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# Panic attacks, depression and anxiety symptoms, and substance use behaviors during late adolescence

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**Abstract**

This study examines panic attacks and substance use in a sample of incoming college freshman ($n = 399$) using questionnaires. Panickers ($n = 47$) were significantly more likely than nonpanickers ($n = 290$) to report having ever used sedatives, stimulants, opiods, and other drugs, but not tobacco, alcohol, cocaine, or hallucinogens. Gender and race did not substantially moderate the associations between substance use and panic attacks. Sedative, stimulant, opiod, and other drug use was not associated with panic attack frequency or the occurrence of unexpected attacks. The relationships of anxiety and depression with substance use were larger for panickers than nonpanickers. These results are consistent with the idea that self-medication and symptom exacerbation play a role in the development of co-occurring substance use disorders and mood and anxiety disorders.


*Keywords:* Panic; Adolesence; Substance use; Depression; Anxiety

There is a surprisingly high rate of dual diagnosis of substance use disorders with anxiety and mood disorders, particularly panic disorder (Regier, Rae, Narrow, Kaelber, & Schatzberg, 1998). Explanations for this dual diagnosis often emphasize that many individuals may use psychoactive substances, particularly

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sedatives and alcohol, to reduce negative emotions, such as panic attacks, depression, and anxiety (e.g., Kushner, Sher, & Beitman, 1990). Some explanations for the high dual diagnosis rates also recognize that some substances can precipitate panic attacks or other negative emotions. For example, Breslau and Klein (1999) reported that tobacco use contributes to the subsequent development of panic disorder, presumably through the chronic withdrawal symptoms typically associated with chronic cigarette smoking. Early explanations of dual diagnosis sometimes treated anxiety and mood disorders as primary and substance use disorder as secondary (e.g., Quitkin, Rifkin, Kaplan, & Klein, 1972), or substance use disorders as primary and anxiety or mood disorders as secondary (e.g., Aronson & Craig, 1986). Contemporary theorists (e.g., Blume, Schmaling, & Marlatt, 2000; Kushner, Abrams, & Borchardt, 2000) now view dual diagnosis as related to a mutual influence and interplay between substance use behaviors and psychological symptoms.

The idea that the behaviors and symptoms underlying substance use disorders and anxiety and mood disorders contribute to each other has implications for understanding the development of these disorders. The anxiety and mood disorders most closely associated with substance use disorders are social phobia, panic disorder, and major depressive disorder (Kushner et al., 2000). Social phobia typically begins in adolescence (Regier et al., 1998). Panic disorder often begins in early adulthood (Regier et al., 1998) and many adolescents report having experienced a panic attack (Norton, Cox, & Malan, 1992). Major depressive disorder usually appears to begin in early to middle adulthood (Regier et al., 1998; Sandanger, Nygard, Ingebrigtsen, Sorensen, & Dalgard, 1999), but often begins earlier (Lewinsohn, Gotlib, & Seeley, 1995). Similarly, substance use disorders typically begin in adulthood (Himle & Hill, 1991), but experimentation with different substances typically begins in adolescence and full-blown substance use disorders are prevalent during adolescence (Lewinsohn et al., 1995). Studies of the relationships between substance use behaviors and negative emotions during adolescence (e.g., Wills, Sandy, Shinar, & Yaeger, 1999) are needed to understand the mutual influence between the premorbid and early onset problems with substance use and emotion. These types of studies may help us to better understand the development of the constellation of co-occurring substance use and mood and anxiety disorders.

