The influence of depression on treatment for methamphetamine use

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Methamphetamine use is common internationally, and Australia has one of the highest rates of use in the world.[1] Users risk a range of negative effects, including dependence,[2] issues with personal relationships and employment, and mental health problems.[3]

Up to a quarter of methamphetamine users report psychiatric symptoms of sufficient severity to warrant hospitalisation.[4] In particular, several Australian studies have found high rates of mood disorder among methamphetamine users, with up to 75% reporting symptoms of anxiety or depression.[5] Depressive symptoms are even higher among methamphetamine users than among cocaine users, and persist longer.[6] It is often comorbid conditions such as depression that prompt users of methamphetamines to seek treatment,[7] with general practitioners often being the first port of call.[8]

The existence of depression prior to treatment has been associated with poorer treatment adherence and poorer methamphetamine use outcomes among methamphetamine users in the United States.[9] Despite the high prevalence of depression among methamphetamine users, little is known about the clinical course of depression during treatment for methamphetamine use or how depression affects clinical outcomes.[9]

To our knowledge, our study is the first to examine the relationship between depression and the response to treatment for methamphetamine use. Our aim was to determine whether depression among regular methamphetamine users moderates the response to brief psychological treatment focused on methamphetamine use. It was expected that participants with comorbid depression would report more severe symptom profiles at baseline and a poorer response to treatment for methamphetamine use.

METHODS

A detailed description of our study methods has been reported elsewhere.[10,11] We give only a brief outline here.

The study was conducted between 2001 and 2005 at two sites in Australia: the Hunter Region of New South Wales and the city of Brisbane, Queensland.

ABSTRACT

Objective: To determine whether the presence of comorbid depression influences response to psychological treatment for methamphetamine use.

Design: Randomised controlled clinical trial.

Setting and participants: Our study was conducted between 2001 and 2005 at two sites in Australia: the Hunter Region of New South Wales and the city of Brisbane, Queensland. The 214 participants, who were all using methamphetamine at least once a week in the month prior to the study, were self-referred or referred from health services or drug and alcohol clinical services. Participants were divided into two groups based on whether or not they had depressive symptoms at baseline.

Interventions: The control group received only a self-help booklet; the two treatment groups received either two or four counselling sessions involving cognitive behaviour therapy and motivational interviewing techniques to manage methamphetamine use.

Main outcome measures: Changes in methamphetamine use and depression at 5 weeks and 6 months after baseline.

Results: Over 70% of participants met criteria for depression at baseline, and depression was associated with significantly greater severity of methamphetamine use and related issues. Benzodiazepine use was significantly higher among depressed than non-depressed participants. Reductions in methamphetamine use between baseline and 5 weeks were independently predicted by comorbid depression, in favour of increased change among those with baseline depression. Depressed participants who received three or four counselling sessions showed a significant reduction in depression at 5 weeks. However, reductions in methamphetamine use and depression compared with baseline were no longer evident at 6 months.

Conclusions: Over the short term, comorbid depression did not negatively affect response to treatment, with some evidence of a dose–response treatment effect for reduction in depression. This was not maintained at 6 months, indicating that methamphetamine-focused treatment may not enable people with comorbid depression to make sustained improvement at the level of their counterparts without depression.

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Participants

The inclusion criterion was current use of methamphetamine, at least once a week, and participation was limited to people aged 16 years or over. People with psychotic disorder or suicidal ideation were excluded. Potential participants were screened by telephone to determine eligibility for the study.

Of a total of 282 people referred, 68 (24%) were excluded for various reasons: failing to meet the inclusion criterion (27), having a psychotic disorder (16), being at high risk of suicide (7), having an acquired cognitive impairment (4), or declining further participation after referral (14).[10] This left a final study sample of 214. Participants were self-referred (57 [27%]) or referred from health services (20 [9%]) or drug and alcohol clinical services (137 [64%]),

including community counselling, detoxification programs, needle and syringe programs, pharmacotherapy services (eg, for heroin dependence) and general practices.

