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Anxiety, Depression, & Behavioral Symptoms of Executive Dysfunction in Ecstasy Users: Contributions of Polydrug Use

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Abstract

Background—Given ecstasy's (MDMA) potential serotonergic neurotoxicity, it is plausible that regular ecstasy users would have an elevated prevalence of behavioral executive dysfunction or mood symptoms. However, recent studies have found that the relationship between ecstasy use and psychological symptoms was no longer significant after controlling for marijuana use (e.g., Morgan et al., 2002). The goal of the present study was to examine the relationship between ecstasy exposure and self-reported executive functioning and psychological symptoms after controlling for gender, ethnicity, and other drug use.

Methods—Data were collected from 65 men and women with a wide range of ecstasy use (including 17 marijuana-using controls). Participants were administered the Frontal Systems Behavioral Scale, State-Trait Anxiety Inventory for adults, and the Beck Depression Inventory-2nd Edition.

Results—Although 19–63% of the ecstasy users demonstrated clinically elevated psychological symptoms, frequency of ecstasy use did not predict the psychological symptoms. No gender differences or interactions were observed.

Conclusions—These results revealed that, although ecstasy users demonstrate elevated levels of psychological symptoms and executive dysfunction, these symptoms are not statistically associated with their ecstasy consumption. Instead, other drug use (alcohol, marijuana, opioids, and inhalants) significantly predict psychological symptoms in this sample of polydrug users.

Keywords

3-4-methylenedioxymethamphetamine; MDMA; polydrug use; adverse effects; drug effects; depression; anxiety; disinhibition; apathy; frontal behavioral syndromes; gender differences

I. INTRODUCTION

Ecstasy (which primarily contains 3,4-Methylenedioxymethamphetamine, or MDMA) is a popular drug among young adults and adolescents, and the lifetime prevalence rates for use among college students is approximately 10–14% (Boyd et al., 2003; Johnston, O'Malley &

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Bachman, 2001;Medina et al., submitted). Ecstasy (MDMA) binds to brain serotonin transporters, which prevents serotonin reuptake and increases serotonin receptor activation, although MDMA can also affect dopamine, noradrenaline, acetylcholine and histamine (Parrott, 2001;Reneman, Booij, et al., 2002). Numerous animal studies have demonstrated MDMA (ecstasy) induced selective serotonin damage in several species, including rats (O'Shea et al., 1998;Scheffel et al., 1992), squirrel monkeys (Hatzidimitriou et al., 1999), rhesus monkeys (Taffe et al., 2001), and baboons (Scheffel et al., 1998). Further, there is evidence to suggest that ecstasy may be a selective serotonin neurotoxin in humans as well (McCann et al., 1994;McCann et al., 1998;McCann et al., 2005;Reneman, Endert, et al., 2002), and females may be at particular risk for ecstasy-induced semi-acute serotonin neurotoxicity (Buchart et al., 2004;Croft, Klugman, et al., 2001;McCann et al., 1994;Reneman et al., 2001).

Serotonin is involved in the regulation of several behavioral domains, including mood, behavioral control, and vigilance (Brown et al., 1979;Coccaro, 1989;Naughton et al., 2000;Verkes et al., 1998). Given ecstasy's potential neurotoxic effects, one may suspect that regular ecstasy use would be associated with increased mood symptoms such as anxiety and depression and executive dysfunction. Indeed, a growing body of evidence supports the hypothesis that ecstasy users have elevated levels of both semi-acute (Curran and Travill, 1997;Curran et al., 2004;Verheyden et al., 2002) and chronic psychiatric symptoms, including dysphoria, depression, anxiety, panic attacks, paranoia, obsessionality, hostility, sleep disturbances, impulse control disorders, and eating disorders (e.g., Parrot and Turner, 2000;Schifano et al., 1998). More specifically, several studies have demonstrated elevated levels of depression (de Win et al., 2004;McCardle et al., 2004;Thomasius et al., 2005) and anxiety (Parrott et al., 2001;Parrott et al., 2000;Thomasius et al., 2005) in ecstasy users compared to controls, although not all studies have identified increased depressive symptomatology (Daumann et al., 2001;Parrott et al., 2001;Parrott et al., 2000). Studies that examined gender differences have reported that, in general, female ecstasy users report greater acute/sub-acute depressive symptoms compared to male users (Milani et al., 2004;Topp et al., 1999;Verheyden et al., 2002).