Given that both substance use disorders and panic disorders often begin after adolescence, it is not surprising that most research on the dual diagnosis of substance use and panic disorders has focused on adult clinical populations (e.g., Page & Andrews, 1996). There is, however, some evidence that substance use behaviors and panic symptoms co-occur during late adolescence (Deacon & Valentiner, 2000). In that prior study of panic attacks and substance use behaviors during late adolescence and early adulthood (Deacon & Valentiner, 2000), we found that individuals who reported having experienced a panic attack were more likely to report having used sedatives, but not other drugs. Although this finding is consistent with expectations based on research with

clinical populations (e.g., Mirin, Weiss, Griffin, & Michael, 1991; Page & Andrews, 1996), the relative paucity of research on panic and substance use during this developmental period makes replication desirable. The purpose of the current study is to replicate the finding from our previous study and to examine several related issues.

In addition to examining whether panic attacks are associated with sedative use during late adolescence, this study will examine the relationship between panic attacks and the use of other substances. The emphasis in the adult literature on the relationship between panic disorder and alcohol use and alcohol use disorders (see Kushner et al., 2000) led us to revise our assessment of alcohol use. In our previous study (Deacon & Valentiner, 2000), we found no relationship of overall alcohol use with panic attacks, anxiety, or depression. In the current study, we assess overall alcohol use and binge drinking using a format similar to the format we use to assess the use of other substances. In addition, recent evidence for an association between tobacco use and panic disorder (Breslau & Klein, 1999) led us to include an assessment of tobacco use.

The idea that panic attacks motivate some individuals to use substances that relieve anxiety and panic led us to examine the hypotheses that panickers more often report sedative use, alcohol use, and binge drinking. The idea that the use of some drugs may lead to panic attacks in nonclinical populations led us to examine the hypotheses that panickers more often report stimulant use, cocaine use, and tobacco use than nonpanickers.

Past studies of tension-reduction expectancies suggested that a self-medication framework may be applicable to men, but not women (Kushner et al., 2000). Accordingly, we explored whether the associations between panic and substance use are moderated by gender. Previous research has not addressed the question of whether these frameworks are equally applicable to different racial groups. This study included a preliminary examination of whether these associations are moderated by race. Finally, this study examined the relationships between substance use behaviors and anxiety and depression. Based on the idea that some individuals, such as those that experience panic attacks, may self-medicate and exacerbate their psychological symptoms through substance use, we hypothesized that these links are especially strong for individuals who have experienced panic attacks.

## 1. Method

### *1.1. Participants*

Study participants ($n = 399$) were college freshman recruited at a mid-sized Midwestern university through introductory psychology, communication, and other courses freshman typically attend, and through postings in freshman

residence halls. Participants received either an entry in a drawing or partial course credit for their participation. Participants completed the study questionnaires in small groups (up to 26 participants). Participants also completed questionnaires for another study (Mounts, in press). Participants were informed that their responses would be kept completely confidential and that they were free to withdraw from the study at any time. The mean age of the sample was 18.4 years (S.D. = 0.8). The sample was 62% women and was composed of 56% Caucasians, 22% African Americans, and 20% participants of other race. Most of the participants (96%) were single. A majority of the sample (73%) indicated that they lived with a roommate assigned to them by University Housing, 14% were living with a friend or friends, 8% with parents, and 5% in another living arrangement. About half the sample (46%) reported that neither parent had received a Bachelor's degree, and about half (54%) reported that at least one parent had received a Bachelor's degree.

### *1.2. Measures*

#### *1.2.1. Demographics*

A demographic questionnaire was constructed for the study to assess participants' age, gender, race, religion, education, marital status, and family income level.

