

An Examination of the Phenomena of Panic and Nonpanic  
Anxiety in a Student Sample

by

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## ABSTRACT

There is significant debate in the literature on anxiety as to whether panic attacks and trait anxiety are qualitatively distinct or mere variations of anxiety intensity. Using a student sample, the present research attempts to investigate differences between subjects with panic attacks and subjects without panic attacks on a variety of self-report measures related to anxiety phenomena. Panickers were found to be more anxious, to have more cognitive and somatic symptoms of anxiety, to have more catastrophic cognitions about their anxiety, to be more fearful of experiencing anxiety, to be less rational, and to be more subjectively cognitively impaired. However, when panickers were compared to nonpanickers with an equal amount of trait anxiety, they were only found to have more severe somatic symptoms of anxiety and to be more fearful of being anxious. The obtained results are supportive of a distinction between the two types of anxiety and the nature of this distinction is discussed. The results are discussed in relation to the contemporary taxonomy of anxiety states, the cognitive model of panic attack maintenance and/or etiology, and methodological issues in panic research. Additionally, comment is made on the utility of nonclinical panic research as an analogue of related clinical states.

## DEDICATION

This thesis is dedicated to my grandparents, John and Catherine Munro. Thank you for giving me the encouragement to pursue education and the self-esteem to persevere.

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**CHAPTER I**  
**INTRODUCTION**

A Brief Context

One of the specified mandates of the American Psychiatric Association (A.P.A.) has been to produce and publish a classification system of clinical mental disorders. This publication has become known as the Diagnostic and Statistical Manual of Mental Disorders, which is presently in its third edition revised (DSM-III-R; A.P.A., 1987). In order to revise diagnostic criteria for successive editions, the decision was made to base changes primarily upon "the presence of empirical support from well conducted research ..." (A.P.A., 1987, p. xxi). This focus has resulted in several changes to the classification system, yielding greater diagnostic reliability and increased taxonomical validity with each successive edition (A.P.A., 1987).

One cluster of mental disorders that has undergone significant diagnostic modification is that of the Anxiety Disorders. Notable changes have included the separation of Panic Disorder from Generalized Anxiety Disorder in DSM-III (A.P.A., 1980) and the subsuming of Agoraphobia With Panic Attacks under the more general rubric of Panic Disorder in DSM-III-R (A.P.A., 1987). These respective changes were based in part on the research findings that: 1) patients

with panic attacks respond differentially to anti-depressant and anxiolytic medications compared to non-panicking anxiety patients (Zitrin, Klein, & Woerner, 1978); and 2) that Agoraphobic patients are not significantly distinct symptomatically from Panic Disorder patients (A.P.A., 1987).

The differentiation of Panic Disorder from Generalized Anxiety Disorder has proved controversial. The debate centers upon whether the anxiety symptomatology evident in Panic Disorder and Generalized Anxiety Disorder is qualitatively distinct, or simply quantitatively variant. In other words, is a panic attack different than intense anxiety?

Although the diagnostic separation was initiated by pharmacological research, the separation has spawned a great deal of psychological research. This psychological research has been primarily directed towards determining personality and cognitive factors associated with the clinical presentations of the two disorders (e.g. Barlow, Vermilyea, Blanchard, Vermilyea, Di Nardo, & Cerny, 1985). Attempts have also been made to systematically investigate possible etiologically significant psychological factors (e.g. Clark, 1986).

Also pertinent to the present study has been the increasing use of nonclinical samples in the investigation of panic and nonpanic anxiety and their associated clinical phenomena (e.g. Norton, Harrison, Hauch, & Rhodes, 1985; Norton, Dorward, & Cox, 1986; Telch, Lucas, & Nelson, 1989).

Subjects drawn from nonclinical samples (e.g., university students) who indicate that they have experienced panic attacks commonly have been termed as "nonclinical panickers." However, the term "nonclinical" may be a bit of a misnomer in that not all studies have assessed whether, or not, their subjects were receiving treatment. Considering that the obtained estimates of panic prevalence in nonclinical samples is much higher than estimates obtained with clinical samples, it would appear that the non-clinical panic phenomenon is distinct and the label is appropriate.