Studies examining executive function among ecstasy users have typically used laboratory-based neuropsychological measures, with conflicting results. Some have found working memory, fluency, and abstract reasoning deficits among ecstasy users, especially among male users (Montgomery et al., 2005;Verdejo-García et al., 2005;von Geusau et al., 2004), while others found relatively intact executive functioning among ecstasy users after controlling for other drug use (e.g., Croft et al., 2001;Fox et al., 2002;Gouzoulis-Mayfrank et al., 2003;Medina et al., 2005). In addition to this inconsistency in the literature about the presence or absence of executive dysfunction in ecstasy users, little attention has been paid to the possible *behavioral* deficits that interfere with everyday function, most commonly caused by prefrontal system damage. These behaviors include impulsivity, disinhibition, apathy, inattentiveness, indecision, emotional lability, and poor mental flexibility (e.g., Malloy and Grace, 2005). Many of these behaviors are difficult to measure during traditional neuropsychological evaluation, as they are more likely to occur within unstructured or ambiguous contexts. Existing research that has examined behavioral symptoms of executive dysfunction in ecstasy users have found, with few exceptions (McCann et al., 1994), elevations in impulsivity or disinhibition (Gerra et al., 1998;Morgan, 1998;Parrott et al., 2000;Schifano et al., 1998).

One consistent critique of the current studies is that the observed effects may be due to comorbid polydrug use, which is the norm among ecstasy users (e.g., Parrott, 2001). Indeed, it is difficult to determine whether reported psychological symptoms are due to ecstasy use, or rather to other drugs of abuse such as alcohol, marijuana, and cocaine. In support of the latter hypothesis, recent research has found that the relationship between ecstasy and psychological problems

was no longer significant after taking into account other drug use, especially marijuana use (Daumann et al., 2001; Daumann et al., 2004; Morgan et al., 2002; Roiser and Sahakian, 2004; Verdejo-García et al., 2005). A related finding is that Lieb and colleagues (2002) found that ecstasy use is often initiated after the development of an Axis I mood or anxiety disorder. This pattern highlights the need to exclude individuals with independent mood or anxiety disorders that predate their substance use.

Finally, most of the research on the psychological effects of ecstasy abuse is based on comparisons between an ecstasy group, which typically includes individuals with wide variations in usage patterns, and controls. Most studies have not examined exposure effects, although there is preliminary evidence that a positive relationship between *extent* of ecstasy use and increased psychological symptoms exists, in that heavy users demonstrate more impairment compared to light users (Parrott et al., 2000; Roiser and Sahakian, 2004). Therefore, the goal of the present study was to examine the relationship between ecstasy exposure (from none to heavy use) and self-reported executive functioning, anxiety, and depressive symptoms after controlling for frequency of other drug use, gender, and ethnic identification among ecstasy and marijuana users without independent DSM-IV mood or anxiety disorders. It was hypothesized that heavier ecstasy use would be significantly associated with increased levels of psychological symptoms after controlling for other drug use. We also examined gender differences; it was hypothesized that female ecstasy users would demonstrate greater depressive and anxiety symptoms while males would demonstrate increased behavioral symptoms of executive dysfunction.

2. METHODS

2.1 Participants

Data were collected in conjunction with a study that focused on the neuropsychological consequences of ecstasy use; see Medina and colleagues (2005) for more detailed methodology information. Individuals were recruited through advertisements in a free metropolitan newspaper and screened by phone to determine eligibility. Participants were required to be fluent English speakers, 18 years of age or older, and had to fall within one of the predetermined stratified bins of ecstasy exposure (detailed below). Exclusion criteria included mental retardation, major medical or neurologic illnesses including traumatic brain injury, preexisting (independent of substance use) psychiatric conditions, or use of prescribed psychiatric medications.

Axis I psychotic, anxiety, and mood disorders were screened utilizing a modified SCID I/P interview based on DSM-IV-TR criteria (First et al., 2001). Interested participants who had positive responses to the screening questions were discussed in committee; if clear decisions could not be reached then they were re-contacted and administered the appropriate SCID I/P module by a trained interviewer. Individuals who met current diagnostic criteria (independent of their substance use) for any psychotic disorder, bipolar disorder, major depressive disorder, or anxiety disorder (generalized anxiety disorder, panic disorder, and post traumatic stress disorder) were excluded from the study.