#### *1.2.2. Substance use*

Substance use was assessed using a modified version of the questionnaire used in a previous study (Deacon & Valentiner, 2000). This questionnaire used the drug category sheet in the Structured Clinical Interview for DSM-IV Axis I Disorders, Non-Patient Edition (SCID-I/NP, Version 2.0; First, Spitzer, Gibbon, & Williams, 1996). As in the original questionnaire, participants were asked to answer ''Yes'' or ''No'' as to each of the following questions: (a) ''Have you ever used a sedative, hypnotic, or anxiolytic ('downers'), such as Quaaludes ('ludes'), Seconal ('reds'), Valium, Xanax, Librium, barbiturates, Miltown, Ativan, Dalmane, Halcion, or Restoril?''; (b) ''Have you ever used Cannabis such as marijuana, hashish ('hash'), THC, 'pot,' 'grass,' 'weed,' or 'reefer'?''; (c) ''Have you ever used a stimulant ('uppers') such as Amphetamines, 'speed,' crystal meth, dexadrine, ritalin, diet pills, or 'ice'?''; (d) ''Have you ever used an opioid such as heroine, morphine, opium, Methadone, Darvon, codeine, Percodan, Demerol, or Dilaudid?''; (e) ''Have you ever used Cocaine such as snorting, IV, freebase, crack, or 'speedball'?''; (f) ''Have you ever used a hallucinogen ('psychedelics') such as LSD ('acid'), mescaline, peyote, psilocybin, STP, mushrooms, Extasy or MDMA?''; (g) ''Have you ever used PCP ('angel dust')?''; and (h) ''Have you ever used other drugs such as Steroids, 'Glue,' ethyl chloride, paint, inhalants, nitrous oxide ('laughing gas'), amyl or butyl nitrate ('poppers'), Special K, or nonprescription sleep or diet pills?'' As in the previous study (Deacon & Valentiner, 2000), the PCP category was combined with the ''other drug''

category due to the low frequency of PCP use (seven participants in the entire sample).

The following questions were added with a ''Yes'' or ''No'' response format: (a) ''Have you ever, on one or more occasions, used alcohol?''; (b) ''Have you ever, on one or more occasions, had five alcoholic drinks in one setting?''; and (c) ''Have you ever, on one or more occasions, used any product containing tobacco?'' The questionnaire also asked: ''In the past 30 days, about how often have you used any product containing tobacco?'' with response options of ''Not at all'' (coded as 0), ''Less often than weekly'' (1), ''1–2 days per week'' (2), ''3–4 days per week'' (3), ''5–6 days per week'' (4), and ''Everyday'' (5). A tobacco use (daily) index was defined as having ever used tobacco and having used tobacco daily in the past month.

*1.2.3. Psychological symptoms*

Participants completed a modified version of the Panic Attack Questionnaire (PAQ; Norton, 1989). Panic attacks were defined using the modified description from Brown and Cash (1989) to reduce the number of false positives. To assess panic attack history, participants were asked: ''Have you ever experienced a panic attack at any time in your life?'', with responses measured using a yes/no format. Participants were also asked: ''How many panic attacks have you had in the past year?'' To assess the frequency of panic attacks, participants were asked: ''How many panic attacks have you had in the past four weeks?'' To assess unexpected panic attack history, panickers were asked: ''Have you ever had a panic attack that was unexpected ('out of the blue')?'', with responses measured using a yes/no format. Responses to these four questions were used as indices of panic history, number of panic attacks in the past month, and unexpected panic attacks. The PAQ also assesses the severity of 13 DSM-IV panic symptoms, with responses measured on a five-point Likert-type scale ranging from 0 (did not occur) to 4 (very severe). A total panic symptom severity index was calculated as the sum of the responses to these 13 items. Following previous studies, panickers were defined as participants who reported having a panic attack during the past year that was accompanied by at least four symptoms of moderately severe (i.e., a response of ''2'') or greater intensity (Brown & Cash, 1990; Cox, Endler, Norton, & Swinson, 1991). Nonpanickers were defined as those individuals who reported no history of panic attacks. In order to obtain more ''pure'' groups, participants reporting a history of panic attacks but not meeting the criteria for panic (i.e., limited-symptom panickers) were excluded from all subsequent analyses.

Participants also completed the Beck Anxiety Inventory (BAI; Beck, Epstein, Brown, & Steer, 1988) and the Beck Depression Inventory (BDI; Beck, Ward, Mendleson, Mock, & Erbaugh, 1961). These widely used measures of anxiety and depression symptoms have demonstrated good reliability and validity in past studies (Beck, Steer, & Garbin, 1988; Borden, Peterson, & Jackson, 1991). In the current sample, the BAI and BDI showed internal consistencies of .88 and .85, respectively.