Assessment instruments

Basic demographic information was collected, along with the following specific assessments at baseline, 5 weeks and 6 months.

Beck Depression Inventory II (BDI-II)[12]

The BDI-II is a 21-item self-report questionnaire used to screen for the presence of depressive symptoms over the previous 2-week period. The maximum possible score is 63 points. It is commonly used to screen for depressive symptoms among people with substance misuse problems.[13]

Respondents scoring in the clinical range (≥ 20 points) should be referred for further assessment for major depressive disorder.[12]

Structured Clinical Interview for DSM-IV, Research Version (SCID-IV-RV)[14]

The SCID-IV-RV provides a diagnostic, clinician-rated measure of methamphetamine misuse and dependence based on criteria set out in the *Diagnostic and statistical manual of the mental disorders*, 4th edition (DSM-IV).[15] For the purposes of our study, SCID diagnosis was dichotomised into two separate variables, indicative of misuse/no misuse and dependence/no dependence.

Severity of Dependence Scale (SDS)[16]

The SDS is a five-item self-report scale used to assess dependence. It is based on criteria for psychological dependence outlined in DSM-IV.[15] Scores above 4 for methamphetamine users are indicative of dependence, and the maximum possible score is 25.

Opiate Treatment Index (OTI)[17]

The OTI quotient (Q) score is a self-report measure of the quantity and frequency of use of 11 substances, including methamphetamine. The OTI Q score represents the average number of use occasions in the previous month. A score of 1 equates to once daily use, a score of 0.14 to once weekly use.

Based on their OTI scores, participants were given a polydrug use rating for the previous month, obtained by adding together the number of drugs in the previous month with an OTI score greater than zero.[17]

Procedure

After providing consent, participants completed the baseline assessment, which took about 45 minutes. Reimbursement of expenses ($20) was provided for each assessment completed (at baseline, 5 weeks and 6 months). After the baseline assessment, participants were randomly allocated to one of three treatment groups, with participants and assessors blind to allocation until that point. The treatment conditions offered were: (a) control (provision of a self-help booklet, *A user's guide to speed* [available at http://ndarc.med.unsw.edu.au/ NDARCWeb.nsf/page/Resources#speed], but no further counselling) (n = 74); (b) two sessions of counselling (n = 74); or (c) four sessions of counselling (n = 66).

Participants allocated to counselling completed their first treatment session immediately after randomisation. Counselling sessions were typically 45–60 minutes in duration. After the initial treatment session, subsequent sessions occurred weekly for either 1 or 3 weeks, depending on the treatment allocation.

Follow-up was at 5 weeks and 6 months after the initial assessment, undertaken by a researcher blind to treatment allocation.

Content of the interventions

Structured treatment sessions were administered according to a treatment manual,[18] and focused on reducing methamphetamine use. Cognitive behaviour therapy and motivational interviewing techniques were combined in each treatment session.

Statistical analysis

Data were analysed using PASW Statistics 18 for Windows, release 18.0.0 (SPSS Inc, Chicago, Ill, USA).

Participants were regarded as having comorbid depression if their BDI-II scores at baseline were ≥ 20, the cut-off score for moderate depressive symptoms (these participants were defined as the "depressed group", others as the "non-depressed group"). This threshold was selected as sufficient to suggest that clinical levels of depression were present. Although many of the symptoms of methamphetamine withdrawal can mirror depressive symptoms, this threshold was also considered a valid estimation of actual depressive symptoms, given that a respondent would have endorsed 20 of the 21 items on the BDI-II scale.

The sample selected for analysis consisted of participants who completed the baseline assessment and at least one follow-up assessment (at 5 weeks or 6 months). These were defined as "follow-up completers". Differences between the depressed and non-depressed groups were examined using one-way analysis of variance (ANOVA) and χ^2 analyses.