Once deemed eligible, participants were screened with regard to their lifetime ecstasy use. In order to ensure that there were adequate participants across the expected range of ecstasy use, we utilized a proportional quota sampling technique, stratified by lifetime ecstasy use. The estimated range of ecstasy use was split into four bins prior to data collection in order to collect data from approximately equal numbers of participants across the full range of ecstasy use, balanced for gender. Further, because polydrug use is the norm among ecstasy users and marijuana use is particularly prevalent in this population, marijuana users were recruited to fill the first bin. This resulted in the following distribution: bin 1 (8 male, 9 female marijuana users

with *no ecstasy use*); bin 2 (9 male, 10 female ecstasy users who used 1–60 lifetime tablets); bin 3 (8 male, 6 female ecstasy users who used 61–200 tablets); and bin 4 (9 male, 6 female ecstasy users who used over 200 tablets). For descriptive purposes, participants in the first bin will be labeled “marijuana-users,” and individuals who used ecstasy at least once will be labeled “ecstasy-users” from this point on. It is important to again emphasize that these bins reflect a sampling strategy only and do not represent separate groups in the multiple regression analysis; rather, the data from all bins were combined to create a *continuous* variable reflecting lifetime ecstasy exposure (from 0–2310 tablets).

Participants were required to remain abstinent from ecstasy and other drugs of abuse for one week. The participant’s self-reported length of abstinence was assessed on two separate occasions during the study session (questionnaire prior to testing and during the Time Line Follow-Back).¹

2.2 Procedure

The Institutional Review Board at the University of Cincinnati approved all aspects of this study. Prior to beginning the study, informed consent was obtained.² Participants completed a brief background questionnaire, psychological questionnaires, and were administered a drug use interview. They also completed a comprehensive neuropsychological evaluation, the results of which have been reported previously (Medina et al., 2005). At the conclusion of the study, participants were paid \$35 and given informational pamphlets and drug and alcohol treatment referrals. A total of 34 men and 31 women were enrolled in the study.

2.3 Frequency of Drug Use

A modified version of the *Time-Line Follow-Back* (Sobell et al., 1979) technique was used, which utilizes memory cues of common holidays and personal events to measure frequency of drug use over the past year (assessed month-by-month). Additionally, a semi-structured interview was administered to measure lifetime drug use. For each substance of use, participants were asked their average weekly use each year that they used. Memory cues appropriate to adolescents and young adults, such as developmental milestones, school grades, and relationship changes, were utilized. The following drug categories were assessed: ecstasy, marijuana, alcohol, sedatives (e.g., downers, ketamine, GHB), stimulants (amphetamine, methamphetamine, cocaine, crack cocaine), hallucinogens (mushrooms, PCP, LSD, peyote), opioids (heroin, opium), and inhalants (nitrous oxide, paint, glue, household cleaners, gas). The participant’s drug use was measured in standard units (tablets for ecstasy; standard drinks for alcohol; joints for marijuana; grams for stimulants; number of hits for inhalants, hallucinogens, and opioids; and pills or hits for sedatives).

2.4 Self-Administered Questionnaire

2.4.1. Anxiety—The *State-Trait Anxiety Inventory* (STAI) measures both temporary ‘state anxiety’ and more chronic, long-term ‘trait anxiety’; both scales assess feelings of apprehension, tension, nervousness, and worry (Spielberger et al., 1983). Norm-based T-scores were utilized for this study.

2.4.2. Depression—The *Beck Depression Inventory- 2nd Edition* (BDI-II) is a 21-item self-report questionnaire that was designed to elicit respondents’ descriptions of their depressive

¹When discrepant reports were given, the shortest length of abstinence was used and re-confirmed later in the interview. Two participants were excluded from the study due to consuming drugs within the past week.

²Participants had the right to waive signing the informed consent document (per the University of Cincinnati IRB’s request), in which case KLM and a research assistant signed the informed consent document as witnesses that informed consent was obtained.

symptoms over the past two weeks (Beck et al., 1993). The BDI-II total score represents an individual's global severity of depressive symptoms. Raw scores were utilized for this study.

2.4.3. Self-Reported Executive Function—The Frontal Systems Behavioral Scale (FrSBe) is a 46-item self-report instrument that measures executive functioning ability in daily life (Grace and Malloy, 1999). (The scale was designed to elicit ratings of behavior both before and after an acute illness or injury. For the purposes of this study, participants filled out only the “after” ratings, reporting on their *current* behavior.) Individuals rate their own behavior on a scale of 1–5 (almost never to almost always). Example items include *Speaks only when spoken to*, *Laughs or cries too easily*, *Swears*, *Is unable to plan ahead*. Responses to the FrSBe comprise three subscales: Apathy, Disinhibition, and Executive Dysfunction. Elevations on the Apathy subscale indicate possible problems with initiation, psychomotor speed, drive, blunted affect, or anhedonia. Elevations on the Disinhibition scale may indicate impulsivity, hyperactivity, social inappropriateness, or antisocial behavior. Scores elevated on the Executive Dysfunction scale may indicate poor complex attention, mental rigidity, poor problem solving, planning deficits, or lack of self-monitoring. Norm-based T-scores were utilized for each scale, on which higher scores denote greater executive dysfunction.

