

# SUBSTANCE USE AND NON-CLINICAL PANIC ATTACKS IN A YOUNG ADULT SAMPLE

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**ABSTRACT:** *This study examines panic attacks and substance use in a non-clinical, young adult sample. Two hundred seventy-nine college students completed questionnaires that assessed non-clinical panic attacks, alcohol and drug use behavior, and anxiety and depression symptoms. Non-clinical panickers (n = 25) were significantly more likely than non-panickers (N = 222) to report using sedatives, but not alcohol, cocaine, stimulants, or other drugs. Among non-clinical panickers, sedative use was not associated with distress about panic attacks, panic attack frequency, the occurrence of unexpected attacks, or general anxiety or depression symptoms. Coping-motivated alcohol use, though not associated with non-clinical panic, was significantly associated with anxiety and depression symptoms. These results are discussed in terms of theories of the co-morbidity between substance use and panic disorder.*

Several explanations for the high co-morbidity between panic disorder and substance use disorders have been offered (e.g., Aronson and Craig, 1986; Kushner et al., 1990). Although substance use disorders typically start in adulthood, many individuals begin using alcohol and drugs during adolescence (Lewinsohn et al., 1995). Similarly, panic disorder often begins in young adulthood, but many adolescents report having experienced a panic attack (Norton et al., 1992). Research on the co-morbidity of substance use and panic disorder, however, has focused on adult clinical populations (Cox et al., 1989; Norton et al., 1989; Page and Andrews, 1996). Studies that examine the co-occurrence of substance use behavior and panic symptoms among non-clinical adolescents and young adults may shed light on explanations of the co-morbidity of these disorders and help us better understand how these disorders develop.

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Tension-reduction theory suggests that emotional problems may lead to substance use because individuals use drugs to alleviate their anxiety (Kushner et al., 1990). Consistent with this model, individuals with panic disorder have been found to prefer substances believed to decrease anxiety and panic, such as alcohol and sedatives, over other types of substances (Mirin et al., 1991; Page and Andrews, 1996; Quitkin et al., 1972). Several researchers have reported that most of their chemically dependent participants who experienced panic attacks reported using alcohol to self-medicate their anxiety symptoms (Bibb and Chambless, 1986; Cox et al., 1989). In addition, alcohol consumption in individuals with panic disorder has been shown to attenuate panic and anxiety symptoms following laboratory panic-challenges (Kushner et al., 1996). Studies of non-clinical samples have used tension-reduction expectancy (Kushner et al., 1994) and coping motivation (Cooper et al., 1995) models to predict alcohol consumption. These findings support the view that panic, anxiety, and mood symptoms contribute to substance use, and thus may play a causal role in the development of substance use disorders.

There are several findings, however, that suggest that substance use may lead to the development of panic disorder. A substantial portion of individuals with co-morbid substance use and panic disorders report that their substance use disorder began before their panic disorder (Kushner et al., 1990). In laboratory studies, researchers have directly triggered panic attacks using sodium lactate, caffeine, carbon dioxide, and other substances (Cox et al., 1990; Roy-Byrne and Udhe, 1988). In support of the notion that substance use may induce panic attacks, cocaine abusers have been found to be at an increased risk of panic disorder (Anthony et al., 1989). Other studies have reported that cocaine can precipitate both panic attacks and panic disorder (Aronson and Craig, 1986; Louie et al., 1989). This body of research suggests that many drugs, especially stimulants and cocaine, may induce panic attacks.

In their literature review, Norton et al. (1992) reported that panic attacks are a surprisingly common experience, with about 20 percent of non-clinical samples (including adolescents) reporting having experienced a panic attack. Studies of non-clinical panic, which are often viewed as an opportunity to better understand panic disorder and its development, have identified several characteristics of panic attacks which may contribute to clinically significant impairment. These characteristics include the frequency of panic attacks, the occurrence of unexpected attacks, and worry or distress about panic attacks and their consequences. Regarding the issue of co-morbidity, it is not clear which features of panic attacks or panic disorder are most closely related to substance use disorders and substance use behavior.

The purpose of this study is to investigate the associations between panic attack and drug use variables in a non-clinical, young adult sample. Based on the idea that non-clinical panic attacks motivate some individuals to use substances that relieve anxiety and panic, this study examined whether non-clinical panickers reported more sedative use, alcohol use, and coping-motivated alcohol use than non-panickers. Based on the idea that the use of some drugs may lead to panic attacks in non-clinical populations, this study examined whether non-clinical panickers reported more stimulant and cocaine use than non-panickers. In addition, this study investigated the links between the use of various types of substances and different characteristics associated with panic attacks, including panic attack frequency, the occurrence of unexpected attacks, panic-related distress, and general anxiety and depression symptoms.