## 2. Results

### 2.1. Panic attacks

A total of 109 participants (27.3% of the sample) reported experiencing a panic attack. Sixty-two participants reported having had a panic attack but did not meet the criteria for panic (15.5% of the sample) and were dropped from all subsequent analyses comparing panickers to nonpanickers. Based on the criteria of having had a panic attack during the past year that was accompanied by at least four symptoms rated as moderately severe or worse, 47 participants (11.8% of the sample) were classified as panickers. Thirty-four (72.3%) of the panickers reported having an unexpected panic attack. Fourteen (29.8%) of the panickers reported having at least one panic attack during the previous 4 weeks. Using chi-square and *t*-tests, no differences were found between panickers and nonpanickers on any demographic variable except gender: 18.7% ($n = 40$) of the women were classified as panickers compared to 5.7% of the men ($\chi^2[1, n = 337] = 11.00$, $p < .01$).

### 2.2. Panic attacks and substance use

Chi-square analyses were used to examine the associations between panic attacks and each type of substance use (see Table 1). These results showed that panickers were significantly more likely to report sedative use, stimulant use, opiod use, and other drug use, compared to nonpanickers. Panickers were not significantly more likely to report tobacco use (ever), tobacco use (daily), alcohol use (ever), alcohol use—five or more drinks, cocaine use, cannabis use, or hallucinogen use.

Table 1
Percentage of nonpanickers and panickers who reported using different substances

| | Nonpanickers ($n = 290$) | Panickers ($n = 47$) | Total ($n = 337$) | Chi-square (*df*) |
|---|---|---|---|---|
| Tobacco (ever) | 53.4 (155) | 66.0 (31) | 55.2 (186) | 2.56 (1) |
| Tobacco (daily) | 16.3 (47) | 23.4 (11) | 17.3 (58) | 1.47 (1) |
| Alcohol | 81.4 (236) | 85.1 (40) | 81.9 (276) | 0.38 (1) |
| Alcohol—five or more drinks | 56.9 (165) | 63.8 (30) | 57.9 (195) | 0.80 (1) |
| Sedatives | 4.8 (14) | 12.8 (6) | 5.9 (20) | 4.57 (1)* |
| Cocaine | 3.4 (10) | 6.4 (3) | 3.9 (13) | 0.94 (1) |
| Cannabis | 44.5 (129) | 55.3 (26) | 46.0 (155) | 1.91 (1) |
| Stimulants | 13.4 (39) | 34.0 (16) | 16.3 (55) | 12.56 (1)** |
| Opioids | 4.5 (13) | 21.3 (10) | 6.8 (23) | 17.94 (1)** |
| Hallucinogens | 14.1 (41) | 23.4 (11) | 15.4 (52) | 2.66 (1) |
| Other drugs | 11.4 (33) | 29.8 (14) | 13.9 (47) | 11.42 (1)** |

*Note*. The number of participants in each group who reporting ever using each type of substance is listed in parentheses.

* $p < .05$.

** $p < .01$.

*2.2.1. Gender as moderator*

We considered whether these associations between panic and substance use variables differed as a function of gender. We tested for gender differences in these associations using loglinear modeling analyses. Only one substance use variable showed a moderation effect: male panickers were significantly more likely to report cocaine use than male nonpanickers (42.9 vs. 6.9%; $\chi^2[1, n = 123] = 10.48$, $p < .01$). Among women, panic was not significantly related to cocaine use (1.1% for nonpanickers vs. 0.0% for panickers; $\chi^2[1, n = 214] = 0.46$, n.s.). The difference in proportions for men was significantly greater than that for women ($\chi^2[1, n = 337] = 3.98$, $p < .05$).