2.5 Data Analysis

Although the relationship between ecstasy use and psychological functioning was examined with a continuous variable reflecting lifetime or past year ecstasy consumption, for *descriptive* purposes a non-parametric statistical analysis (Mann-Whitney U and chi-square tests) was run to test whether the ecstasy and marijuana-users differed demographically. In addition, in order to provide normative data for the groups, the percentage of ecstasy and marijuana-users who demonstrated impairment on the psychological tests were compared and reported (Table 2).

Following bivariate analysis, the primary analyses included two series of multiple regressions that tested whether 1) *past year* or 2) *lifetime* ecstasy exposure was significantly associated with psychological functioning after controlling for gender, ethnicity (which differed between the ecstasy and marijuana-users; see below), and frequency of other drug use besides ecstasy. Further, interactions between ecstasy use and gender were explored. Ordinary least squares (OLS) multiple regressions were utilized in order to examine the unique variance accounted for by ecstasy use above and beyond the other variables included. This analytic strategy also allowed for examination of the dose-dependent relationship between other drug use (e.g., marijuana and alcohol) and psychological functioning. Interpretations about statistical significance were made if $p < .05$.

More specifically, the two series included six separate regressions each (for each dependent variable). For both series, the dependent variables included: STAI-Trait and STAI-State T-scores; BDI-II total raw score; and FrSBe Apathy, Disinhibition, and Executive Dysfunction Subscale T-scores. In the first series, *past year* ecstasy use was the primary independent variable (IV) of interest. In the second series, *lifetime* ecstasy use was primary IV. Independent variables were entered first as a block (standard entry): ecstasy use, gender, ethnicity, and comorbid drug use that may affect psychological functioning (including alcohol, marijuana, sedatives, opioids, stimulants, hallucinogens, and inhalants; frequency of past year use in the first series, and lifetime use in the second series). Interactions between ecstasy use and gender were then entered as a second block. If the interaction term did not significantly add to the regression model, only results from the first block were reported.

3. RESULTS

3.1 Descriptive Information: Demographic, Drug Use, and Psychological Data

3.1.1 Descriptive Information: Demographic Information The ecstasy and marijuana-users ($N=48$ and $N=17$, respectively) did not differ significantly in age ($U=358$, $p<.45$), length of education ($U=333$, $p<.25$), or gender composition [$\chi^2(1)=.25$, $p<.61$]. On average, participants were 23 years old (range 18–35) with an average of 13 years of education (range 9–16). However, the ecstasy and marijuana-users did significantly differ in their ethnic identification [$\chi^2(4)=11.6$, $p<.02$]. Therefore, ethnicity was statistically controlled for in all the regressions. Among the ecstasy-users, 81% were Caucasian, 11% African American, 2% Asian American, and 6% endorsed ‘other.’ In contrast, among the controls 47% were Caucasian, 41% African American, 6% Hispanic, and 6% endorsed ‘other.’

Length of Abstinence The groups did not differ significantly in their average length of abstinence from all drugs, which was 31 days for the marijuana-users ($SD=89$, range=7–378 days) and 15 days for the ecstasy-users ($SD=17$, range=7–117 days) ($U=286$, $p<.07$). For the majority of participants, marijuana was used more recently than the other aforementioned drugs. For the ecstasy users, the average length of abstinence from ecstasy was approximately five months ($M=161$ days, $SD=128$, range=11–491 days).

Frequency of Use As expected, frequency of ecstasy use was significantly different between the ecstasy and marijuana-users. Further, although marijuana and alcohol use did not differ significantly, in general, the ecstasy-users used significantly more drugs at higher frequencies compared to the marijuana-users. See Table 1 for a detailed description of the type and frequency of past year drug use. As stated previously, frequency of drug use of all the aforementioned drug use categories were included in all the regression models. For additional demographic information, including frequency of lifetime drug use, see Medina and colleagues (2005).

3.1.3 Descriptive Information: Psychological Data The ecstasy and marijuana-users did not differ significantly on any of the self-report measures of anxiety, depression, or executive function. (See Table 2 for the normative T-scores on the STAI and FrsBe and raw scores on the BDI-II.) However, as described in detail below, many of the ecstasy and marijuana-users reported clinically elevated levels of state and trait anxiety, depressive symptoms, and apathy, disinhibition, executive dysfunction (planning, organizing, working memory) compared to published norms.