Within the last 10 years there has also been a realization that panic attacks meeting DSM-III criteria are more frequent in nonclinical samples than had been previously assumed. Incidence rates of panic attacks have been found to range from 25% of a small scale university sample within a three week period prior to testing (Norton, et al., 1986) to a 14% lifetime rate in a large community survey (Salge, Beck, & Logan, 1988).

Anxiety is a universal phenomenon and is the most prevalent clinical complaint in the general population (Hallam, 1985). A consideration in research with nonclinical subjects is that these subjects may later develop clinical complaints, and thus their nonclinical presentation may be etiologically significant (Telch, et al., 1989). Nonclinical panickers may also provide an analogue of Panic Disorder patients, providing a more accessible source of subjects. Of course, the analogy of

nonclinical panic to Panic Disorder is not a perfect one. The two groups do have some features in common (e.g., the experience of panic attacks), but are dissimilar in other important ways (e.g. level of impairment and/or avoidance).

Nonclinical panic research has served both basic research purposes (e.g. Norton, et al., 1985; Norton, et al., 1986) and analogue purposes (e.g. Donnell & McNally, 1989). Nonclinical subjects have served as analogues and/or comparisons for Generalized Anxiety Disorder (Craske, Rapee, Jackel, & Barlow, 1989), Obsessive-Compulsive Disorder (Rachman & Hodgson, 1980), and Panic Disorder (Norton, et al., 1986). However, there remains a significant amount of research to be conducted examining nonclinical panic, given its potential etiological and analogue significance.

#### The Classification of Anxiety States as per DSM-III-R

DSM-III-R defines panic attacks as "discrete periods of intense fear or discomfort, with at least four characteristic associated symptoms..." (A.P.A., 1987, p. 235). Associated symptoms include: 1) dyspnea or smothering sensations; 2) dizziness, unsteady feelings, or faintness; 3) palpitations or tachycardia; 4) trembling or shaking; 5) sweating; 6) choking; 7) nausea or abdominal distress 8); depersonalization or derealization; 9) numbness or parasthesia; 10) flushes or chills; 11) chest pain or

discomfort; 12) fear of dying; and 13) fears of going crazy or losing control. The attack must occur suddenly with symptoms rising in intensity within 10 minutes. Depending upon the frequency of the attacks, the degree of anticipatory anxiety, and the level of precipitated avoidance, a diagnosis of Panic Disorder, with or without Agoraphobia, would be indicated.

DSM-III-R defines Generalized Anxiety Disorder as being characterized by "unrealistic or excessive anxiety or worry (apprehensive expectation) about two or more life circumstances, e.g., worry about possible misfortune to one's child (who is in no danger) and worry about finances (for no good reason), for six months or longer..." (A.P.A., 1987, p. 251). At least six of the following 18 symptoms should often be present: "1) trembling, twitching, or feeling shaky; 2) muscle tension; 3) restlessness; 4) increased fatigability; 5) dyspnea or smothering sensations; 6) palpitations or tachycardia; 7) sweating or cold palms; 8) dry mouth; 9) dizziness or lightheadedness; 10) nausea, diarrhea, or abdominal distress; 11) hot flashes or chills; 12) frequent urination; 13) trouble swallowing; 14) feeling keyed up or on edge; 15) exaggerated startle response; 16) difficulty concentrating or "mind going blank" because of anxiety; 17) trouble falling or staying asleep; and 18) irritability..." (A.P.A., 1987, p. 252-253).

As noted in DSM-III-R, Panic Disorder is often accompanied by anticipatory anxiety concerning future

attacks. This anxiety is comparable symptomatically to Generalized Anxiety Disorder. Additionally, both the Generalized Anxiety Disorder and Panic Disorder criteria include symptoms of somatic arousal, cognitive impairment, and obsessive fears. The great deal of symptom overlap apparent in DSM-III-R is indicative of the issues underlying the debate concerning the distinction between the two disorders. As a consequence, research has attempted to further delineate the two manifestations of anxiety.

