.

**Risks to you:**
There are no risks for you by participating in this study. Your confidentiality/privacy: All information you provide will be kept confidential and not shared with other parties. You will be given a number so that your name will not be connected to your questionnaires. Any data reported from this study would not identify individuals. However, if we are of the view that you are ever at risk of harming yourself or another, we are required to take action to protect you or others. Whenever necessary, we would discuss this with the staff of the Needle Syringe Exchange Service.

All information collected as part of the study will be locked in a storage cabinet at Griffith University and will be kept for 5 years following publication (after which time it will be safely destroyed). Any published material will make reference to group data and will not identify individual participants.

**Your participation is voluntary:**
Your participation in this program is completely voluntary and you may withdraw from the study at any time. Any refusal to continue participating will not effect in any way your entitlements at Needle Syringe Exchange Service.

Questions / further information: If you have any questions during or after the interview, we are happy to provide you with answers and feedback. Please feel free to contact Kely Lapworth on 3875 3371.
The ethical conduct of this research:
This project has been viewed and approved by the Griffith University Human Ethics Committee in accordance with the National Statement on Ethical Conduct in Research Involving Humans. If you have any concerns or complaints about the ethical conduct of the research project you can contact the Manager, Research Ethics on 3875 5585 or research-ethics@griffith.edu.au. This research has also been approved by the relevant Ethics Committee of Queensland Health.

Feedback to you: We will provide a brief outline of the findings from the research at the end of the project. These will be given to the staff at the NSP. Further information can be obtained by contacting Kely Lapworth on 3875 3371.
Project Title: An Investigation of Thinking Styles and Beliefs in Methamphetamine Users

STANDARD CONSENT FORM FOR PARTICIPANTS

Name of Researchers:
Ms Kely Lapworth, Professor Sharon Dawe, and Dr Penelope Davis – School of Psychology, Griffith University.

I agree to participate in the above named project and in doing so acknowledge that:

1. I have read the attached Information Sheet outlining the nature and purpose of the project and the extent of my involvement, and have had these details explained to me. I have had the opportunity to ask further questions and I am satisfied that I understand.

2. I understand that my participation will take approximately 40 minutes in total. During this time, I will be required to complete several questionnaires.

3. I have been informed as to the nature and extent of any risk to my health or well-being.

4. I am aware that although the outcome of the project is aimed at expanding psychological knowledge generally, it may not result in any direct benefit to me.

5. I have been informed that I may withdraw from the project at my request at any time and that this decision will not affect me or penalise me in any way. It will have no bearing on my ability to continue accessing services such as the Needle Exchange Service.

6. I have been advised the District Manager, on recommendation from The Prince Charles Hospital Human Research Ethics Committee and the Ethics Committee at Griffith University, have given approval for this project.

7. I am aware that I may request further information about this project upon completion.

I have read the information sheet and the information contained in the consent form above. I agree to participate in the project titled - An Investigation of Thinking Styles and Beliefs in Methamphetamine Users and give my consent freely.

I understand that I can contact the Manager, Research Ethics, at Griffith University Human Research Ethics Committee on 3735 5585 (or G.Allen@griffith.edu.au) if I have any concerns about the ethical conduct of the project. I have had all questions answered to my satisfaction.

Signatures:

Participant’s Signature ______________________ Date ______________________

Investigator’s Signature ______________________ Date ______________________
## Appendix G
Demographic and Data Collection Sheet – Study 2

16. Age ................... years

17. Sex: Male ...... 0  Female ...... 1

18. Present Occupation: _________________________

<table>
<thead>
<tr>
<th>Occupation</th>
<th>Code</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not employed</td>
<td>0</td>
</tr>
<tr>
<td>Full time</td>
<td>1</td>
</tr>
<tr>
<td>Part time/casual</td>
<td>2</td>
</tr>
<tr>
<td>Student</td>
<td>3</td>
</tr>
<tr>
<td>Home duties</td>
<td>4</td>
</tr>
</tbody>
</table>