Anxiety Symptoms Fifty-two percent of the ecstasy-users and 47% of the marijuana-users scored 1 standard deviation or more above the mean on the state anxiety scale (STAI-S) [$\chi^2(1)=.00$, $p<.99$]. The majority of both groups (63% ecstasy, 77% marijuana) scored 1 SD above the mean on the trait anxiety scale (STAI-T) [$\chi^2(1)=1.09$, $p<.38$].

Depressive Symptoms Twenty-five percent of the ecstasy-users were in the mildly to moderately depressed range on the BDI-II (scores between 14–28) and 2% were in the severely depressed range (scores between 29–63); while 29% of the marijuana-users were in the mildly to moderately depressed range [$\chi^2(1)=.13$, $p<.75$].

Executive Functioning On the Frontal Systems Behavior Scale (FrSBe), 19% of the ecstasy-users and 29% of the marijuana-users had clinically elevated ($>1SD$ above the mean) Apathy scales [$\chi^2(1)=.84$, $p<.49$]. Twenty-seven percent of the ecstasy-users and 18% of the marijuana-users were clinically elevated on the Disinhibition scale [$\chi^2(1)=.60$, $p<.53$]. Twenty-five

percent of the ecstasy-users and 12% of the marijuana-users were clinically elevated on the Executive Dysfunction scale [$\chi^2(1)=1.30, p<.32$].

3.2 Bivariate Relationships

Frequency of ecstasy use, age, education, gender, or length of drug abstinence did not correlate significantly with any of the psychological variables. Higher levels of alcohol, marijuana, and opioids were related to increased psychological symptoms. Table 3 shows the significant Kendall's tau correlations (Newson, 2001) between the past year and lifetime drug use frequency variables and psychological variables of interest in all participants.

3.3 Primary Results: Multivariate Relationships

To reiterate, two series of multiple regressions were conducted, past year or lifetime ecstasy use were the primary IV's and frequency of other drug use, gender, and ethnicity served as covariates. Further, an interaction between gender and ecstasy use was examined. We found that past year or lifetime ecstasy use was not significantly associated with any of the anxiety, depression, or executive functioning measures after controlling gender, ethnic identification, and frequency of other drug use. Further, no significant relationships were found between ethnicity, gender, or interactions between gender and ecstasy use, and psychological functioning in this sample. Presented below are the significant multivariate predictors.

State Anxiety After controlling for ethnicity and gender, no past year or lifetime drug use variables were significantly associated with state anxiety. *Trait Anxiety.* Higher levels of past year marijuana use [$t(64) = 2.17, \beta = .32, p < .04$] and opioid use [$t(64) = 2.50, \beta = .70, p < .02$] were associated with higher levels of self-reported trait anxiety. Higher levels of lifetime marijuana use [$t(64) = 2.15, \beta = .29, p < .04$] and inhalant use [$t(64) = 2.38, \beta = .32, p < .02$] were associated with increased of self-reported trait anxiety.

3.3.2 Multivariate Relationships: Depression Increased past year marijuana use was significantly associated with higher levels of depressive symptoms [$t(64) = 2.38, \beta = .35, p < .02$]. Higher levels of lifetime opioid use [$t(64) = 2.77, \beta = .47, p < .008$] and lower levels of lifetime hallucinogen use [$t(64) = -2.44, \beta = -.33, p < .02$] were associated with increased levels of self-reported depressive symptoms. A trend between increased lifetime marijuana use and higher levels of depression was observed [$t(64) = 1.83, \beta = .25, p < .07$].

Apathy Subscale Increased past year use of inhalants was associated with higher levels of self-reported apathy [$t(64) = 2.00, \beta = .25, p < .05$]. Increased lifetime use sedatives [$t(64) = 2.13, \beta = .32, p < .04$] was associated with higher levels of self-reported apathy. A trend between increased lifetime inhalant use and higher levels of apathy was also observed [$t(64) = 1.92, \beta = .25, p < .06$].

Disinhibition Subscale Higher past year consumption of alcohol [$t(64) = 2.31, \beta = .30, p < .03$] and opioids [$t(64) = 2.80, \beta = .80, p < .007$] were significantly associated with elevated levels of self-reported disinhibition. Lower past year use of sedatives was associated with elevated levels of disinhibition [$t(64) = -2.23, \beta = -.83, p < .03$]. Higher levels of lifetime alcohol [$t(64) = 2.22, \beta = .28, p < .03$] and opioid use [$t(64) = 2.88, \beta = .50, p < .006$] were also significantly associated with increased levels of self-reported disinhibition.