A Summary and Evaluation of the Evidence for the  
Differentiation of Panic and Nonpanic Anxiety

The assertion that panic attacks are treatable with anti-depressant medication (whereas Generalized Anxiety Disorder is not) has come under increasing criticism. For example, Sheehan (1982) reported that 86% of panic patients treated with imipramine relapsed within three months after discontinuing medication. It appears that anti-depressant medication is an efficacious treatment for the symptoms of panic, but has no effect upon any underlying panic-specific pathology (Wolpe & Rowan, 1988). Furthermore, Cox, Lee, & Swinson (1990) conducted a meta-analysis of various treatment studies of Panic Disorder and found the triazolobenzodiazepine, Alprazolam, to be more efficacious than the anti-depressant, Imipramine.

In addition to differential psychopharmacological responding, the separation of Panic Disorder from Generalized Anxiety Disorder was also justified to a large extent by studies which demonstrated a differential response to various biological challenges. Pitts & McClure (1967) found that sodium lactate infusions could produce panic attacks in a sample of Panic Disorder subjects, but not in other anxiety disordered patients. Increased frequencies of panic attacks in Panic Disorder patients have been reported in challenge studies using oral yohimbine (Charney, Heninger, & Breier, 1984); caffeine (Charney, Heninger, & Jatlow, 1985); inhalation of carbon dioxide (Griez, Lousberg, van den Hout, & van der Molen, 1987); isoproterenol infusions (Rainey, Pohl, Knitter, Freedman, & Etedgui, 1984); and voluntary hyperventilation (Rapee, 1986). Klein (1981) has suggested that there is a panic-specific biological mechanism that underlies panic attacks, and it is this which is activated by the various biological challenge tests.

Additional evidence in support of the distinction between the two disorders comes from reported differences in childhood and familial patterns (Torgensen, 1986) and reports that the two disorders have different clinical courses (Breier, Charney, & Heninger, 1985). However, these conclusions are based largely upon retrospective data, making the conclusions somewhat questionable.



The anti-depressant response, challenge response, familial pattern, and clinical course differences, have led some researchers (e.g. Klein, 1981) to conclude that Generalized Anxiety Disorder and Panic Disorder are qualitatively different. Their reasoning being that, if Panic Disorder and Generalized Anxiety Disorder were simply quantitative variations of a unidimensional construct known as anxiety, one would not expect to find differences in course, prognosis, and associated biological mechanisms.

However, several researchers have disputed the interpretation of the evidence used as a basis for the separation of Panic Disorder from Generalized Anxiety Disorder. Alternative explanations of the challenge studies have been forwarded. Clark (1986) has suggested that sodium lactate infusions may indeed trigger somatic arousal, but that it is the catastrophic misinterpretation of these symptoms which is panic specific.

The original sodium lactate studies have also been criticized on methodological grounds. Margraf, Ehlers, & Roth (1986) argued that when baseline levels of trait anxiety are taken into consideration the differential response is not evident. Indeed most studies fail to consider trait anxiety as a potential mediator of diagnostic group differences when studying anxiety disordered patients (Borden & Turner, 1989).

It has also been noted that the manner in which one operationally defines a panic attack can influence self-

reports of panic attacks. Holt & Andrews (1989) measured self-reports of anxiety in a sample of various anxiety disordered patients. Somatic anxiety (e.g. tachycardia), psychic anxiety (e.g. fearfulness and worry), and fears of impending doom were assessed in response to carbon dioxide inhalation and voluntary hyperventilation. Diagnostic group differences were only significant for the measure of fears of impending doom. Panic Disorder patients reported more fears of impending doom than other anxiety disordered patients, but not more psychic or somatic anxiety. Thus, somatic arousal did not differ across groups suggesting a lack of any panic-specific somatic arousal mechanism. The authors suggested that had earlier researchers divided their panic attack criteria into the above three components, the conclusion that there were different biological mechanisms for Panic Disorder and Generalized Anxiety Disorder would not have been supportable.

It has also been reported that the majority of anxiety disordered patients, regardless of diagnosis, experience panic attacks. Barlow et al. (1985) found that at least 86% of patients with a diagnosis of either Agoraphobia, Panic Disorder, Generalized Anxiety Disorder, or Obsessive-Compulsive Disorder reported panic attacks. However, the Panic Disorder and Agoraphobic groups reported more frequent attacks. These results indicate that any potential biological trigger mechanism of panic may not be specific to Panic Disorder, and that other factors may account for

differences in panic frequency and the prominence of attacks in the clinical presentations of anxiety patients.