Linear regression was used to predict change in methamphetamine scores between baseline and 5-week follow-up and baseline and 6-month follow-up assessments. Predictors in these models were sex, whether receiving pharmacotherapy for heroin dependence (yes/no), number of treatment sessions attended, and depression status (depressed/not depressed). We created an interaction variable that accounted for the level of depression and number of treatment sessions attended. This was also entered into each linear regression model as a predictor variable. Logistic regression was used to predict abstinence from metham-phetamine (yes/no) at 5 weeks and at 6 months using the same set of predictors.

Changes in depression between baseline and 5 weeks (n = 109) or 6 months (n = 111) were examined only for participants who met criteria for comorbid depression at baseline (the depressed group).

Ethics approval

Our study was carried out in accordance with the National Health and Medical Research Council's *National statement on ethical conduct in research involving humans*. Ethics approval was gained from the relevant human research ethics committees at each study site.

RESULTS

Baseline characteristics of the entire sample have been reported elsewhere.[11] The mean age of participants was 30 years (range, 16–55 years). No significant differences existed between treatment groups on a range of demographic and treatment variables and the primary measures at baseline, indicating that randomisation was successful.[11]

Profile of participants based on comorbid depressive symptoms

Sixty-three per cent of participants (n = 135) met criteria for methamphetamine use and comorbid depression at baseline. Demographic characteristics of participants who completed at least one of the post-treatment assessments (n = 187) are summarised in Box 1. Of these, 155 were assessed at 5 weeks and 153 at 6 months. A significantly higher proportion of males than females were in the non-depressed group (χ^2_1 = 8.697; P = 0.003). Consequently, sex was included as a covariate in subsequent analyses.

Rates of service use were comparable between depression groups. About 20% of each group (27 in the depressed group and 10 in the non-depressed group) had a history of treatment for another mental health condition, such as psychosis, personality disorder or attention deficit hyperactivity disorder. No significant differences existed between depressed and non-depressed participants in terms of previous treatment for a substance misuse problem (χ^2_1 = 0.964; P = 0.326) or psychiatric hospitalisations (χ^2_1 = 1.470; P = 0.480).

As found previously,[11] enrolment in pharmacotherapy for heroin dependence was associated with significantly lower baseline methamphetamine use (mean OTI score for

methamphetamine use, 0.86 [heroin pharmacotherapy] v 1.61 [no heroin pharmacotherapy]); $F_{1,186} = 8.086$; $P = 0.005$). Although not statistically significant, higher rates of pharmacotherapy for heroin dependence were observed in the depressed group compared with the non-depressed group (29% v 19%; $\chi^2_1 = 1.811$; $P = 0.178$). Hence pharmacotherapy-for-heroin status was included as a covariate in subsequent analyses.

Use of methamphetamines and other drugs

We have previously reported that urine samples were collected from 19/109 participants (17%) who completed the 6-month follow-up assessment at the research clinics associated with the trial. All test results were consistent with self-reported use of methamphetamines.[11]

Compared with the non-depressed group, methamphetamine use (based on OTI Q score) was twice as high in the depressed group at baseline ($F_{2,186} = 10.130$; $P = 0.002$ [Box 1]). There was a non-significant tendency for methamphetamine misuse (based on SCID ratings) to be higher in the depressed group (95% v 86%, $\chi^2_1 = 3.098$; $P = 0.078$). Similarly, methamphetamine dependence (based on SCID ratings) tended to be higher in the depressed group, but not significantly so (91% v 86%; $\chi^2_1 = 0.529$; $P = 0.467$). Self-reported dependence, as measured by the SDS, was significantly higher for the depressed group (8.86 [SD, 3.28] v 6.25 [SD, 3.69]; $F_{1,186} = 22.135$; $P < 0.001$).

Comorbid depression did not generally affect usage levels of drugs other than methamphetamines (Box 1). Exceptions were the use of benzodiazepines ($F_{1,186} = 7.039$; $P = 0.009$), tobacco smoking ($F_{1,186} = 5.565$; $P = 0.019$), and polydrug use ($F_{1,186} = 7.754$; $P = 0.006$), all of which were significantly higher in the depressed group.