Executive Dysfunction Subscale Higher past year use of alcohol [$t(64) = 2.24, \beta = .29, p < .02$] and opioids [$t(64) = 2.71, \beta = .74, p < .009$] were significantly associated with greater executive dysfunction. Higher levels of lifetime opioid use was also significantly associated with increased levels of executive dysfunction [$t(64) = 2.68, \beta = .46, p < .01$].

4. DISCUSSION

These results revealed that, although many of the ecstasy-users demonstrated clinically elevated levels of anxiety, depressive, and executive dysfunction symptoms compared to published norms, no *dose-dependent* relationships were observed between ecstasy use and psychological functioning. Rather, higher levels of alcohol, opioids, marijuana, and inhalants were associated with more psychological symptoms in this sample of ecstasy and marijuana polydrug users. Therefore, polydrug use continues to be an important confound in the examination of psychological consequences of ecstasy use and should be thoroughly examined in all ecstasy studies.

Perhaps most notably, the majority of the ecstasy users demonstrated elevated state (52%) and trait (63%) anxiety. However, extent of self-reported trait anxiety was predicted by increased marijuana use, as well as opioid and inhalant use. This is consistent with more recent research indicating that anxiety symptoms observed in ecstasy polydrug users is likely due to comorbid marijuana use (Daumann et al., 2004; Daumann et al., 2001; Morgan et al., 2002). Although relatively understudied, previous research has also found elevated anxiety symptoms among individuals with opioid dependence (Ahmadi et al., 2005; Callaly, et al., 2001) and inhalant abuse (Joe et al., 1991). However, it should be noted that it remains possible that the *simultaneous use* of marijuana (or other substances) and ecstasy are necessary for the development of clinically significant psychological symptoms in ecstasy users (Sala and Braida, 2005; Young et al., 2005).

Although a quarter of the ecstasy users demonstrated mild-to-severe levels of depressive symptoms, after controlling for ethnicity and other drug use, depressive symptoms were best predicted by extent of past year marijuana use and lifetime opioid use. This is consistent with Morgan and colleagues (2002), who found that depressive symptoms among ecstasy polydrug users were best predicted by extent of recent marijuana use. In addition, although de Win and colleagues (2004) demonstrated a significant bivariate correlation between lifetime ecstasy consumption and depressive symptoms, these symptoms did not correlate with serotonin transporter density. Therefore, it is not yet clear that the observed depressive symptoms were the direct result of ecstasy-induced alterations in the serotonergic system. Inhalant use has also been previously associated with increased risk for depression (Rienour, 2005; Sakai et al., 2004). The current finding that increased hallucinogen use was unexpectedly associated with decreased depressive symptoms is consistent with previous research that has demonstrated low risk for depression among individuals with hallucinogen abuse or dependence (Grant, 1995). However, these results should be viewed with caution in the current study as they may be due to multicollinearity in that the simple bivariate relationship between hallucinogens and depressive symptoms was non-significant ($t_b = -.01$).

Approximately one quarter of the ecstasy-users demonstrated clinical elevations on both the FrSBe Disinhibition and Executive Dysfunction subscales. This finding is consistent with previous research showing that ecstasy users demonstrate impulsivity and poor abstract reasoning, working memory, and fluency (Gerra et al., 1998; Montgomery et al., 2005; Morgan, 1998; Parrott et al., 2000; Schifano et al., 1998; Verdejo-García et al., 2005; von Geusau et al., 2004). However, as a group, the ecstasy users did not differ from marijuana users. Further, higher frequency of alcohol and opioid use, but not ecstasy use, was a significant predictor of greater self-reported executive dysfunction and disinhibition. These findings are consistent with previous literature demonstrating significant executive dysfunction associated with alcohol and opioid use (e.g., Selby and Azrin, 1998; Verdejo-García et al., 2004).

Similarly, both ecstasy and marijuana users (19% and 29%, respectively), reported elevated levels of apathy. Frequency of inhalant and sedative use was significantly associated with these

symptoms, which is consistent with previous research demonstrating symptoms of apathy following solvent abuse (Fukui et al., 1992; Goodwin and Hasin, 2002). Less is known regarding the long-term impact of sedative use on symptoms of apathy. Of note, this relationship was not observed at the bivariate level ($t_b=.05$). Therefore, the increased risk of apathy symptoms associated with sedative use should be interpreted with caution, as it may be a result of a complex multivariate relationship in this sample.