Although, no panic specific biological mechanism has been found, one of the more consistently reported differences found between Panic Disorder patients and other anxiety disordered patients has been that Panic Disorder patients report more severe somatic symptoms when anxious. Hoehn-Saric (1982) found that Panic Disorder patients reported more autonomic symptoms compared to Generalized Anxiety Disorder patients. Similar findings have been reported by Ehlers, Margraf, Roth, Taylor, & Birbaumer (1988) and Thyer & Himle (1987).

The failure to discover a biological trigger for panic attacks has led researchers to search for other potentially important differences. The most common approach has been to attempt to distinguish the diagnostic categories of Generalized Anxiety Disorder from Panic Disorder on the basis of standard psychometric and self-report comparisons.

Borden & Turner (1989) administered a variety of questionnaires including the State-Trait Anxiety Inventory-trait form (STAI-T) and the Revised Hopkins Symptom Checklist (SCL-90-R) to Panic Disorder, Generalized Anxiety Disorder, and Obsessive-Compulsive Disorder patients. The trait scale of the STAI measures the amount of anxiety that characterizes the patient at most times. The SCL-90-R measures the severity of a variety of anxiety symptoms. The results indicated that Panic Disorder and Generalized

Anxiety Disorder subjects had comparable levels of trait anxiety. However, the Panic Disorder group reported more severe symptoms of trembling, heart pounding, difficulty breathing, spontaneous fear onset, thoughts of dying, catastrophic thoughts, and spells of terror compared to the Generalized Anxiety Disorder group. These results would indicate that overall levels of anxiety are comparable between Generalized Anxiety Disorder and Panic Disorder patients, but that some symptoms when experienced in a severe form are more prototypical of Panic Disorder.

The finding that Generalized Anxiety Disorder and Panic Disorder patients did not differ on trait anxiety levels is a commonly reported result (McNally, 1989). Thus, it becomes important to consider the effects of trait anxiety per se when discussing phenomena hypothesized to be panic-specific. Unfortunately, a significant amount of research fails to consider this.

For example, Thyer & Himle (1987) suggested that to address the issue of qualitative differences it is necessary to find different patterns of differences rather than differences alone. Thyer & Himle (1987) compared Simple Phobic patients to Panic Disorder patients on a 55 item Anxiety Symptom Questionnaire. The Panic Disorder group reported more severe symptoms on approximately half of the items. A subsequent rank order correlation between the symptom severity ratings of the two groups did not account for a significant amount of the variance. The authors

concluded that the two symptom patterns were different. However, the question of qualitative or quantitative differences can't be addressed by the statistical methodology employed by the authors. In other words, at what point does the absolute value of the rank order correlation reflect a qualitative, rather than a quantitative difference? A more logical approach would be to control for high trait anxiety levels (the key symptomatological criterion for Generalized Anxiety Disorder) and then investigate differences. Any differences found would then reflect the unique contribution of panic attacks. Thus, Thyre & Himle's (1987) conclusions can be criticized for not controlling for the potential confound of trait anxiety levels.

### Cognitive Theories of Panic Etiology and Maintenance

With the realization that the majority of panic attacks might be precipitated and/or mediated by non-biological factors, cognitive conceptualizations of panic and nonpanic anxiety have been given ever increasing research attention (Warren, Zgourides, & Jones, 1989). Several researchers have provided compelling evidence for the importance of cognitive factors in panic. Panic Disorder patients have been found to react to false high heartrate feedback with panic reactions suggesting that they had psychologically produced the panic reaction (Anderson, Noyes, & Crowe,

1984). Similar evidence was reported by Beitman, Basha, Flaker, DeRosear, Mukerji, & Lanberti (1987). They reported that many cardiac patients report as severe somatic distress as do Panic Disorder patients, but do not report a subjective state of panic. This would suggest that Panic Disorder patients have high somatic anxiety, but that additional cognitive variables may be important in the development of panic attacks. Additionally, a common clinical observation made of Agoraphobic patients is that panic attack frequency decreases in the presence of trusted companions. Argyle (1988) suggests that this reduction of panic frequency clearly demonstrates the importance of cognitive factors as mediators of panic symptomatology.