## METHOD

### Participants

Study participants ( $N = 279$ ) were recruited from introductory psychology classes at a mid-sized Midwestern university and received partial course credit for their participation. Participants completed the study questionnaires in groups ranging from 1 to 27 participants ( $M = 12.4$ ,  $SD = 9.0$ ). Participants signed consent forms prior to data collection and were informed that their responses would be kept completely confidential and that they were free to withdraw from the experiment at any time without penalty. The mean age of the sample was 19.7 years ( $SD = 1.9$ ). The sample was 52.0 percent male and was composed of 55.9 percent Caucasians, 22.6 percent African Americans, and 21.5 percent participants of other ethnicity. Most of the participants (98.2%) had never been married. The median family income of the sample was between \$45,001 and \$50,000 per year.

### Measures

#### DEMOGRAPHICS

A demographic questionnaire was constructed for the study to assess participants' age, gender, ethnicity, marital status, family income level, and whether they had ever been to see a psychologist, therapist, or other mental health professional.

#### ALCOHOL USE

Alcohol use was assessed in two ways. First, a quantity-frequency approach that consisted of four questions was used. One question assessed alcohol use frequency during the past 30 days, one assessed alcohol use quantity during the past 30 days, one assessed alcohol use frequency during the past year, and one assessed alcohol use quantity during the past year. A 30-day alcohol use index was calculated as the product of the two 30-day questions, and a past-year index as the product of the two past-year questions. An alcohol use composite was calculated as the sum of the standardized scores on the 30-day and past-year indices. The questions and the method for constructing the alcohol use index were taken from Kushner and Sher (1996).

Alcohol use was also assessed using the Drinking Motives Questionnaire (DMQ; Cooper et al., 1992). The DMQ asks respondents to rate the relative frequency of their alcohol use for each of 15 indicated reasons. The DMQ consists of three five-item scales: coping motives, enhancement motives, and social motives. Coping motives refer to drinking to reduce or avoid negative affective states such as anxiety or depression. Enhancement motives refer to drinking to increase or maintain positive affective states. Social motives refer to drinking to achieve social goals such as peer approval or feelings of affiliation. Scale scores were calculated by summing scores across the five items on each scale. The DMQ has been shown to possess adequate psychometric properties (Cooper, 1994; Cooper et al., 1992; Stewart et al., 1996). In the present study, the internal consistencies of the coping motives, enhancement motives, and social motives scales were 0.79, 0.88, 0.77, respectively.

## **DRUG USE**

A drug use questionnaire was created for the current study using 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 et al., 1996). Participants were asked to answer "Yes" or "No" 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 heroin, 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 non-prescription sleep or diet pills?". Due to the low frequency of endorsement of the use of PCP in the current sample, this category was combined with the "other drugs" category.

## **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. Following past versions of the PAQ (e.g., Brown and Cash, 1989; Norton, 1989), the definition of panic attacks was followed by several questions. To assess panic attack history, participants were asked: "Have you ever experienced a panic attack at any time in your life?" and responses were measured using a yes/no format. To assess the frequency of panic attacks, participants were asked: "How many panic attacks have you had in the past year?" and "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')?" and responses were measured using a yes/no format. To assess distress about panic attacks, panickers were asked: "How disturbing or distressing are your panic attacks?" and responses were measured using a five-point Likert-type scale ranging from "no concern at all" (coded as "0") to "I get extremely anxious" (coded as "4"). To assess panic attack symptoms, participants were asked to rate the severity of 13 panic attack symptoms taken from *DSM-IV* (American Psychiatric Association, 1994) during their worst panic attack in the past year. Following previous studies, non-clinical 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 or greater intensity (Brown and Cash, 1990; Cox et al., 1991). Non-panickers were defined as those individuals who reported no history of panic attacks. Following past research (see Norton et al., 1992) participants reporting a history of panic attacks but not meeting the criteria for non-clinical panic (i.e., limited-symptom panickers) were excluded from all subsequent analyses.

Participants also completed the Beck Anxiety Inventory (BAI; Beck et al., 1988a; Beck et al., 1988b) and the Beck Depression Inventory (BDI; Beck et al., 1961). These widely used measures of anxiety and depression symptoms have demonstrated good reliability and validity in past studies (Beck et al., 1988a,b; Borden et al., 1991).