We did not find any gender differences in psychological functioning, which is consistent with the report of Fingeret and colleagues (2005). However, these results stand in contrast to previous findings demonstrating increased acute/semi-acute depressive symptoms (Milani et al., 2004; Topp et al., 1999; Verheyden et al., 2002) and increased serotonin neurotoxicity (Buchart et al., 2004; Croft, Klugman, et al., 2001; McCann et al., 1994; Reneman et al., 2001) among female ecstasy users. One potential reason for these discrepant findings is that the current study excluded participants with pre-existing, independent psychological disorders, which occur more frequently in adolescent girls (Garber, Keiley, and Martin, 2002) and women (Kessler et al., 2003). Thus, previous findings that were based on samples that did not exclude individuals who had independent mood disorders may find increased depressive symptoms among female users due to higher population base-rates (e.g., Lieb et al., 2002). Further, the men in this sample demonstrated heavier alcohol, marijuana, and opioid use, which were each significantly associated with depression, anxiety, and executive dysfunction in this sample. Therefore, it is possible that interactions exist between gender and other drug use among ecstasy users, which was not examined in the current sample. Future studies are necessary to determine whether previously reported gender differences are due to ecstasy use, increased comorbid polydrug use, or preexisting, independent mood disorders.

As with any study, there are methodological limitations that need to be considered. One potential weakness of this study is that we did not utilize urinalysis or hair analysis when assessing length of abstinence. Still, attempts were made to maximize the reliability of self-reported drug use, such as guaranteeing confidentiality and privacy by not requiring a signature on the informed consent document. Further, the last date of use was assessed on two separate occasions; once utilizing the *Time Line Follow-Back* technique, which has shown high re-test reliability, high convergent and discriminant validity, and high agreement with patients' urine assays (Fals-Stewart et al., 2000). Another potential weakness is that the ecstasy users were not compared to a non-drug using control group. Thus, the relationship between ecstasy exposure and psychological functioning may have been underestimated in this sample. It should, therefore, be emphasized again that, compared to normative controls, a large percentage of ecstasy users did demonstrate significant psychological impairment, including increased depressive, anxiety, and behavioral symptoms of executive dysfunction.

It should also be noted that results cannot necessarily be generalized to other samples with different lengths of abstinence or different duration of ecstasy use. It is possible that samples with substantially different patterns of ecstasy use would yield different psychological symptoms. For example, the ecstasy users in this study were abstinent from ecstasy for approximately five months; therefore, more sub-acute (*Monday blues*) or short-term psychological effects caused by ecstasy may not have been detected. Further, it is possible that psychological symptoms due to ecstasy use resolve within a few months of abstinence, which has not been sufficiently investigated. Therefore, longitudinal research is necessary to assess how continued ecstasy use or abstinence affects behavioral symptoms of executive dysfunction, anxiety, and depressive symptoms.

Further, because individuals with Axis I mood or anxiety disorders that existed independent of substance use were excluded from the study, other samples of ecstasy users may demonstrate higher levels depressive or anxiety symptoms, although these symptoms may predate their drug

use (Lieb et al., 2002). Still, due to potential serotonin neurotoxicity, ecstasy use may exacerbate these preexisting psychiatric conditions. Further, other moderator variables may put certain subgroups at elevated risk for ecstasy-induced psychological symptoms. For example, individuals with certain genetic polymorphisms in the serotonin transporter gene may be at higher risk for deleterious psychological effects of ecstasy exposure (Roiser et al., 2005).

In conclusion, these results are consistent with previous research indicating that comorbid substance use is predictive of elevated psychological symptoms among ecstasy polydrug users (Daumann et al., 2001; Daumann et al., 2004; Morgan et al., 2002; Roiser and Sahakian, 2004). This study added to the current literature by demonstrating significant dose-dependent relationships between marijuana, alcohol, opioid, and inhalant use and psychological symptoms among ecstasy users. Future studies are necessary to assess the effects of combining ecstasy with other drugs (especially ecstasy+opioids, alcohol, and marijuana), or the effects of ecstasy use on psychological symptoms in individuals with independent, preexisting psychiatric conditions.