The depressed group attended significantly more treatment sessions than the non-depressed group (mean, 2.88 v 2.08; $F_{1,82} = 4.650$; $P = 0.003$), but overall treatment completion rates were similar for both groups (78% [depressed group] v 67% [non-depressed group]; $\chi^2_1 = 4.054$; $P = 0.132$).

Changes in primary outcomes over time

One-way ANOVAs showed no significant differences between follow-up completers ($n = 187$) and non-completers ($n = 27$) at baseline according to age ($F_{1,213} = 0.001$; $P = 0.972$), level of depression ($F_{1,213} = 0.243$; $P = 0.623$), or methamphetamine use ($F_{1,213} = 2.197$; $P = 0.140$). There was also no significant difference in sex distribution between completers and non-completers ($\chi^2_1 = 0.048$; $P = 0.827$). A significantly higher proportion of follow-up completers than non-completers had attended all of their allocated treatment sessions (58% v 28%; $\chi^2_1 = 24.337$; $P < 0.001$).

1 Baseline demographic profile of methamphetamine users who completed at least one follow-up assessment (n = 187), by presence or absence of comorbid depressive symptoms

	Depression status at baseline	
Demographic factors	Comorbid depression* (n = 135)	No comorbid depression (n = 52)
Sex[†]		
Male	72 (53%)	40 (77%)
Female	63 (47%)	12 (23%)
Marital status		
Single, never married	86 (64%)	36 (70%)
Married/defacto	31 (23%)	8 (15%)
Separated/divorced/widowed	18 (13%)	8 (15%)
Cultural background		
Aboriginal/Torres Strait Islander	9 (7%)	2 (4%)
Anglo-Australian	115 (85%)	47 (90%)
European/Pacific rim	11 (8%)	3 (6%)
Study site		
Hunter Region	66 (59%)	26 (50%)
Brisbane	69 (51%)	26 (50%)
Qualifications		
Nil	27 (20%)	15 (29%)
Secondary school	35 (26%)	14 (27%)
Trade	59 (44%)	15 (29%)
Tertiary degree/diploma	14 (10%)	8 (15%)
Current income		
Wage/salary	22 (16%)	11 (21%)
Pension/benefit	113 (84%)	41 (79%)
Drug use scores	**Mean (SEM)**	**Mean (SEM)**
OTI Q score[‡]		
Methamphetamines[†]	1.64 (0.14)	0.87 (0.11)
Alcohol	2.30 (0.38)	2.58 (0.67)
Cannabis	6.53 (1.07)	6.40 (1.28)
Heroin	0.13 (0.04)	0.09 (0.06)
Other opiates	0.12 (0.04)	0.03 (0.03)
Cocaine	0.05 (0.03)	0.01 (0.01)
Hallucinogens	0.05 (0.02)	0.01 (0.01)
Benzodiazepines[†]	1.32 (0.27)	0.14 (0.08)
Tobacco[†]	18.46 (1.11)	13.77 (1.37)
Polydrug use score[†§]	4.53 (0.13)	3.88 (0.18)

OTI Q = Opiate Treatment Index quotient. * Comorbid depression was defined as Beck Depression Inventory II score ≥ 20. † $P < 0.05$ for difference between depressed and non-depressed groups. ‡ The OTI Q score indicates quantity/frequency of use during the month prior to assessment. A score of 1 equates to once daily use, a score of 0.14 to once weekly use. § Polydrug use was calculated by summing the number of drugs (including alcohol and tobacco) the participant used in the month prior to assessment. ◆

Prediction of methamphetamine use

Patterns of methamphetamine use at baseline, 5 weeks and 6 months for participants with and without comorbid depression are summarised in Box 2.