19. Marital Status:

<table>
<thead>
<tr>
<th>Status</th>
<th>Code</th>
</tr>
</thead>
<tbody>
<tr>
<td>Single</td>
<td>0</td>
</tr>
<tr>
<td>Defacto</td>
<td>1</td>
</tr>
<tr>
<td>Married</td>
<td>2</td>
</tr>
<tr>
<td>Separated</td>
<td>3</td>
</tr>
<tr>
<td>Divorced</td>
<td>4</td>
</tr>
<tr>
<td>Widowed</td>
<td>5</td>
</tr>
</tbody>
</table>

20. What are your current living arrangements?

<table>
<thead>
<tr>
<th>Arrangement</th>
<th>Code</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alone</td>
<td>0</td>
</tr>
<tr>
<td>With partner</td>
<td>1</td>
</tr>
<tr>
<td>With family</td>
<td>2</td>
</tr>
<tr>
<td>Share accommodation</td>
<td>3</td>
</tr>
<tr>
<td>Other</td>
<td>4</td>
</tr>
</tbody>
</table>

Specify: ____________________

21. What is the highest level of formal education you have obtained?

<table>
<thead>
<tr>
<th>Education</th>
<th>Code</th>
</tr>
</thead>
</table>
| High school        | 0    | (Specify grade___________)
| Trade qualification| 1    |
| TAFE               | 2    |
| Undergraduate      | 3    |
| Postgraduate       | 4    |

22. What is the main language you speak at home (or first language if participant lives alone)?

<table>
<thead>
<tr>
<th>Language</th>
<th>Code</th>
</tr>
</thead>
</table>
| English  | 1    | (Specify_________________)
| Other    | 2    | (Specify_________________)

23. What is your ethnicity?

____________________________________________________________________________

24. Have you ever been diagnosed with a psychiatric problem?

No .................................. 0
Yes .................................. 1

Specify: 

____________________________________________________________________________
____________________________________________________________________________
____________________________________________________________________________
____________________________________________________________________________

10. Are you currently taking medication for a psychiatric problem or have you taken this type of medication in the past?

No 0.................................
Yes 1.................................

Specify name and circle all that apply below: Specify what participant was medicated for:

<table>
<thead>
<tr>
<th>Specifying Medication</th>
<th>Participant's Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td></td>
</tr>
<tr>
<td>Antipsychotic</td>
<td>1 (e.g. Zyprexa, Olanzapine, Chlorpromazine, Clozapine, Risperidone, Aripiprazole)</td>
</tr>
<tr>
<td>Benzodiazepines</td>
<td>2 (e.g. Valium, Rohys, Serepax, Temazepam)</td>
</tr>
<tr>
<td>Antidepressant</td>
<td>3 (e.g. Tryptanol, Prozac, Prothiaden, Sinequan, Zoloft)</td>
</tr>
<tr>
<td>Other</td>
<td>4 Specify:________________________</td>
</tr>
</tbody>
</table>

None 0
Antipsychotic .................................. 1 (e.g. Zyprexa, Olanzapine, Chlorpromazine, Clozapine, Risperidone, Aripiprazole)
Benzodiazepines ....................... 2 (e.g. Valium, Rohys, Serepax, Temazepam)
Antidepressant ......................... 3 (e.g. Tryptanol, Prozac, Prothiaden, Sinequan, Zoloft)
Other .................................. 4 Specify:________________________
Appendix H
Psychosis Screener

(i) Delusional mood
(a) Has the person ever felt something strange, unexplainable was going on?
   0 = No
   1 = Yes ■
(b) If yes, was this so strange that others would find it very hard to believe?
   0 = No
   1 = Yes ■

(ii) Grandiose delusions
(a) Has the person ever believed they have special powers, talents that most people lack?
   0 = No
   1 = Yes ■
(b) If yes, do they belong to a group that believes they have special powers, talents?
   0 = No
   1 = Yes ■

(iii) Delusions of reference/persecution
(a) Has the person ever felt people were too interested in them?
   0 = No
   1 = Yes ■
(b) If yes, did they feel harm might come to them?
   0 = No
   1 = Yes ■

(iv) Delusions of control
(a) Has the person ever felt thoughts were directly interfered with, controlled by others?
   0 = No
   1 = Yes ■
(b) If yes, did this happen in a way others would find hard to believe, e.g. telepathy?
   0 = No
   1 = Yes ■
(v) Hallucinations
(a) Has the person ever heard voices or had visions when there was no-one around?
0 = No
1 = Yes ■