## RESULTS

A total of 57 participants (20.4% of the sample) reported experiencing a panic attack. 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, 25 participants (9.0% of the sample) were classified as non-clinical panickers. Thirty-two participants who reported having had a panic attack but who did not meet the criteria for non-clinical panic (11.5% of the sample) were dropped from subsequent analyses comparing non-clinical panickers to non-panickers. Eighteen of the 25 non-clinical panickers reported having an unexpected panic attack. The number of panic attacks reported in the past year ranged from 1 to 50 ( $M = 8.4$ ,  $SD = 13.6$ ), with 14 non-clinical panickers reported having at least one panic attack during the previous 4 weeks. Using  $\chi^2$  and  $t$ -tests, no differences were found between non-clinical panickers and non-panickers on any demographic variable except gender: 76.0 percent of non-clinical panickers were women ( $N = 19$ ) compared to 45.9 percent of non-panickers ( $N = 102$ ;  $\chi^2 [1, N = 247] = 8.12$ ,  $p < .05$ ).

To examine the association between alcohol use and panic attacks,  $t$ -tests were used to compare the alcohol use composite and drinking motives scale scores of non-panickers and non-clinical panickers. Non-clinical panickers and non-panickers did not significantly differ in their scores on the alcohol use composite. For example, each group reported both “getting drunk” and consuming “five or more drinks at a single sitting” approximately twice in the past month. Non-clinical panic was not significantly related to any of the DMQ scales. Non-clinical panickers did not score significantly higher on the coping motives scale (overall  $M = 3.66$ ,  $SD = 3.15$ ), and all participants reported drinking for enhancement motives (overall  $M = 5.64$ ,  $SD = 4.04$ ) and, particularly, social motives (overall  $M = 6.17$ ,  $SD = 3.41$ ) more often than coping motives. We also examined the associations of anxiety and depression with the alcohol use composite and each DMQ scale. Coping-motivated alcohol use demonstrated significant relationships with BAI scores ( $r = .21$ ,  $p < .01$ ) and BDI scores ( $r = .17$ ,  $p < .01$ ). Anxiety and depression symptoms were not associated with drinking for social or enhancement motives (all  $p > .10$ ).

To examine the associations between non-clinical panic attacks and each type of drug use,  $\chi^2$  statistics were used. Table 1 displays the frequencies associated with these analyses. These results show that non-clinical panickers were significantly more likely to report having used sedatives ( $\chi^2 [1, N = 247] = 14.00$ ,  $p < .01$ ) than non-panickers. Non-clinical panic was not significantly associated with any other type of substance use. To examine the possibility that the association between non-clinical panic and sedative use was due to professional interventions involving prescription medications, we repeated the  $\chi^2$  analyses for sedative use excluding participants who reported ever having sought help from a mental health professional ( $n = 51$ ) or who did not answer this item. In this subsample ( $n = 196$ ), non-clinical panic was again significantly associated with

**TABLE 1**  
Percentage of Non-Panickers and Non-Clinical Panickers Who Reported Ever Using Different Drugs

	<i>Non-panickers</i> ( <i>n</i> = 222)	<i>Non-clinical</i> <i>panickers</i> ( <i>n</i> = 25)	<i>Total</i> ( <i>N</i> = 279)
Sedatives**	4.5% (10)	24.0% (6)	6.5% (16)
Cocaine	7.2% (16)	16.0% (4)	8.1% (20)
Cannabis	51.6% (114)	48.0% (12)	51.2% (126)
Stimulants	15.4% (34)	20.0% (5)	15.9% (39)
Opioids	15.8% (35)	20.0% (5)	16.3% (40)
Hallucinogens	19.0% (42)	28.0% (7)	19.9% (49)
Other drugs	17.6% (39)	32.0% (8)	19.1% (47)

*Note.* \*\* $p < .01$ . The number of participants in each group who reported ever using a type of drug is listed in the parentheses.

sedative use (rate of sedative use among non-clinical panickers = 17.6% [ $n = 3$ ], among non-panickers = 3.9% [ $n = 7$ ],  $\chi^2$  [1,  $N = 196$ ] = 6.00,  $p < .05$ ).

Analyses of non-clinical panickers' data were conducted to examine the associations of sedative use with panic-related variables, anxiety, and depression. Using  $t$ -tests, we found no significant difference in the number of panic attacks in the past year between non-clinical panickers who reported sedative use and those who reported no sedative use. Using  $\chi^2$  analyses, we found no significant association between the occurrence of unexpected panic attacks and sedative use. A  $\chi^2$  test was used to examine differences in the prevalence of sedative use among frequent panickers (non-clinical panickers reporting having had a panic attack during the past 4 weeks) and infrequent panickers (non-clinical panickers not reporting having had a panic attack during the past 4 weeks). Four out of 14 (28.6%) frequent panickers and two out of 11 infrequent panickers (18.2%) reported using sedatives, yielding a non-significant  $\chi^2$  ( $p > .10$ ). A  $t$ -test was used to examine whether sedative use was associated with anxiety, depression, and distress about having panic attacks. Non-clinical panickers who used sedatives did not report significantly greater distress about their panic attacks, more anxiety symptoms, or more depression symptoms than non-clinical panickers who had not used sedatives.