At 5 weeks, changes in methamphetamine use relative to baseline were significantly predicted by the linear regression model containing sex, pharmacotherapy-for-heroin status, number of sessions attended, comorbid depression, and the interaction between sessions attended and presence of comorbid depression ($F_{5,154}$ = 2.64; $P = 0.027$; $R^2 = 0.079$). Comorbid depression was the only independently significant predictor of reduction in methamphetamine use at 5 weeks' follow-up. However, using the same regression model, comorbid depression was not a significant predictor of change in methamphetamine use at 6 months ($F_{5,152}$ = 1.436; $P = 0.215$; $R^2 = 0.043$).
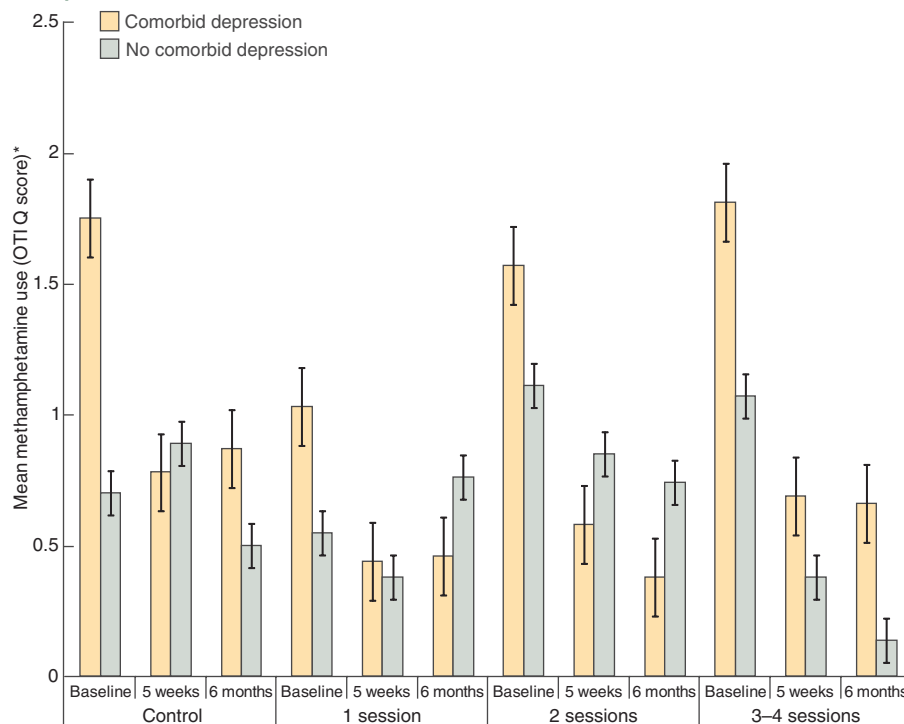
Abstinence from methamphetamine use was reported by 18% of participants at 5 weeks and 41% of participants at 6 months. Logistic regression was used to predict abstinence from methamphetamine use at 5 weeks and at 6 months using the following predictors: sex, pharmacotherapy-for-heroin status, number of sessions attended, comorbid depression and the interaction between sessions attended and comorbid depression. Changes relative to baseline were not significantly predicted by the regression model at 5 weeks ($\chi^2_5 = 4.891$; $P = 0.429$) or at 6 months ($\chi^2_5 = 8.232$; $P = 0.144$). However, at 6 months, the number of treatment sessions attended was a significant independent predictor of abstinence, suggesting that with each additional session attended (0, 1, 2, 3–4), the likelihood of abstinence increased by 1.5 times.

Prediction of depression

Participants who completed at least one follow-up assessment and who also met criteria for comorbid depression at baseline ($n = 135$) were selected for closer analysis of their depression scores at follow-up. Mean depression scores reported by this subgroup at each assessment time point are shown in Box 3.

At 5 weeks, changes in depression relative to baseline were significantly predicted by the linear regression equation ($F_{3,108}$ = 7.192; $P < 0.001$; $R^2 = 0.180$). The number of treatment sessions attended was the only independent predictor of changes in depression, such that attendance at more treatment sessions was predictive of a greater reduction in depression at 5 weeks. However, at 6 months, the linear regression model was not a significant predictor of change in depression ($F_{3,110}$ = 1.057; $P = 0.370$; $R^2 = 0.031$), and there were no independent significant predictors of change.

