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The effects of stereotype threat on cognitive function in ecstasy users

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Abstract

Stereotype threat occurs when individuals, believed to be intellectually inferior, perform badly on cognitive tests they perceive to confirm stereotypes about them. Due to the wide media coverage of studies purporting to show cognitive deficits in ecstasy users it is possible that they experience stereotype threat. This study tested ecstasy and non-ecstasy using polysubstance misusers on a variety of cognitive tests after they had been exposed to stereotype threat. This priming consisted of exposing them to information about the long-term effects of ecstasy which either stated that ecstasy caused memory loss or that it did not. Ecstasy users that had been primed that ecstasy did not cause cognitive deficits performed better than the other three groups on the delayed portion of the prose recall task from the Rivermead Behavioural Memory Test battery. There were no other statistically significant differences between any of the groups on any of the other cognitive tests used. This suggests that stereotype threat exists in ecstasy users and may be influencing their performance in experiments designed to identify cognitive deficits. In order to prevent this occurring in future studies, experimenters must be careful how they conduct their experiments and discuss their results with the media.

Keywords

ecstasy, MDMA, stereotype threat, cognition, polysubstance misuse

Introduction

The social stigma of intellectual inferiority ascribed to some groups within society, such as African Americans and women, has been hypothesized to be a significant contributor to their poor performance on tests, and this in turn may reinforce this perception of intellectual inferiority (Aronson *et al.*, 2002). As it is believed that these individuals are under extra pressure to succeed due to their fears that they will confirm the stereotypes held about them, this phenomenon is called stereotype threat (Steele and Aronson, 1995). Reducing the salience of these stereotypes reduces individuals' concerns that they might confirm the stereotype and improves performance. For example, Steele and Aronson (1995) found that African American students performed significantly worse than white students on a (standardized) test when the test was presented as a diagnosis of their intellectual abilities but not when it was presented in a neutral way. Similarly, women who were led to believe that a test had been biased against them performed worse than women who believed it was a gender neutral test (Spencer *et al.*, 1999). Stereotype threat has also been demonstrated in other ethnic groups (e.g. Gonzales *et al.*, 2002), the economically disadvantaged (Croizet and Claire, 1998), homosexual men (Bosson *et al.*, 2004), clinical populations with head injury (Suhr and Gunstad, 2002), the elderly (Levy, 1996), and even psychology students (Croizet *et al.*, 2004).

These studies suggest that stereotype threat creates an additional burden that interferes with cognitive ability. Increased anxiety has been observed in some, but not all, studies of stereotype threat and reducing anxiety reduces the impact of stereotype threat (Spencer *et al.*, 1999). Stereotype threat also reduces working memory capacity, which suggests a cognitive mechanism by which an individual's performance on a complex cognitive task may be impaired (Schmader and Johns, 2003). Taken together, these findings suggest that individuals experiencing stereotype threat have a higher mental workload. Croizet and colleagues (2004) demonstrated that heart rate variability was decreased in psychology students experiencing stereotype threat, suggesting that this threat had a psychophysiological effect. As heart rate variability is a useful measure of mental workload that is independent of performance outcomes, this demonstrates that individuals under stereotype threat have a higher mental workload.

Many studies of ecstasy users have demonstrated that they have poorer performance on a variety of cognitive tests relative to non-ecstasy using controls (e.g. Morgan, 1999; Thomasius *et al.*, 2003), though this is not always found to be the case (e.g. Simon and Mattick, 2002; Dafters *et al.* 2004). The hypothesized cause of