## DISCUSSION

This study found that non-clinical panickers were more likely than non-panickers to report having used sedatives, but not other drugs. Although they comprised only nine percent of the overall sample, non-clinical panickers accounted for 37.5 percent of participants who reported sedative use. The similarity between this study's findings and those using clinical populations (Mirin et al., 1991; Page and Andrews, 1996; Quitkin et al., 1972) suggests that the relationship between panic attacks and sedative use is present prior to typical age of onset of clinical disorders.

Consistent with previous research showing that individuals with panic and substance use disorders prefer sedatives (Mirin et al., 1991; Page and Andrews, 1996; Quitkin et al., 1972), this study found a significant relationship between sedative use and non-clinical panic attacks. Among non-clinical panickers, sedative users did not report higher levels of

distress about panic attacks, greater panic attack frequency, more unexpected panic attacks, or higher levels of anxiety and depression symptoms than non-clinical panickers who had not used sedatives. These results suggest that non-clinical panic attacks alone may lead to sedative use behavior. It is important to note, however, that the current study did not examine the time frames associated with panic attacks and drug use. In addition, the low power resulting from the small sample size may have obscured less robust but still meaningful relationships between sedative use and characteristics of panic attacks (e.g., panic attack frequency). Future research might investigate whether non-clinical panic attacks precede the use of sedatives and whether cognitive models (e.g., Cooper et al., 1995) apply to sedative use among panickers.

Contrary to previous research documenting the positive relationship between cocaine use and panic attacks (e.g., Anthony et al., 1989), non-clinical panickers were not more likely than non-panickers to report having used cocaine. In addition, non-clinical panic was not significantly associated with the use of stimulants, opioids, cannabis, hallucinogens, or other drugs. Despite the low power associated with the small sample size and the statistical non-significance of these relationships, trends suggesting higher rates of drug use among non-clinical panickers were evident for all drugs except for cannabis. However, only sedative use was robustly related to non-clinical panic. These results suggest that the use of cocaine, stimulants, and other drugs are probably not inducing many panic attacks in non-clinical populations.

In contrast with the literature indicating a relationship between panic disorder and alcoholism (e.g., Himle and Hill, 1991), this study did not find an association between alcohol use, drinking motives, and non-clinical panic. Some researchers have hypothesized that a “kindling” process may occur where the long-term use of specific substances may trigger panic attacks and subsequent panic disorder even after substance use is discontinued (Louie et al., 1989). For example, George et al. (1990) suggested that the chemical and cognitive effects of repeated alcohol withdrawal might produce a heightened state of anxiety that gives rise to panic attacks in susceptible individuals, even during sobriety. The use of a young adult sample probably excluded severely alcohol dependent individuals whose repeated episodes of alcohol withdrawal may have kindled panic attacks. In addition to kindling, there may be other effects of alcohol use (e.g., negative psychosocial consequences) that may contribute to anxiety and panic (Kushner et al., 1990; Norton et al., 1989). Furthermore, the relatively low rate of alcohol use in the current study may have reduced this study’s power to examine relationships with alcohol use. Finally, alcohol use among individuals with panic disorder may be motivated by symptoms that are not present among non-clinical panickers (e.g., severe distress). The current findings provide no evidence that alcohol use is associated with non-clinical panic attacks, suggesting that theories of the co-morbidity of alcoholism and panic disorder should highlight the difference between sub-clinical and clinically significant symptoms and behaviors.

It is surprising that non-clinical panickers, a group traditionally characterized by higher levels of anxiety than non-panickers, did not report drinking to cope more often than less anxious individuals. Despite the null findings regarding alcohol use and non-clinical panic, the present study did find an association between anxiety and depression symptoms and coping-motivated alcohol use. This is consistent with hypothesized models of alcohol use that suggest anxious and depressed individuals use alcohol as a strategy to regulate their emotions (Cooper et al., 1995). The results of this study suggest that future researchers

may want to continue to explore cognitive variables to clarify the reasons anxious individuals use alcohol.

It should be pointed out that we found an association between *reports* of panic attacks and *reports* of substance use. This study used a strict description of panic attacks to reduce the number of false positives (Brown and Cash, 1989) and a conservative definition of non-clinical panickers (Brown and Cash, 1990; Cox et al., 1991). However, the use of self-report measures, while allowing for an economical design, suggests that some caution be taken in interpreting these results. It is likely that the association between reports of panic attacks and substance use may be influenced by method variance. Concern about possible method variance is partially addressed by the specificity of our findings: associations were found for sedative use, but not for alcohol consumption or the use of other types of drugs. The most tenable explanations of the current results are that non-clinical panickers may use sedatives to ameliorate their panic attacks.

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