Most participants who were above the threshold for comorbid depression at baseline remained above that threshold at 5 weeks, regardless of whether they attended one treatment session (67% of group above threshold) or two (53% of group above threshold) or were in the control group (67% of group above threshold). However, only a quarter ($n = 16$) of those who attended three to four treatment sessions still met criteria for comorbid depression at 5 weeks ($\chi^2_3 = 12.652$, $P = 0.005$). This apparent treatment effect at 5 weeks among people with comorbid depression at baseline was no longer evident at 6 months, when 48%–51% were above the threshold for comorbid depression, regardless of the number of treatment sessions attended) ($\chi^2_3 = 0.083$; $P = 0.994$).

DISCUSSION

Our study is one of the first to report on baseline symptom severity and associated variables relating to methamphetamine users with comorbid depression, and the first to do so with Australian methamphetamine users.

Our results support those of a US study[9] in identifying an association between pre-treatment depression and increased severity of problems at presentation. Specifically, participants with comorbid depression reported significantly higher levels of methamphetamine use, dependence, polydrug use, tobacco and benzodiazapine use compared with their non-depressed counterparts. It is a significant concern that rates of benzodiazepine use among methamphetamine users with depression (a group more likely to develop dependence) were almost 10 times higher than rates among those without depression. Moreover, benzodiazepine use has been shown to exacerbate depressive symptoms.[19,20] It may be that depressed methamphetamine users take benzodiazepines to help them "come down" after methamphetamine use. This potentially keeps them in the cycle of increased use, leading to increased dependence and increased depression. Clinicians are advised to closely monitor and try to minimise benzodiazepine use among depressed methamphetamine users, and perhaps offer alter-

2 Self-reported levels of methamphetamine use at baseline, 5 weeks and 6 months, by number of treatment sessions attended and presence of comorbid depression at baseline

OTI Q = Opiate Treatment Index quotient. * Use occasions per day averaged over previous month. ◆

3 **Self-reported levels of depression at baseline, 5 weeks and 6 months among participants with comorbid depression* at baseline (*n* = 135), by number of treatment sessions attended**

BDI-II = Beck Depression Inventory II. * Comorbid depression was defined as BDI-II score ⩾ 20. ◆
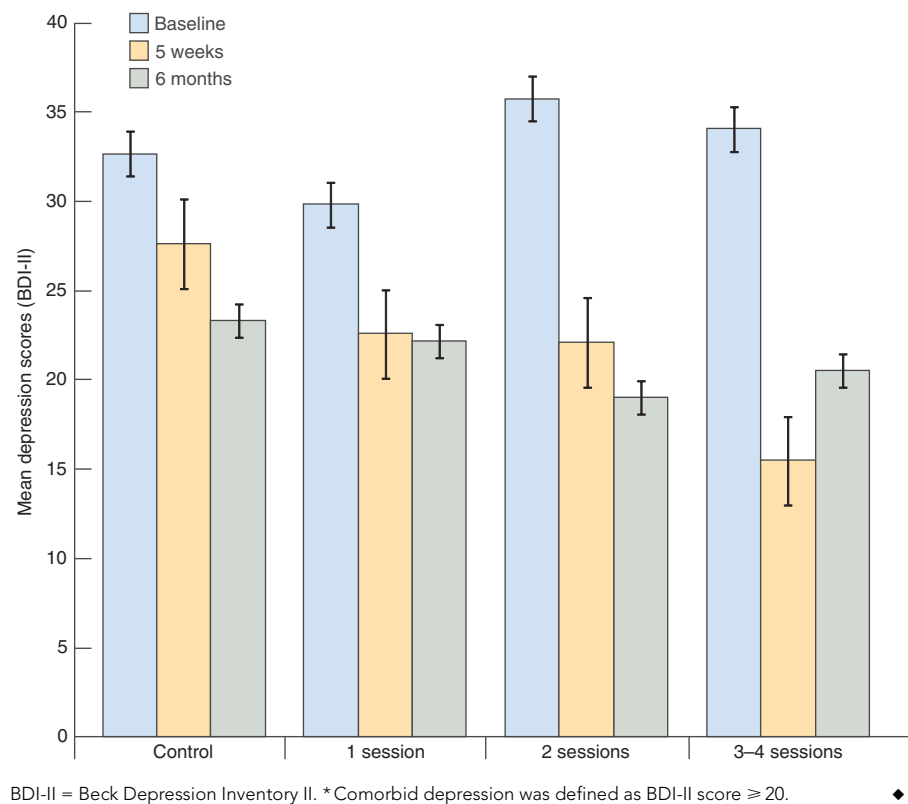
natives, such as antidepressant medication or psychological treatment, to better manage depression and other undesirable effects of methamphetamine in this group.