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these cognitive deficits is MDMA-induced serotonergic neurotoxicity (e.g. Parrott, 2001), a phenomenon detected after MDMA is repeatedly administered to non-human animals (e.g. Miller and O'Callaghan 1994; O'Loinsigh *et al.* 2000; Morley *et al.* 2004). The relationship between ecstasy use and cognitive function is widely reported in the media (e.g. 'Ecstasy leaves gaps in the memory', *Guardian*, UK 15/1/2004), and by government sponsored drugs education campaigns (e.g. Talk to Frank (www.talktofrank.co.uk) in the UK and NIDA Club Drugs in the USA (www.clubdrugs.org)), and therefore disseminated throughout the general population (Cole *et al.*, 2002a; Green, 2004). It is conceivable that this dissemination of information about brain damage and memory deficits may be creating stereotypes about intellectual inferiority in the minds of ecstasy users. If this is the case, then it is possible that ecstasy users may be experiencing stereotype threat when they participate in experiments that they believe to be testing their memory. A direct test of this hypothesis would be to replicate previous stereotype threat experiments where the stereotype (such as intellectual inferiority in African Americans (Steele and Aronson, 1995)) is primed prior to the test in order to produce performance decrements. To do this, experimental participants would need to be primed with information that ecstasy use causes brain damage that leads to permanent memory deficits in users. This priming should have no effect on the performance of non-ecstasy users (relative to those who are not primed). However, ecstasy users should show poorer memory scores after being primed than ecstasy users who are not primed, as they are experiencing stereotype threat.

The purpose of this study was to investigate the possible contribution of stereotype threat to ecstasy users' performance on measures of verbal memory, working memory and executive function that have frequently been employed in studies of ecstasy users, by comparing test performance in ecstasy users and controls primed with information supportive of the link between ecstasy use and brain damage and ecstasy users and controls given information that does not support this link. It was hypothesized that ecstasy users would perform worse than controls in the prime condition while the two groups would perform similarly when not primed.

Methods

Subjects

Sixty-eight unpaid volunteers (aged 18–36) were recruited by means of posters across the University campus and city centre, Internet advertisements, the 'snowball' technique and the key informant access method, which are all standard methods of recruitment for this type of research. People were excluded from study participation if they reported their first language was not English, current or past use of prescribed psychiatric medication, lifetime diagnosis of a psychological or affective disorder and lifetime diagnosis of a psychological or affective disorder in an immediate family member. Current users of heroin or crack cocaine would also have been excluded but none were recruited. Subjects were allocated to one of four sex matched groups ($n = 17$/group), according to self-reported drug history and experimental condition (see below). Polysubstance misusers who reported lifetime use of ecstasy were classed as ecstasy users, which is standard for this type of experiment. Both controls and ecstasy using groups had abstained from using any substance for a mean of 10.3 ± 5.9 and 7.1 ± 0.9 days respectively. All control subjects were alcohol users with experiences of illicit substances, but who did not report a lifetime use of ecstasy. All participants self-reported being drug free on the day of testing but this was not objectively verified due to lack of funds. Written informed consent was obtained from all volunteers and the study proceeded with the approval of the Research Ethics Committee of the University of Liverpool.

Questionnaire design

Substance use assessment A questionnaire, similar to that used in our previous work (e.g. Sumnall *et al.*, 2004) collected a variety of substance use related information, including lifetime and last month prevalence and frequency, and simultaneous polysubstance misuse. Additional questions relating to the previous month were included as indicators of current substance use. Whilst use of a wide variety of substances was assessed, more detailed information was requested for alcohol (number of units drunk/week; number of episodes/week), cannabis (number of joints smoked/week; number of episodes/week) and ecstasy (number of tablets taken/episode). The questionnaire also included items regarding volunteer's beliefs about the effects of drug use on memory and psychopathology, and whether they believed media reports about drugs. These measures were included to act as attitudinal covariates in other analyses. Naloxone was included to identify subjects who falsely reported their drug use. Participants who reported the use of naloxone would be removed from the study as their self-reported drug use would be deemed unreliable.

Substance use disorder and psychopathology Problematic substance misuse and self-reported levels of anxious and depressive symptomatology were measured with the Alcohol Use Disorders Identification Test (AUDIT; Babor *et al.*, 2001), the Severity of Dependence Scale for cannabis (SDS-Cannabis; Swift *et al.*, 1998), the Drug Abuse Screening test (DAST; Skinner 1982) and the Hospital Anxiety and Depression Scale (HADS; Johnston *et al.*, 2000).

The AUDIT is a ten item screening and research questionnaire for early detection of harmful alcohol use, with subscales assessing patterns of alcohol consumption, drinking behaviour, adverse reactions and problem drinking. The AUDIT assesses current drinking behaviour and discriminates between hazardous and non-hazardous drinking. The SDS-Cannabis was derived from the original SDS (Gossop *et al.*, 1995) by Swift and colleagues (1998) and has excellent psychometric properties. There is a strong association between SDS score and those behavioural patterns of substance use indicative of drug dependence (Gossop *et al.*, 1995). The DAST is a quantitative indicator of problematic use for substances other than alcohol and tobacco for non-medical purposes.