Goldstein & Chambless (1978) were the first to outline a cognitive theory of panic which has become known as "fear of fear." Fear of fear is a conditioned association between maladaptive, catastrophic cognitions and the somatic arousal produced from anticipatory anxiety. This theory was used to explain the maintenance of panic attacks and the development of phobic avoidance (Agoraphobia). Adler, Craske, Kirshenbaum, & Barlow (1989) noted that the inclusion of "anticipatory anxiety of future panic attacks" as an alternative to panic frequency as a diagnostic criterion for Panic Disorder in DSM-III-R is reflective of the acceptance of the fear of fear theory.

In one of the few prospective studies available, Mavissakalian (1988) found that fear of fear was an

important mediator of the relationship between panic severity and phobic avoidance in agoraphobics. Specifically, agoraphobics who overpredicted the amount of anxiety they would experience in a behavioural avoidance task were found to engage in more avoidance regardless of panic severity. In disagreement, Adler, et al. (1989) noted that the majority of investigations have found only small positive correlations between measures of fear of fear and avoidance.

In an extension of the fear of fear model, Ley (1985) and Clark (1986) have put forth a model of panic that incorporates both somatic arousal and panic-specific cognitive processes. They propose that somatic arousal is a very common experience and that when the arousal is combined with a tendency to interpret it as a sign of impending ill physical health, loss of consciousness, and/or insanity, it will cause additional increases in somatic arousal. Eventually through a continued activation of a positive feedback loop, panic attacks are precipitated. The primary difference between this model and the fear of fear model, is that the fear of fear model relies on a conditioning paradigm to explain the relationship between catastrophic cognitions and avoidance. The models proposed by Clark (1986) and Ley (1985) incorporate a more dynamic relationship between cognitions and the genesis of panic attacks, from which avoidance may, or may not, result.

Available evidence is generally supportive of this model. Hibbert (1984) found that Panic Disorder patients reported more catastrophic cognitions with themes of death, insanity, and losing control than did Generalized Anxiety Disorder patients. Similar findings had previously been reported by Beck, Laude, & Bohnert (1974). Mavissakalian (1989) concluded that panic anxiety is strongly related to the catastrophizing by the patient of his/her own anxiety symptoms.

Peterson & Reiss (1987) have suggested that, in addition to a conditioned fear of fear and catastrophic ideation, a panicker also evidences a specific cognitive sensitivity to anxiety symptoms. In support of Clark's (1986) model of panic, Reiss & McNally (1985) developed the construct of anxiety sensitivity. Anxiety sensitivity is a cognitive predisposition to interpret perceived anxiety symptoms in a fearful manner. Panic Disordered patients have been found to score high on the Anxiety Sensitivity Index (ASI; Peterson & Reiss, 1987), a measure developed to assess anxiety sensitivity (McNally & Lorenz, 1987; Holloway & McNally, 1987). Additionally, Panic Disorder patients have been found to have higher levels of anxiety sensitivity compared to Generalized Anxiety Disorder patients (Sartory & Olajide, 1988). Research has also indicated that differential responses to challenge tasks can be predicted from measures of anxiety sensitivity (Holloway & McNally, 1987; Donnell & McNally, 1989). Holloway & McNally (1987)



also found trait anxiety scores to be significant predictors of challenge response. This further emphasizes the importance of considering trait anxiety levels when doing panic research.

Other cognitive variables have also been found to mediate panic and anxiety phenomena. It has often been noted that cognitive errors including over-generalization, misconstruction, over-estimation, and dichotomous thinking characterize anxiety patients (Ellis & Dryden, 1987; Mizes, et al., 1987; Argyle, 1988). Warren et al. (1989) reported that a measure of irrational beliefs (based upon Ellis, 1962) could predict avoidance in a sample of patients with various anxiety disorders. Additionally, McNair, Lorr, & Droppleman (1971) have noted that anxiety lowers the efficiency of cognitive processing, which leads to higher scores on a measure of subjective cognitive dysfunction (e.g., difficulties with concentration). Holt & Andrews (1989) have argued that cognitions are the best discriminators between anxious and non-anxious states.