Comorbid depression was present in most methamphetamine users in our study, highlighting the need to screen for the presence of depression when treating methamphetamine users. In general, and contrary to expectation, our results showed that the presence of comorbid depression did not have a negative impact on treatment for methamphetamine use, at least in the short term.

Methamphetamine use outcomes

Comorbid depression independently predicted greater reduction in methamphetamine use between baseline and 5-week follow-up, but this was not maintained at 6 months. As the depressed group reported significantly higher methamphetamine use at baseline, this reduction may in part be explained by regression to the mean.

There was some evidence for a treatment effect on abstinence from methamphetamine use at 6 months, with attendance at three to four counselling sessions associated with almost five times the chance of achieving abstinence at this time point, independent of depression status. Despite the high initial severity of methamphetamine use and depression, the rates of abstinence achieved by the sample at 6-month follow-up were similar to those reported in other substance use research (eg, 30% abstinence from alcohol[21]), indicating the potential for methamphetamine users to reach and maintain their treatment goals similarly to users of other drugs.

Depression outcomes

There was some evidence that methamphetamine-focused treatment produced an accelerated reduction in depressive symptoms for people with comorbid depression, in that depression levels at 5-week follow-up were lower for people who attended three to four treatment sessions compared with those in the control group. However, the change was not maintained at 6-month follow-up. Considering the finding that dependence on methamphetamine was and remained signi-

ficantly higher among those with comorbid depression, people with comorbid methamphetamine use and depression may be at increased risk of relapse to methamphetamine use over the longer term, despite an initial treatment response. As methamphetamine-focused treatment may not be sufficient for sustained improvement, there is some support for considering an integrated model of treatment among people with comorbid methamphetamine use and depressive problems, offering extended, targeted psychological support for mental health symptoms combined with pharmacotherapy for depression if indicated.

Limitations

Several limitations to our study are worthy of note. Firstly, substance use among all participants was heavy and regular, with a range of reported psychological, educational and employment problems present. This likely made it difficult to detect a differential effect of depressive symptoms (present in the majority of cases) on these important outcomes. Nearly three-quarters of participants met criteria for comorbid depression and also had long histories of methamphetamine use. This too may have influenced the ability to effect change using a relatively brief intervention over a short period. We also did not determine whether a person's depression was independent of methamphetamine use or was methamphetamine-induced, which may have influenced how quickly their depression responded to the treatment provided. In addition, participants were largely recruited from drug and alcohol services, and the results can not necessarily be generalised to other settings.

In summary, our results suggest that it may be important to target comorbid depression in the context of methamphetamine use, given the higher levels of disability and morbidity reported by participants with comorbid depression, and the potential for participants to experience ongoing difficulties that could influence relapse and recovery from both conditions. This seems particularly important given the direct links between depressive symptoms at baseline and subsequent treatment retention and outcomes reported in previous research,[22] and the finding that conditions such as depression and substance misuse can respond to psychological and pharmacological treatment.[19]

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