Cognitive tests

Volunteers were required to complete the prose recall task from the Rivermead Behavioural Memory Test battery (RBMT; Wilson *et al.*, 1985), Digit Span (Weschler, 1981), and the Controlled Oral Word Association (COWA) task (Troyer *et al.*, 1998).

The RBMT prose recall test is a short and reliable measure of everyday memory, used to measure immediate and delayed recall. A brief news story is played from an audio CD, and the subject is required to recall words and ideas immediately after presentation, and then after a 20 minute delay. Forward and backward digit span was taken from the revised Weschler Adult Intelligence Scale (WAIS-R). In this task, subjects are required to repeat progressively longer sequences of numbers in both forwards and backwards directions. The COWA task assessed verbal fluency and required the generation of words beginning with the letters F, A and S in individual 1 min periods. Three scores were generated: the total number of words; clusters, which are words beginning with the same first two letters, words differing by only a vowel sound, rhymed or were homonyms; and switches, which are the number of shifts between clusters. Frequent switching between clusters reflects optimal verbal fluency performance (Troyer *et al.*, 1998).

Procedure

Before completing the questionnaire and cognitive tests, volunteers were provided with a study information sheet, which served as the stereotype threat manipulation. Half of the participants (17 ecstasy users and 17 controls) received the *prime* version, which stated that there was strong evidence linking ecstasy use and mnemonic dysfunction. The remaining participants (17 ecstasy users and 17 controls) received the *no prime* information, which asserted that there was no conclusive evidence supporting a link. The manipulations were also verbally reinforced by the experimenter (KM) after the subject had read the information sheet. Volunteers then completed the questionnaire and the cognitive assessments. Heart rate was recorded with a commercially available monitor before and after the experimental sessions in order to assess physiological responses to task performance (Boots Plc, UK). After completing all of the tasks, participants were debriefed, the purpose of the experiment was explained to them, and they were informed that they had been misled as part of the procedure and given accurate information about the long-term effects of controlled drug use.

Statistical analysis

Differences in drug use parameters were examined with one-way ANOVA and chi square tests. Two-way ANOVA with planned comparisons was used to examine the effects and interactions of ecstasy use, priming status and gender on test scores. Change in heart rate was examined with repeated measures ANOVA. Backwards stepwise linear and logistic regression was used to explore demographic, drug use, attitudinal, test and scale variables. Relationships between self-reported drug use parameters were further analysed by Spearman's correlation with a *modified* Bonferroni correction applied to significant results to control for type I statistical errors (Jaccard and Wan, 1996). A significance level of $p < 0.05$ was set for all tests and data were analysed using SPSS v 12.

Results

It was found that 83.8% of all participants believed that drug use caused memory loss and 76.5% that drug use caused psychiatric problems, like depression. Table 1 displays substance use parameters reported by the four groups. A higher proportion of ecstasy users reported a lifetime use of amphetamine, cannabis, cocaine, ecstasy and LSD than control subjects, although there were no differences in drug use between primed and unprimed ecstasy users (see Table 1 for χ^2). Furthermore, both groups of ecstasy users reported more frequent use of cannabis and a greater number of episodes and joints smoked in the previous week; more frequent use of, and fewer days of abstinence from, cocaine; and more frequent use of LSD (although only the primed ecstasy group reported use in the last month) (see Table 1 for F and P values). The frequency of ecstasy use per month was found to correlate positively with monthly use of amphetamine ($r = 0.772$, $p < 0.001$), monthly use of poppers ($r = 0.801$, $p < 0.001$), monthly use of cocaine ($r = 0.403$, $p < 0.05$) and the number of ecstasy tablets taken per episode ($r = 0.744$, $p < 0.001$).