*2.2.2. Race as moderator*

We considered whether these associations between panic and substance use variables differed as a function of race. We tested for race differences in these associations using loglinear modeling analyses. Responses to the race question on the demographic questionnaire was used to form three groups: African Americans ($n = 87$); Caucasians ($n = 231$); and Others ($n = 81$). Only one substance use variable showed a moderation effect: African American panickers ($n = 11$) were significantly more likely to report alcohol use—five or more drinks than African American nonpanickers (54.5 vs. 21.1%; $\chi^2[1, n = 68] = 5.31$, $p < .05$). Among Caucasians and Others, panic was not significantly related to alcohol use—five or more drinks (63.3 vs. 69.3% for Caucasians, respectively, and 54.9 vs. 83.3% for Others, respectively; $\chi^2$s$[1, n$s $= 192$ and $77$, respectively$] = 0.59$ and $1.82$, respectively, both n.s.). The difference in proportions for African Americans was significantly greater than that for Caucasians and Others ($\chi^2\ [2, n = 337] = 6.38$, $p < .05$).

*2.2.3. Panic characteristics and substance use*

Analyses of panickers' data were conducted to examine the associations of the substance use variables with panic characteristics. These analyses were limited to the substance use variables that showed significant relationships with panic attacks. Using *t*-tests, we found no significant difference in the number of panic attacks in the past year between panickers who did and did not report sedative use, stimulant use, opiod use, and other drug use. Similarly, we found no significant differences in panic attack symptom severity between panickers who did and did not report sedative use, stimulant use, opiod use, and other drug use. Using chi-square analyses, we found no significant association of the occurrence of unexpected panic attacks with sedative use, stimulant use, opiod use, and other drug use.

*2.3. Depression and substance use*

A correlation approach using point biserial correlations was used to examine the relationship between depression and the substance use variables. BDI scores

Table 2
Correlations between substance use variables and indices of depression and anxiety for nonpanickers, panickers, and the entire sample, with tests for differences between correlations

| | Beck Depression Inventory | | | Beck Anxiety Inventory | | |
|---|---|---|---|---|---|---|
| | Nonpanickers ($n = 290$) | Panickers ($n = 47$) | Total ($n = 337$) | Nonpanickers ($n = 290$) | Panickers ($n = 47$) | Total ($n = 337$) |
| Tobacco (ever) | .09 | .05 | .10* | .09 | .08 | .14** |
| Tobacco (daily) | .13* | −.17 | .10+ | .18* | .12 | .16** |
| Alcohol | .02 | −.01 | .04 | −.03 | .02 | .03 |
| Alcohol—five or more drinks | .15* | .27+ | .17** | .08 | .16 | .13** |
| Sedatives | .05 | .33* | .17** | .01 | .08 | .12* |
| Cocaine | .11+,a | .50**,a | .28* | −.00b | .37*,b | .17** |
| Cannabis | .16** | .14 | .16** | .02 | .11 | .10+ |
| Stimulants | .15** | .14 | .20** | .10 | .11 | .21** |
| Opioids | .08c | .40**,c | .28** | .09 | .25+ | .27** |
| Hallucinogens | .11+ | .24 | .18** | .06 | .13 | .16** |
| Other drugs | .11+ | .19 | .15** | .05 | .28+ | .20** |

*Note*. Correlations with the same superscript are significantly different at the $p < .05$ level.
+ $p < .10$.
* $p < .05$.
** $p < .01$.

were not significantly related to tobacco use (daily) and alcohol use (ever). BDI scores were significantly related to tobacco use (ever), alcohol use—five or more drinks, sedative use, cocaine use, cannabis use, stimulant use, opiod use, hallucinogen use, and other drug use. These correlations are presented in Table 2.