(vi) Diagnosis of Psychosis
(a) Has the person ever been prescribed psychotic medicine, diagnosed as psychotic by a doctor?
0 = No
1 = Yes ■
Please specify:
______________________________________________________________

(vii) Rating of Psychosis by Key Worker
(a) Using clinical judgment, is this person psychotic or has ever been psychotic?
0 = Definitely not
1 = Possibly
2 = Definitely

Additional comments:
____________________________________________________________________
____________________________________________________________________

NOTE: The cut-off point applied for recording a person as screen positive for psychosis is at least 2 positive items (Items 1–6) subject to the following provisos:

• ‘yes’ to item 6 only and ‘definitely positive’ to item 7 = positive for psychosis;
• ‘yes’ to item 6 and ‘yes’ to one other item 1– item 5 and ‘maybe’ in item 7 = positive for psychosis;
• ‘yes’ to item 6 only and ‘possibly’ in item 7 = negative for psychosis.
If the clinician considers the person
### Appendix I
Barrett Impulsiveness Scale – Version 11

**DIRECTIONS:** People differ in the ways they act and think in different situations. This is a test to measure some of the ways in which you act and think. Read each statement and put an X on the appropriate number on the right side of this page (0, 1, 2, or 3). Do not spend too much time on any statement. Answer quickly and honestly.

<table>
<thead>
<tr>
<th>Statement</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>1  I plan tasks carefully.</td>
<td>Rarely/ Never</td>
<td>Occasionally</td>
<td>Often</td>
<td>Almost Always/Always</td>
</tr>
<tr>
<td>2  I do things without thinking.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>3  I make-up my mind quickly.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>4  I am happy-go-lucky.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>5  I don’t “pay attention.”</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>6  I have “racing” thoughts.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>7  I plan trips well ahead of time.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>8  I am self-controlled.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>9  I concentrate easily.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>10 I save regularly.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>11 I “squirm” at plays or lectures.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>12 I am a careful thinker.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>13 I plan for job security.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>14 I say things without thinking.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>15 I like to think about complex problems.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>16 I change jobs.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>17 I act “on impulse.”</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>18 I get easily bored when solving thought problems.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>19 I act on the spur of the moment.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>20 I am a steady thinker.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>21 I change residences.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>22 I buy things on impulse.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>23 I can only think about one thing at a time.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>24 I change hobbies.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>25 I spend or charge more than I earn.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>26 I often have extraneous thoughts when thinking.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>27 I am more interested in the present than the future.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>28 I am restless at the theatre or lectures.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>29 I like puzzles.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>30 I am future oriented.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>
Appendix J
Aggression Questionnaire

Please rate each of the following 29 items in terms of how characteristic or alike they are of you.

1. Once in a while I can’t control the urge to strike another person.
   
<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extremely Uncharacteristic of me</td>
<td>Extremely Characteristic of me</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

2. Given enough provocation, I may hit another person.
   
<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extremely Uncharacteristic of me</td>
<td>Extremely Characteristic of me</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

3. If somebody hits me, I hit back.
   
<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extremely Uncharacteristic of me</td>
<td>Extremely Characteristic of me</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

4. I get into fights a little more than the average person.
   
<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extremely Uncharacteristic of me</td>
<td>Extremely Characteristic of me</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

5. If I have to resort to violence to protect my rights, I will.
   
<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extremely Uncharacteristic of me</td>
<td>Extremely Characteristic of me</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

6. There are people who pushed me so far that we came to blows.
   
<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extremely Uncharacteristic of me</td>
<td>Extremely Characteristic of me</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
7. I can think of no good reason for ever hitting a person.
   
   | Extremely Uncharacteristic of me | Extremely Characteristic of me |
   | 1 2 3 4 5 |

8. I have threatened people I know.
   
   | Extremely Uncharacteristic of me | Extremely Characteristic of me |
   | 1 2 3 4 5 |

9. I have become so mad that I have broken things.
   
   | Extremely Uncharacteristic of me | Extremely Characteristic of me |
   | 1 2 3 4 5 |