Drug use. Table 2 shows descriptive statistics for all four experimental groups on age, physiological and substance use measures. Two way ANOVA showed significantly higher total SDS-cannabis ($F_{1,64} = 34.809$, $p < 0.001$) and DAST ($F_{1,64} = 112.797$, $p < 0.001$) scores for ecstasy users when compared with controls. In order to examine those measures that predicted DAST and SDS-cannabis outcomes further, drug use variables that had previously been shown to significantly correlate with total DAST and SDS-cannabis score (data not shown), were entered into independent backwards-stepwise linear regression analyses. SDS-cannabis scores were significantly predicted ($R^2 = 0.627$, $p < 0.01$) by DAST ($\beta = 0.147 \pm 0.047$, $p < 0.01$), ecstasy tablets per use episode ($\beta = 0.004 \pm 0.001$, $p < 0.001$) and monthly frequency of ecstasy ($\beta = 0.705 \pm 0.165$, $p < 0.001$), ketamine ($\beta = 0.982 \pm 0.287$, $p < 0.01$) and LSD ($\beta = 2.108 \pm 0.756$, $p < 0.001$). DAST score was significantly predicted ($R^2 = 0.863$, $p < 0.001$) by the number of ecstasy tablets taken per use episode ($\beta = 0.024 \pm 0.001$, $p < 0.001$); the monthly frequencies of use of amphetamine ($\beta = 4.757 \pm 0.860$, $p < 0.001$), ecstasy ($\beta = 1.944 \pm 0.400$, $p < 0.001$), ketamine ($\beta = 2.617 \pm 1.203$, $p < 0.05$), poppers ($\beta = 2.211 \pm 0.491$, $p < 0.001$), cigarettes ($\beta = 0.035 \pm 0.005$, $p < 0.001$) and GHB ($\beta = 2.282 \pm 1.110$, $p < 0.05$); fewer days of abstinence since last use of ecstasy ($\beta = 0.185 \pm 0.070$, $p < 0.05$) and poppers ($\beta = 0.340 \pm 0.111$, $p < 0.01$); and the number of occasions that cannabis was used in the previous week ($\beta = 0.869 \pm 0.140$, $p < 0.001$).

ANOVA of within and between factors was performed and showed an interaction between the priming condition and ecstasy user status for the delayed component of the prose recall task from the RBMT ($F_{1,60} = 4.174$, $p < 0.05$; Table 3, Fig. 1) but none of the

Table 1 Drug use variables in experimental groups[a]

	Ecstasy prime $n = 17$	Ecstasy no prime $n = 17$	Control prime $n = 17$	Control no prime $n = 17$	Between group differences F/χ^2
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	
Alcohol					
% reporting lifetime use	100.0	100.0	100.0	100.0	–
Frequency of use (days)	11.36 ± 9.72	12.59 ± 3.35	8.47 ± 5.58	13.88 ± 5.81	1.004
Days since last use[a]	2.62 ± 1.98	3.94 ± 2.91	2.69 ± 1.89	3.59 ± 4.14	1.372
Episodes/week	2.41 ± 1.46	2.18 ± 1.88	2.06 ± 1.35	2.82 ± 1.74	0.738
Units/week	18.76 ± 22.76	14.94 ± 16.53	10.82 ± 9.48	12.35 ± 12.11	0.798
Amphetamine					
% reporting lifetime use	41.2	47.1	0.0	5.9	16.346**
Frequency of use (days)	4.67±3.22	2.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	2.016
Days since last use[a]	–	14.00 ± 0.00	–	–	0.399
Cannabis					
% reporting lifetime use	94.1	88.2	58.8	58.8	9.647*
Frequency of use (days)	21.71 ± 8.34	15.47 ± 10.06	1.67 ± 0.58	3.67 ± 2.31	22.25***
Days since last use[a]	1.54 ± 0.66	3.31 ± 4.63	20.00 ± 0.00	7.00 ± 0.00	1.164
Episodes/week	3.35 ± 2.55	3.24 ± 2.39	0.00 ± 0.00	0.06 ± 0.24	19.794***
Joints/week	23.12 ± 31.42	12.35 ± 11.41	0.00 ± 0.00	0.12 ± 0.49	7.513***
Cigarettes					
% reporting lifetime use	82.4	82.4	58.8	64.7	3.725
Frequency of use (days)	27.62 ± 7.26	25.07 ± 13.21	12.14 ± 14.22	16.75 ± 12.66	2.413
Days since last use[a]	1.00 ± 0.00	1.00 ± 0.00	9.50 ± 6.36	7.60 ± 12.58	0.628
Cocaine					
% reporting lifetime use	94.1	76.5	0.0	11.8	44.760***
Frequency of use (days)	2.33 ± 1.97	2.50 ± 1.41	0.00 ± 0.00	0.00 ± 0.00	7.454***
Days since last use[a]	13.43 ± 5.94	8.33 ± 3.14	0.00 ± 0.00	0.00 ± 0.00	6.064**
Ecstasy					
% reporting lifetime use	100.0	100.0	0.0	0.0	–
Frequency of use (days)	3.82 ± 2.83	2.94 ± 1.60	0.00 ± 0.00	0.00 ± 0.00	–
Days since last use[a]	10.25 ± 5.94	7.14 ± 0.86	0.00 ± 0.00	0.00 ± 0.00	–
Tablets/episode	2.94 ± 3.33	3.44 ± 1.75	0.00 ± 0.00	0.00 ± 0.00	–
LSD					
% reporting lifetime use	47.1	35.3	0.0	11.8	13.077**
Frequency of use (days)	1.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	3.429*
Days since last use[a]	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	–