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Table 1

Past Year Drug Use (in Standard Units)

Drugs	Ecstasy-Users (n=48)				Marijuana-Users (n=17)				U
	M (N)	Median	SD	Range	M (N)	Median	SD	Range	
Ecstasy	15 (47)	8	23	1–116	0	0	0	0	8.5***
Alcohol	421 (45)	259	446	14–1,836	328 (17)	97	428	2–1,254	339
Marijuana	504 (44)	124	835	1–3,650	280 (17)	274	224	5–728	366
Cocaine	12 (24)	2	25	.01–110	54 (2)	54	70	4–103	244**
Methamphetamine	8 (12)	1	19	.03–67	0	0	0	0	297*
Opioids [±]	21 (10)	5	50	1–164	0	0	0	0	213***
Sedatives [‡]	14 (33)	2	31	1–150	1 (2)	1	0	2	299*
LSD/PCP	4 (14)	3	4	1–18	0	0	0	0	280**
Mushrooms	8 (24)	2	19	1–91	0	0	0	0	204***
Inhalants	24 (12)	11	31	3–104	0	0	0	0	162***

Notes: U = Man-Whitney U tests were run to assess group differences.

Mean frequencies were calculated only for participants who reported using the specified drug *at least one time* during the past year; participants can appear in multiple rows of the tables.

[±]Frequency includes heroin and opioids.

[‡]Frequency includes GHB, ketamine, barbiturates, 'downers,' valium, Xanax, and Ativan.

*
p<.05.

**
p<.01.

p<.001.

Table 2
Mean, Median, SD, and Range of Psychological Variables According to Group

	Ecstasy-Users (n=48)				Marijuana-Users (n=17)				U
	M	SD	Range	Elevated Score [*]	M	SD	Range	Elevated Score [*]	
STAI-S	48	8.9	28–69	52%	46	7.6	36–58	47%	376, ns
STAI-T	53	12.2	34–82	63%	53	7.3	41–63	77%	392, ns
BDI-II	9	8.1	0–37	27%	10	7.7	1–27	29%	389, ns
Apathy [±]	52	15.5	31–109	19%	55	13.6	31–76	29%	335, ns
Disinhibition [±]	56	13.6	29–81	27%	55	11.4	32–76	18%	389, ns
Exec Dysfunction [±]	55	14.7	27–95	25%	56	12.6	42–86	12%	396, ns

Notes: U = Man-Whitney U tests were run to assess group differences.

STAI-S= State Trait Anxiety Inventory- State anxiety T-score.

STAI-T= State Trait Anxiety Inventory- Trait anxiety T-score.

BDI-II= Beck Depression Inventory- 2nd Edition raw score.

[±] Apathy, Disinhibition, and Executive Dysfunction (Exec Dysfunction) are subscales from the Frontal Systems Behavior Scale (FrSBe); normative T-scores are presented.

^{*} Elevated scores denote the percentage of individuals who demonstrated scores that were greater than 1 SD above the normative group's mean on the STAI and FrSBe scales and raw scores >14 on the BDI-II.

Table 3

Bivariate Relationships Between Psychological Variables and Past Year and Lifetime Drug Use (Including Ecstasy).

	STAI-S	STAI-T	BDI-II	Apathy [±]	Disinhibition	Executive Dysfunction
<u>Past Year Use</u>	.06	-.03	-.03	-.11	.02	-.02
Ecstasy	.16	.10	.13	.08	.22**	.23**
Alcohol	.09	.22**	.26**	.16	.11	.18*
Marijuana	.03	.11	.12	.08	.11	.05
Stimulants	-.03	.16	.07	.13	.26**	.22*
Opioids	.08	.12	.03	.05	-.01	.03
Sedatives	.08	.04	.06	.08	.09	.06
Hallucinogens	.11	.09	.10	.17	.01	.09
Inhalants						
<u>Lifetime Use</u>	.06	.02	.05	-.03	.08	.08
Ecstasy	.10	.19*	.20*	.12	.23**	.20*
Alcohol	.09	.17*	.23**	.08	.05	.05
Marijuana	.03	.07	.09	.07	.07	.04
Stimulants	-.02	.12	.05	.14	.19*	.16
Opioids	.02	.07	.04	.02	-.02	.02
Sedatives	-.01	-.01	-.01	.02	.09	.03
Hallucinogens	.08	.10	.08	.18	.13	.10
Inhalants						

Note: Correlations are Kendall's tau.

STAI-S= State Trait Anxiety Inventory- state anxiety T-score.

STAI-T= State Trait Anxiety Inventory- trait anxiety T-score.

BDI-II= Beck Depression Inventory- 2nd Edition.

[±] Apathy, Disinhibition, and Executive Dysfunction are subscales from the Frontal Systems Behavior Scale (FrSBe).

*
p<.05.

**
p<.01.

p<.001.