10. I tell my friends openly when I disagree with them.
    
    | Extremely Uncharacteristic of me | Extremely Characteristic of me |
    | 1 2 3 4 5 |

11. I often find myself disagreeing with people.
    
    | Extremely Uncharacteristic of me | Extremely Characteristic of me |
    | 1 2 3 4 5 |

12. When people annoy me, I may tell them what I think of them.
    
    | Extremely Uncharacteristic of me | Extremely Characteristic of me |
    | 1 2 3 4 5 |

13. I can't help getting into arguments when people disagree with me.
    
    | Extremely Uncharacteristic | Extremely Characteristic |
    | 1 2 3 4 5 |
14. My friends say that I am somewhat argumentative.

<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extremely Uncharacteristic of me</td>
<td>Extremely Characteristic of me</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

15. I flare up quickly, but get over it quickly.

<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extremely Uncharacteristic of me</td>
<td>Extremely Characteristic of me</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

16. When frustrated, I let my irritation show.

<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extremely Uncharacteristic of me</td>
<td>Extremely Characteristic of me</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

17. I sometimes feel like a powder keg ready to explode.

<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extremely Uncharacteristic of me</td>
<td>Extremely Characteristic of me</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

18. I am an even-tempered person.

<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extremely Uncharacteristic of me</td>
<td>Extremely Characteristic of me</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

19. Some of my friends think I'm a hothead.

<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extremely Uncharacteristic of me</td>
<td>Extremely Characteristic of me</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

20. Sometimes I fly off the handle for no good reason.

<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extremely Uncharacteristic</td>
<td>Extremely Characteristic</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
21. **I have trouble controlling my temper.**

<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extremely</td>
<td>Uncharacteristic</td>
<td>of me</td>
<td>Extremely</td>
<td>Characteristic of me</td>
</tr>
</tbody>
</table>

22. **I am sometimes eaten up with jealousy.**

<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extremely</td>
<td>Uncharacteristic</td>
<td>of me</td>
<td>Extremely</td>
<td>Characteristic of me</td>
</tr>
</tbody>
</table>

23. **At times I feel I have gotten a raw deal out of life.**

<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extremely</td>
<td>Uncharacteristic</td>
<td>of me</td>
<td>Extremely</td>
<td>Characteristic of me</td>
</tr>
</tbody>
</table>

24. **Other people always seem to get the breaks.**

<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extremely</td>
<td>Uncharacteristic</td>
<td>of me</td>
<td>Extremely</td>
<td>Characteristic of me</td>
</tr>
</tbody>
</table>

25. **I wonder why sometimes I feel so bitter about things.**

<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extremely</td>
<td>Uncharacteristic</td>
<td>of me</td>
<td>Extremely</td>
<td>Characteristic of me</td>
</tr>
</tbody>
</table>

26. **I know that 'friends' talk about me behind my back.**

<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extremely</td>
<td>Uncharacteristic</td>
<td>of me</td>
<td>Extremely</td>
<td>Characteristic of me</td>
</tr>
</tbody>
</table>

27. **I am suspicious of overly friendly strangers.**

<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extremely</td>
<td>Uncharacteristic</td>
<td>of me</td>
<td>Extremely</td>
<td>Characteristic</td>
</tr>
</tbody>
</table>
28. I sometimes feel that people are laughing at me behind my back.

<table>
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29. When people are especially nice, I wonder what they want.

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<td>Extremely Uncharacteristic of me</td>
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Appendix K
STOP-IT Task – Instructions for Participants

How to use the STOP-IT task

On every trial you will see a circle or a square in the middle of your computer screen. Your task is to respond as fast and accurately as possible to these GO stimuli. You do this by:

- Pressing the ‘Z’ key with your LEFT index finger when you see a SQUARE.
- Pressing the ‘?’ key with your RIGHT index finger when you see a CIRCLE.

Occasionally, the stimulus (i.e., the square or circle) is followed by a sound – a beep-indicating that you have to STOP your response on that trial (Don’t press any keys).

On approximately half of the trials, the sound will be presented soon after the presentation of the GO stimulus and you will notice that it is easy to STOP your response.