[a]If reported use in the previous month. *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$.

Table 2 Age, heart rate and substance use parameters in the experimental groups

	Ecstasy prime	Ecstasy no prime	Control prime	Control no prime	Between group differences
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	
Age	23.35 ± 4.15	23.82 ± 2.96	25.24 ± 5.35	25.24 ± 4.68	–
AUDIT	10.88 ± 6.54	10. 88 ± 6.62	8.59 ± 5.84	10.76 ± 6.54	–
SDS Cannabis	2.29 ± 2.09	1.82 ± 1.43	0.18 ± 0.73	0.12 ± 0.49	Condition***
DAST	7.88 ± 4.00	8.47 ± 3.43	1.12 ± 1.45	0.94 ± 0.97	Condition***
Heart rate 1	75.76 ± 11.81	74.24 ± 11.33	74.18 ± 15.53	74.24 ± 11.22	–
Heart rate 2	80.24 ± 13.73	76.82 ± 11.53	76.71 ± 17.36	77.35 ± 17.95	–
Δ Heart rate	4.47 ± 9.58	2.59 ± 7.27	2.53 ± 6.93	3.12 ± 11.77	–

Shown are mean and standard deviations, and significant between subjects effects. *Condition*, ecstasy user status; ***$p < 0.001$. Δ Heart rate refers to the change in heart rate from the first to the second measurement.

Table 3 Summary of overall ANOVAs on outcome measures: results of 2 × 2 × 2 factorial model (Group: ecstasy user vs control × prime: priming vs control × gender: male vs female)

Source	F df	HADS-A	HADS-D	RBMT-I	RBMT-D	Digit span-F	Digit span-B	Digit span-T	FAS-μ	FAS-N	FAS-S	ΔHR
User status	1	6.485*	0.026	1.996	0.727	2.134	0.101	0.551	0.161	0.368	1.052	0.086
Prime	1	1.425	1.770	0.378	2.275	1.985	1.064	2.398	0.295	0.629	0.358	0.045
Gender	1	0.247	7.962**	1.266	0.564	0.272	2.780	1.750	2.552	4.489*	1.997	0.142
Gender × user status	1	0.125	0.869	0.613	0.222	0.061	0.022	0.044	1.395	0.102	0.711	0.242
Gender × prime	1	1.602	0.731	1.071	0.674	2.173	0.157	0.396	0.002	1.123	0.914	0.300
User status × prime	1	0.732	0.076	2.595	4.174*	0.090	0.557	0.059	0.002	0.002	0.044	0.270
Gender × user status × prime	1	0.132	2.092	0.269	0.000	0.221	0.519	0.600	1.177	0.003	0.149	1.693

*$p < 0.05$; **$p < 0.01$; ***$p < 0.001$. Degrees of freedom for error = 60. RBMT-I/D, Rivermead Behavioural Memory Test, immediate/delayed; Digit Span-F/B/T, Digit Span test, forward/backwards/total scores; FAS-μ/N/S, FAS test, mean cluster/number of words/switches; ΔHR change in heart rate across experimental session.