Additional evidence for the cognitive mediation of anxiety has been provided by studies using information processing experimental designs. There is evidence that some cognitive processes operate below the level of conscious awareness in panickers. Given the methodological concern with regard to basing research primarily upon self-report inventories (e.g., reactivity), this body of evidence is quite important (Clark, 1988). Butler & Mathews (1983)

reported that Generalized Anxiety Disorder patients recalled more words of a threatening nature compared to normal controls. Norton, Schaefer, Cox, Dorward, & Wozney (1988) have reported anxiety congruent schema effects in a word recall test in a sample of nonclinical panickers. When primed by a narrative of a panic attack, panickers recalled more adjectives denoting anxiety and its negative consequences than did nonpanickers. Similarly, Nunn, Stevenson, & Whalan (1984) found that agoraphobic patients recalled more phobic words than neutral words in a free recall task.

Similar studies using Generalized Anxiety Disorder patients and other non-panicking anxious subjects report more equivocal results, with some studies finding the effect and others reporting contradictory results (Mathews, Mogg, May, & Eysenck, 1989). Foa, McNally, & Murdock (1989) found that speech phobics actually recalled fewer words denoting anxiety compared to non-anxious words. Mathews et al. (1989) have suggested that in some anxiety states, there is a cognitive defense against the processing of anxious stimuli. However, in more anxious subjects this defense is compromised by fear and the selectively more efficient processing of threat cues becomes ascendent. This would suggest that cognitive mediation of anxiety experiences would be more salient in Panic Disorder than in disorders characterized by less intense anxiety.

There is some evidence to support this contention. Mathews & Macleod (1985) found that patients with Generalized Anxiety Disorder were slower to name threatening words than non-threatening words on a Stroop colour naming task. Slower reactions on the Stroop Test are indicative of some interference of processing. Macleod, Mathews, & Tata (1986) found that threatening words were more distracting than non-threatening words on a visual tracking task in a sample of Generalized Anxiety Disorder patients. Increased distractibility and/or interference of processing would likely lead to lower recall, thus supporting the Mathews et al. (1989) theory. However, most information processing investigations have also failed to consider the potential confounds of trait anxiety in mediating results.

Some research findings fail to support a cognitive model of panic attack precipitation. Rachman, Levitt, & Lopatka (1987) reported that some panic attacks were unaccompanied by fearful cognitions. Wolpe & Rowan (1988) found that catastrophic thoughts of dying, fainting, and going insane were reported after the onset of initial panic attacks. These reports suggest that catastrophic ideas (as measured by self-report) are not the direct precursors of panic attacks since they followed panic onset. However, the inherent difficulty in assessing thoughts prior to and during a panic attack makes any definitive evaluation of the cognitive models of panic difficult. Furthermore, it may be the case that cognitions are less important during an attack

or just prior to an attack, than they are between attacks. In other words, cognitive variables may be more important as trait markers of panic attack susceptibility, rather than as markers of the state of panic.

### Research Findings with Non-Clinical Panickers and Clinical Relevance

Although the debate regarding the separation of Panic Disorder from Generalized Anxiety Disorder has been vigorously pursued via applied clinical research, there has been a trend towards more basic research using nonclinical subjects (e.g. Telch, et al., 1989; Salge, et al., 1988; Norton, et al., 1986; Norton, et al., 1985). Most nonclinical research findings are compatible with those obtained with clinical subjects. Similar patterns of results are obtained, with nonclinical panicker scores on personality/ cognitive measures usually indicating less dysfunction than clinical samples, but more dysfunction than normals. For example, Norton et al. (1986) found that nonclinical panickers scored higher than nonpanickers on self-report measures of psychopathology (e.g. STAI and Beck Depression Inventory), but scored lower than norms reported for Panic Disorder samples. Also, the panic symptoms reported were somewhat less severe than those reported by Panic Disorder patients, although the types of symptoms endorsed were similar.