### 2.3.1. Panic as moderator

We also examined whether there were significant differences between nonpanickers and panickers in the magnitude of these associations of drug use variables and the BDI. These correlations are also presented in Table 2. There were no significant group differences in the magnitude of the associations of the BDI with the tobacco use (ever), tobacco use (daily), alcohol use (ever), alcohol use—five or more drinks, sedative use, cannabis use, stimulant use, hallucinogen use, and other drug use. The association between BDI and cocaine use was significantly larger ($z = 2.73$, $p < .01$) for the panicker group than for the nonpanickers. The association between BDI and opiod use was also significantly larger ($z = 2.73$, $p < .01$) for the panicker group than for the nonpanickers.

## 2.4. Anxiety and substance use

Also using point biserial correlations, BAI scores were not significantly related to alcohol use (ever) and cannabis use. BAI scores were significantly related to tobacco use (ever), tobacco use (daily), alcohol use—five or more drinks, sedative

use, cocaine use, stimulant use, opiod use, hallucinogen use, and other drug use. These correlations are presented in Table 2.

*2.4.1. Panic as moderator*

We also examined whether there were significant differences between nonpanickers and panickers in the magnitude of these associations of drug use variables and the BAI. These correlations are also presented in Table 2. There were no significant group differences in the magnitude of the associations of the BAI with the tobacco use (ever), tobacco use (daily), alcohol use (ever), alcohol use—five or more drinks, sedative use, cannabis use, stimulant use, opiod use, hallucinogen use, and other drug use. The association between BAI and cocaine use was significantly larger ($z = 2.38$, $p < .05$) for the panicker group than for the nonpanickers.

## 3. Discussion

This study found that panickers were more likely than nonpanickers to report having used sedatives, stimulants, opiods, and other drugs. Although they comprised only 13.9% of those studied, panickers accounted for 30.0% of participants who reported sedative use, 29.1% of those who reported stimulant use, 43.5% of those who reported opiod use, 29.8% of those who reported the use of other drugs. In addition, this study found that these associations were not substantially moderated by gender or race. Finally, this study found evidence that substance use behaviors and psychological symptoms had particularly close relationships among panickers. These results are consistent with the idea that subclinical substance use behaviors and negative emotion symptoms contribute to each other during the developmental period prior to the onset of many substance use disorders and anxiety and mood disorders.

The association between panic attacks and sedative use found in the current study replicates the main finding from Deacon and Valentiner (2000). The interpretation that panic attacks are associated with sedative use is consistent with findings from research with clinical samples (Mirin et al., 1991; Page & Andrews, 1996; Quitkin et al., 1972).

The current study found associations between panic attacks and other substance use variables, associations not found in Deacon and Valentiner (2000). Data in that previous study, however, show nonsignificant trends that are similar to the findings from the current study. Given the larger sample size in the current study, the null findings from the Deacon and Valentiner (2000) study may be due to Type II errors. The finding of an association between panic attacks and stimulant use is consistent with the physical effects of stimulants (Shear, 1986). A wide variety of substances, usually stimulants, have been used to induce panic attacks in the laboratory (Shear, 1986). It appears that any substance that can produce physical sensations gives individuals an opportunity to experience the

catastrophic thoughts believed to produce panic attacks. The current study's findings that the use of stimulant, opiods, other drugs are associated with panic attacks are consistent with the prevailing cognitive conceptualization of panic attacks and panic disorder (McNally, 1990).

There was some evidence that gender may moderate the association between cocaine use and panic attacks, such that cocaine may be associated with panic among men, but not women. Similarly, there was some evidence that race may moderate the association between binge drinking and panic attacks, such that binge drinking may be associated with panic among African Americans, but not among Caucasians and those in the other race group. Future research on the moderating roles of gender and race might examine these relationships. Considering, however, the exploratory nature of these analyses and that only 2 of the 20 analyses showed significant moderation effects, the main conclusion that should be taken from these analyses is that gender and race did not appear to substantially moderate the relationships between panic and the substance use variables. Similarly, Costello, Erkanli, Federman, and Angold (1999) reported that gender did not substantially moderate the course of substance use disorders or the associations of substance use with depression during adolescence.