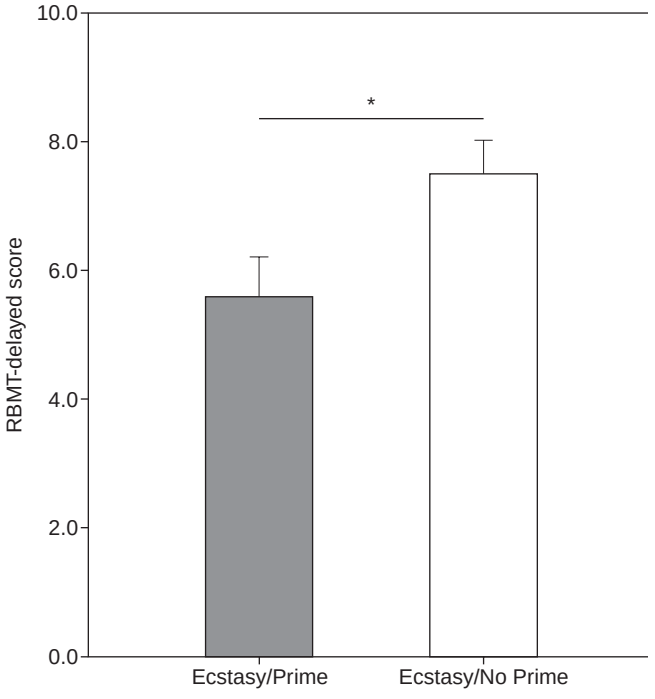

Figure 1 Effect of the presentation of the experiment on delayed verbal recall. *$p < 0.05$ significant difference between groups

other tests. Simple effects were further examined by t-test, which showed that volunteers reputed to have poorer cognitive capability (i.e. ecstasy users) performed worse when they were primed to believe that the experiment was designed as confirmation of poor memory function compared to those who were primed to believe that the experiment was exploratory ($t = 2.026$, $p < 0.05$). This effect remained significant after controlling (using ANCOVA) for self reported anxiety and depression, the belief that ecstasy use led to memory loss, frequency of ecstasy use and length of cannabis and ecstasy abstinence (data not shown). Additionally, there was an effect of ecstasy user status on the anxiety component of the HADS, with control subjects scoring higher than ecstasy users ($F_{1,60} = 6.485$, $p < 0.05$). Males scored higher than females on the depression component of the HADS ($F_{1,60} = 7.962$, $p < 0.01$). There were also gender differences on the number of words generated in the COWA task ($F_{1,60} = 4.489$, $p < 0.05$), with males generating more than females ($t = 2.222$, $p < 0.05$). There were no significant associations between drug use variables and psychopathological and cognitive test scores.

Backwards stepwise logistic regression was used to identify predictors of beliefs about drug use. In separate analyses, the dependent variables were 'taking notice of media reports about ecstasy', the belief that 'drug use causes memory loss', and the belief that 'drug use causes psychiatric problems'. Taking notice of media reports was predicted ($\chi^2 = 29.310$, df = 6, $p < 0.001$; Nagelkerke $R^2 = 0.499$) by lower score on the depression component of the HADS (Wald = 5.854, $p < 0.05$; exp(β) = 0.690), higher heart rate at the beginning of the experimental session (Wald = 4.850, $p < 0.05$; exp(β) = 1.101), and a lower number of ecstasy tablets consumed per year (Wald = 5.883, $p < 0.05$; exp(β) = 0.976). Believing that drug use causes memory loss was predicted ($\chi^2 = 38.579$, df = 14, $p < 0.001$; Nagelkerke $R^2 = 0.737$) by being male (Wald = 5.468, $p < 0.05$; exp(β) = 8.964); scoring higher on both the anxiety and depression components of the HADS (Wald = 4.802, $p < 0.05$; exp(β) = 4.873; Wald = 4.244, $p < 0.05$; exp(β) = 11.255 respectively) scoring higher on delayed recall in the RBMT (Wald = 4.527, $p < 0.05$; exp(β) = 4.516); generating more words (Wald = 4.004, $p < 0.05$; exp(β) = 6.046), but performing fewer clusters (Wald = 4.568, $p < 0.05$; exp(β) = 0.000) and switches (Wald = 4.055, $p < 0.05$; exp(β) = 0.078) on the FAS; having a slower heart rate at the beginning of the session (Wald = 3.985, $p < 0.05$; exp(β) = 0.644),