Nonclinical panickers have been found to score higher on the ASI (range= 21-27; depending on inclusion criteria) compared to nonpanickers, who typically score under 20 (Telch, et al., 1989). Reports of nonclinical panickers scoring higher than nonpanickers on the ASI, but lower than Panic Disorder patients are common (e.g. Telch et al., 1989; Rapee, Ancis, & Barlow, 1988; Cox, 1989). Donnell & McNally (1989) using nonclinical subjects found that ASI scores and a history of panic attacks were together predictive of greater subjective arousal during a hyperventilation challenge. However, panic history without high ASI scores was not predictive of arousal. Since the criteria used for determining panic history included only that the panicker have at least one uncued (i.e. non-phobic) attack in the past year, the sample may have had less panic related features than a sample comprised of more frequent and/or recent panickers. For example, panic attacks reported as having occurred several months ago, may have been associated with increased responsivity at that particular time, but no longer occur due to a decreased level of life stress. Thus, studies examining panic related phenomena should use more stringent criteria for classifying subjects.

Nonclinical subjects have also served as comparisons for other anxiety disordered patients. Craske et al. (1989) compared nonclinical subjects to Generalized Anxiety Disorder patients to investigate both quantitative and qualitative aspects of worrying. Results indicated that

both groups were comparable regarding duration of worry, maximum level of anxiety aroused, degree of aversiveness, expected outcome, and level of anxiety while resisting the worry. However, the Generalized Anxiety Disorder group rated their worries as less controllable, more resistant to control attempts, and less realistic. The Generalized Anxiety Disorder subjects were also more likely to identify somatic health concerns as comprising the content for their worries. Craske et al. (1989) suggest that symptom profiles (e.g. affective intensity and duration) are not good markers for differentiating clinical mood disorders, but that a particular affective expression combined with a sense of uncontrollability best determines whether the symptoms are of clinical significance, or not. The significance of the Craske et al. (1989) study is that it demonstrates that nonclinical subjects do exhibit anxiety symptomatology that has some overlap with that of clinically anxious patients (i.e. Generalized Anxiety Disorder). Thus, the notion of contrasting clinically anxious patients with a "normal control group" is invalid. Any nonclinical group may contain significant elements of anxiety, and this must be addressed in any discussion of results obtained using nonclinical groups as comparisons. Thus, nonclinical research attempting to investigate differences unique to panic attacks must have a non-panicking comparison group with comparable levels of trait anxiety.

The use of nonclinical panickers who fail to meet DSM-III-R criteria for panic frequency as a potential analogue can be substantiated by several lines of evidence. The findings of similar pattern of personality/cognitive differences between panickers and nonpanickers (in both clinical and nonclinical samples) would suggest that the nonclinical panicker and the Panic Disorder groups are indeed related. Several researchers (e.g. Craske & Barlow, 1988; Mavissakalian, 1988) have failed to find a consistent relationship between panic frequency and avoidance (Warren et al., 1989). Since the lifestyle disruption produced by severe avoidance (i.e. agoraphobia) is often the impetus for seeking therapy, it would seem logical to postulate that nonclinical panickers are similar to clinical panickers in panic attack specific phenomena, but not avoidance specific phenomena. Although important differences may exist, it remains to be discovered what these differences may be. The use of nonclinical panickers as analogues becomes more supportable when the research questions addressed follow directly from previous research areas where established similarities exist.

## The Present Study

There are three explicit purposes to the present research: 1) To determine the prevalence of panic attacks in a nonclinical sample; 2) To compare self-reported panickers to nonpanickers on a variety of variables related to anxiety derived from previous clinical and nonclinical research; and 3) To likewise compare self-reported panickers to nonpanickers who are highly anxious as indicated by STAI-T scores.

The dependent variables used have been previously found to be associated with panic and nonpanic anxiety. These variables included measures of: 1) trait anxiety levels; 2) the amount of cognitive anxiety symptomatology typically experienced; 3) the amount of somatic anxiety symptomatology typically experienced; 4) the fears of negative consequences (both social and physical) which are perceived to result from anxiety symptoms; 5) irrational beliefs; 6) anxiety sensitivity; and 7) the amount of cognitive dysfunction typically experienced.

Previous research has suggested that Panic Disorder patients, compared to Generalized Anxiety Disorder patients, have comparable levels of trait anxiety, more somatic symptoms of anxiety, increased anxiety sensitivity, and more fears concerning potentially catastrophic consequences when anxious. Previous research with nonclinical subjects has



indicated that panickers, compared to nonpanickers, have higher trait anxiety, more anxiety sensitivity, and higher scores on measures of general psychopathology. However, trait anxiety levels have not been controlled for in the nonclinical research, and it remains to be seen if nonclinical panickers are different from anxious nonpanickers.