These data also revealed an unusually high association between depression and cocaine use among panickers, but not among nonpanickers. Among panickers, having ever used cocaine accounted for 25.0% of the variance in depressive symptoms. We also found stronger relationships for panickers than for nonpanickers between anxiety and cocaine use, and between depression and opiod use. These findings suggest a close link between substance use behaviors and psychological functioning for individuals who have panic attacks. We note that we found no significant link between substance use behaviors and general indices of anxiety and depression among panickers in a previous study (Deacon & Valentiner, 2000). An inspection of those data does show a similar pattern of results to those reported here. Specificially, the magnitude of the nonsignificant correlations for the panickers were generally larger than those for the nonpanickers in that earlier data set. Although there is some inconsistency between the previous and present studies in the results of the significance tests, the overall pattern suggests that there are somewhat stronger associations between substance use behaviors and general anxiety and depression for panickers than for nonpanickers.

These relationships between psychological symptoms and substance use behaviors suggests that the pattern of dual diagnosis found in clinical populations is evident in the period prior to when these disorders fully develop. This observation is consistent with the idea that the high rates of dual diagnosis may be due to substance use disorders and anxiety and mood disorders emerging together, rather than one disorder causing the other. One possibility that is not ruled out by the results of the current study is that psychological symptoms and substance use behaviors are signs of a common underlying pathology. Another

possibility is that psychological symptoms and substance use behaviors contribute to each other. If this second view is correct, then dual diagnoses (i.e., co-occurring substance use disorders and anxiety and mood disorders) may be better conceptualized as distinct from both substance use disorders and anxiety and mood disorders. It is possible that emotional disturbance and substance use behaviors in individuals with dual diagnoses have etiological factors that do not operate when only an emotional or only a substance use disorder is present. Consistent with this view, the results of genetic epidemiological research (Rende & Weissman, 1999) suggest that anxiety and mood disturbance is central to the family transmission of one type of substance misuse.

Contrary to previous research documenting the positive relationship between cocaine use and panic attacks (e.g., Anthony, Allen, & Petronis, 1989), panickers overall were not more likely than nonpanickers to report having used cocaine. In contrast with the literature indicating a relationship between panic disorder and alcoholism (e.g., Himle & Hill, 1991), this study did not find an overall association of alcohol use and binge drinking with panic. We note, however, that the amount of alcohol and cocaine use in the current sample was relatively low in comparison with those included in previous studies. This low level of alcohol use was probably due, at least in part, to our use of a sample of individuals that were too young to legally obtain alcohol. The relationship of panic with alcohol and cocaine use may only be apparent at high levels of the substance use behaviors, levels at which the effects of chronic use and withdrawal begin to operate (George, Nutt, Dwyer, & Linnoila, 1990). In any event, the current study provides no overall evidence that alcohol and cocaine use are associated with panic attacks.

The main conclusion from this study is that the relationship between panic attacks and substance use appears to be present prior to typical age of onset of substance use and panic disorders, and that psychological symptoms appear to be closely related to substance use behaviors for individuals who report having had a panic attack. There are, however, several important limitations associated with this study. As with the Deacon and Valentiner (2000) study, this study relied upon the use of self-report, pencil-and-paper questionnaires. The approach is problematic for the assessment of both panic attacks (Brown & Cash, 1989) and substance use behaviors (Sobell & Sobell, 1990). While method variance and other method related explanations cannot be ruled out, we note that the consistency of the these findings with research on clinical populations increases our confidence that they are not due entirely to method artifact. Another limitation that affects both the current study and the previous study (i.e., Deacon & Valentiner, 2000) is that no temporal sequence between substance use behaviors and negative emotions has been established. Longitudinal and experimental research is needed to give a more complete and convincing body of evidence for the notion that disorders of substance use and negative emotions develop through a process of mutual influence of behaviors and symptoms.

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