but faster at the end (Wald = 4.420, $p < 0.05$; exp(β) = 1.739); and a fewer number of ecstasy tablets consumed per year (Wald = 4.189, $p < 0.05$; exp(β) = 0.984). Believing that drug use causes psychiatric problems was predicted ($\chi^2 = 5.175$, df = 1, $p < 0.05$; Nagelkerke $R^2 = 0.110$) by higher scores on the anxiety component of the HADS (Wald = 4.310, $p < 0.05$; exp(β) = 1.295).

Discussion

Ecstasy users who were primed that ecstasy use damaged the brain and led to memory problems recalled fewer items in the delayed component of the prose recall task from the RBMT than those ecstasy users who were not primed. This suggests that these individuals may be experiencing stereotype threat as the overwhelming majority of the participants believed that ecstasy use caused memory loss and psychiatric problems. Therefore, the effects of stereotype threat may be a greater problem in this type of experiment than is currently believed. This supports the view that media reports of ecstasy research may be contributing to the phenomena being investigated (Cole *et al.*, 2002a). As the non-primed ecstasy users selectively demonstrated better memory performance relative to the other three groups, the normal confounds in the interpretation of this type of data do not apply. That is, intoxication, withdrawal, sleep deprivation, circadian disruption, nutritional deficiencies, (pre-morbid) psychiatric problems and polysubstance misuse would all be predicted to reduce performance not improve it (Curran, 2000; Cole *et al.*, 2002a; 2002b; 2002c; 2002d; Cole and Sumnall, 2003). Similarly, it is unlikely that an unselected group of ecstasy users would be different from their ecstasy using peers.

The selective effect of priming on the delayed component of the prose recall task from the RBMT is probably due to the fact that this is the most obvious memory test employed. As the priming condition focused specifically on memory function, it is possible that this test generated the greatest degree of threat for the primed participants. Although the digit span tests are also memory tests it is possible that they were not of sufficient complexity to demonstrate the performance decrements observed in other stereotype threat studies (e.g. Spencer *et al.,* 1999; Schmader and Johns, 2003).

These data also demonstrate that there were no differences between non-ecstasy using controls and (primed) ecstasy users, which is consistent with a number of other studies (e.g. Morgan *et al.*, 2002; Thomasius *et al.*, 2003; Curran and Verheyden, 2003; Dafters *et al.*, 2004; Fisk *et al.*, 2004). This suggests that the detrimental effects of ecstasy use on cognitive function may not be as robust as is currently believed (e.g. Parrott, 2001). Given that ecstasy users are typically polysubstance misusers (e.g. Fisk *et al.*, 2004; Sumnall *et al.* 2004), it is highly likely that other controlled drugs are contributing to the observed data and thus creating inconsistency. It is theoretically possible that this polysubstance misuse may affect both the short- and long-term impact of ecstasy use, in particular MDMA-induced serotonergic neurotoxicity (e.g. Miller and O'Callaghan, 1994; O'Loinsigh *et al.*, 2000; Cole and Sumnall, 2003; Morley *et al.*, 2004). In addition, it is highly likely that the increasing use of controlled drugs per se produces the observed deficits rather than any specific compound.

As the primed ecstasy users and the non-user controls were comparable on the delayed component of the prose recall task of the RBMT it is possible that the stereotype threat manipulation worked in the opposite way to that hypothesized. That is, the conflict between the priming information and the negative stereotype led to extra task involvement and thus higher performance (MacKinnon *et al.*, 1985). The failure to demonstrate this with immediate recall may be due to a ceiling effect with this task in the normal population (as it was designed to detect cognitive deficits). Further work is needed to clarify this.