On the other half of the trials, the sound will be presented rather late and it will become difficult or even impossible to STOP your response.

Nevertheless, it is important that you DO NOT WAIT for the STOP signal to occur, because if you start waiting, then the computer will wait with presenting the STOP signals.

If you understand these instructions, please press either the ‘Z’ or ‘?’ key to continue.
Information Sheet

Personality style, learning and self control: the effects of methamphetamine use

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Purpose of the study:
Different learning tasks and the ability to exercise self control depend in part on personality styles. In this research we are interested in how people manage different kinds of tasks and whether substance use affects performance. In order to investigate this, we need to interview and measure the performance of both people who use substances, and those who do not.

First I will ask you to complete some questionnaires. Then you will be asked to participate in two computer based tasks (approximately 15-20 mins each). These tasks are similar to computerised games that are found on the internet where you are competing against the computer, or a competitor. We are interested in measuring the time you take to respond to the stimuli in the tasks.

Method and Duration of Testing:
The total duration of the task you will undertake is approximately 1 hour and fifteen minutes.

Foreseeable Risks:
There are no foreseeable risks to you on participation in this study. All questionnaires and computer tasks have been safely administered to participants in previous studies looking at personality and substance use versus no substance use.

Benefits:
Through your participation in this study you will be assisting in advancing the understanding of a model of personality, substance use, learning and self control. This knowledge could then be applied to better understand the role of personality factors in drug misuse and assist with developing effective interventions.

You will be reimbursed $20 to assist with costs associated with undertaking this research, such as travel expenses, etc.
Confidentiality:
Your participation in the study will be completely anonymous. If you choose to participate in the study, no identifying information will be collected.

Participation in the Study:
Your participation in the study is on a purely voluntary basis. You may withdraw from the study at any point without penalty. You may contact the Chief Investigator about any matter of concern regarding the research on the contact numbers provided.

Feedback:
On completion of your participation, you may receive as much feedback as is available regarding the purpose of and expected outcome of the study. When the project as a whole is completed, if requested, you will be sent an information package regarding the actual outcome of the study.

Complaints Mechanism:

The University requires that all participants be informed that if they have any complaints concerning the manner in which a research project is conducted it may be given to the researcher, or, if an independent person is preferred, the Manager, Research Ethics, at Griffith University Human Research Ethics Committee on 3735 5585 (or G.Allen@griffith.edu.au).

Thank you for your assistance with this research project.
Appendix M
Participant Consent Form – Study 2

(Displayed on Griffith University Letterhead)

**Personality style, learning and self control: The effects of methamphetamine use versus no use**

I agree to participate in the project - *Personality style, learning and self control: the effects of methamphetamine use* - being conducted by Ms Kely Lapworth, Professor Sharon Dawe, and Dr Penny Davis and give my consent freely.

I understand that this study is being conducted to clarify the nature of the relationship between personality, performance on computer tasks, substance use versus no substance use and learning.

I understand that my participation will take approximately 1 hour and fifteen minutes in total. During this time, I will be required to complete some questionnaires and complete two computer tasks involving trial-and-error learning.

I understand that the study will be carried out as described in the information sheet, a copy of which I have retained.

I understand that my data will remain confidential and no identifying information will be collected or connected to this data.

I understand that I may receive feedback regarding the purpose and expected outcomes of the study when I have finished my participation, if I request it.

I understand that I am not required to participate in this research project if I do not wish to do so and that I can withdraw from the study at any time without needing to explain my reasons for withdrawing. No loss of benefit will occur as a result of my withdrawal, nor penalty be incurred.

I have read the information sheet and the consent form. I agree to participate in the *Personality style, learning and self control: the effects of methamphetamine use* project and give my consent freely.

I understand that I can contact the Manager, Research Ethics, at Griffith University Human Research Ethics Committee on 3735 5585 (or G.Allen@griffith.edu.au) if I have any concerns about the ethical conduct of the project. I have had all questions answered to my satisfaction.

Signatures:

………………………………………………. …………………
Participant Date

………………………………………………. …………………
Investigator Date
Appendix N
Histograms Displaying Transformed Variables