As with all studies of this nature, there are numerous methodological problems with this study, some of which are detailed above (e.g. Curran 2000; Cole *et al.*, 2002a; Cole and Sumnall, 2003). It is probable that a large number of the participants may have been recently intoxicated, as it is quite common for this population to use controlled drugs on a near daily basis (Sumnall *et al.*, 2004). However, as stated above, this is unlikely to have led to a selective improvement in memory performance over non-drug using controls. Despite this, controlled drug use should have been quantified; however, without financial support this was not possible. It is common practice to use a measure of pre-morbid cognitive functioning, such as the National Adult Reading Test (NART), to ensure that the experimental groups were theoretically comparable. As this experiment used manipulations both within and between groups, this was likely to have been of little benefit. Therefore it was felt that adding such a measure unnecessarily lengthened the procedure for participants who were not being reimbursed for their participation. It is possible that there were pre-morbid differences between the groups. However, improvement due to the priming was so selective (i.e. to the test of delayed prose recall) that it seems unlikely to be due to pre-morbid differences. It is also possible that these users had not used enough ecstasy to have damaged their brains and/or caused memory loss. However, the users in Morgan (1999) had only used 50 tablets on average, suggesting that deficits on the prose recall task from the RBMT can be detected after only limited ecstasy use. In addition, it is the dose of MDMA taken which is the key to neurotoxicity, rather than cumulative exposure to ecstasy tablets of unknown content (O'Shea *et al.* 1998; Cole *et al.*, 2002c; 2002d; Green, 2004). In the UK during 2001, the average dose, in milligrams, of ecstasy tablets containing MDMA was 73 mg (Cole *et al.*, 2002d) and therefore it is likely that these participants used an average of 3 mg/kg MDMA per use episode (assuming an average 70kg male) if this trend continued into 2004. It has been argued that this dose is large enough to be neurotoxic (McCann and Ricaurte, 2001), however this claim has been widely disputed (e.g. Kish 2002; de la Torre and Farré, 2004).

These data suggest that a large proportion of young people have been exposed to, and believe, the research that purports to demonstrate that even moderate ecstasy use causes memory loss and psychiatric problems. However, no participants were excluded from the study because they self-reported a history of psychiatric problems and neither did any experience clinically-relevant

anxious and/or depressive symptomatology (as indicated by the HADS), suggesting that they were not suffering with clinically-relevant psychiatric problems despite believing that they should be. This may be due to the use of a scale that is not confounded by the somatic effects of controlled drug use (Sumnall and Cole, 2005). Similarly, the ecstasy users' cognitive functioning was comparable to that of their non-ecstasy using peers. During the study, none of them self-reported that they had experienced memory loss, which is consistent with previous studies (e.g. Heffernan *et al.*, 2001). Overall, this suggests that ecstasy users believe what they are told about the detrimental effects of ecstasy use on memory, but do not experience these problems themselves. Though they might believe that negative effects of ecstasy occur in others, ecstasy users' response to a prime meant to trigger stereotype threat suggests that they, like members of other groups facing negative stereotypes, may inadvertently lend more support to beliefs about the negative effects of ecstasy use because of their efforts to dispel the stereotype. This may create a problem for public health interventions aimed at reducing both the short- and long-term harm from ecstasy use. That is, users may believe that the probability of these negative things happening to them to be very low despite the fact that these problems exist (Gamma *et al.*, 2005). This suggests that media reports about the short- and long-term effects of ecstasy use may actually be counterproductive (Cole *et al.*, 2002a; Green, 2004). Further work on risk perception is clearly needed to investigate this.

Conclusions

The findings reported here suggest that stereotype threat may be contributing to the observed memory deficits in ecstasy users detected in other research studies. If this is the case, then researchers need to take extra precautions to guard against inducing or amplifying stereotype threat in their studies of cognitive function in this population. They need to examine advertising strategies and materials explaining the nature of the study, and they may consider ways to reduce possible communication of their beliefs concerning ecstasy effects on cognition. Researchers also need to be careful how they discuss their findings with the media as they may be creating a self-fulfilling prophecy by giving more legitimacy to this stereotype, hence creating conditions of stereotype threat in the very people they seek to examine for signs of cognitive deficit.

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Conflict of interest statement

Lisa Jerome works as a research associate for the Multidisciplinary Association for Psychedelic Studies (MAPS), a non-profit organization currently funding clinical trials of MDMA-assisted psychotherapy. MAPS, however, did not provide any financial support for her collaboration on this study.

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