Given that previous findings with nonclinical panickers have largely replicated research findings with clinical panickers, it is expected that differences between nonclinical panickers and anxious nonpanickers will replicate the differences reported between Panic Disorder and Generalized Anxiety Disorder patients.

Two sets of comparisons were made. First, on all self-report measures nonclinical panickers were contrasted with nonpanickers (without controlling for levels of trait anxiety). Differences found may be attributed to the effects of panic status and trait anxiety. Second, nonclinical panickers were compared to anxious nonpanickers on each self-report measure. Thus, any differences found may be viewed as reflecting the unique contribution of panic.

The specific hypotheses tested in the present study were: 1) That nonclinical panickers would achieve scores indicating significantly more dysfunction than nonpanickers on all psychometric measures. This hypothesis was based on the findings of previous research which indicates that the

measures employed in the present study are all related to panic and/or high trait anxiety. Since the panic group presumably has higher trait anxiety, their scores should indicate more dysfunction; 2) That nonclinical panickers, compared to anxious nonpanickers, would score more dysfunctional on measures typically found to differentiate panic subjects from high trait anxiety subjects. However, it is not expected that nonclinical panickers will achieve scores significantly different from those of anxious nonpanickers on measures typically found to be related only to trait anxiety levels. Specifically, nonclinical panickers, compared to anxious nonpanickers, should be found to report: a) more somatic anxiety; b) greater anxiety sensitivity; and c) more catastrophic fears of social and physical consequences of being anxious. No differences should be found on measures of: a) cognitive anxiety; b) rational beliefs; and c) cognitive dysfunction (confusion).

## CHAPTER II

### METHOD

#### Subjects

Subjects were recruited from undergraduate psychology classrooms from the campuses of Simon Fraser University and Langara College. Subjects were asked to complete a series of questionnaires related to anxiety. Participation was voluntary and informed consent obtained. Two hundred and eighty nine subjects completed the questionnaire package adequately. Approximately 70.9% of the sample was female, and subject age ranged from 17-68 ( $M=25.47$  years).

#### Materials

The questionnaire package consisted of seven measures. These were: 1) the State-Trait Anxiety Inventory trait form (STAI-T; Spielberger, Gorsuch, & Lushene, 1970); 2) the Cognitive Somatic Anxiety Questionnaire trait form (CSAQ; Schwartz, Davidson, & Goleman, 1978); 3) the Barnes Vulcano Rationality Test (BVRT; Barnes & Vulcano, 1982); 4) the Agoraphobic Cognitions Questionnaire-Revised (ACQ-R; Chambless, Caputo, Gallagher, & Bright, 1984); 5) the anger and confusion scales of the Profile of Mood States (POMS; McNair, Lorr, & Droppleman, 1971); 6) the Anxiety

Sensitivity Index (ASI; Peterson & Reiss, 1986); and 7) a revised version of the Panic Attack Questionnaire (PAQ; Norton, Dorward, & Cox, 1986). Some items from the PAQ and the anger scale of the POMS were collected as part of an ongoing research project and are not pertinent to the present study.

Stait-Trait Anxiety Inventory trait form. This 20 item scale is designed to measure a person's anxiety level which characterizes the individual at most times (trait anxiety), as opposed to situation specific anxiety (state anxiety). Each item contains a statement (e.g., "I worry too much over something that really doesn't matter") which the respondent rates on a four point Likert type scale (1-almost never generally feel this way; 4-almost always feel this way). Higher scores are indicative of higher trait anxiety. Mean group scores typical for Generalized Anxiety Disorder patients are in the 50-60 range (e.g. Mathews, et al., 1989). This measure has been extensively used in clinical and nonclinical research. Reliability and validity properties for this measure are reported to be excellent (Speilberger, 1983).

Cognitive Somatic Anxiety Questionnaire trait form. This 14 item questionnaire measures the typical anxiety symptoms experienced by a person in relation to two dimensions. The Cognitive scale measures the degree of anxiety produced cognitive impairment (e.g., "I can't keep anxiety provoking thoughts out of my head"). The Somatic

scale measures the degree of somatic disturbance associated with anxiety (e.g., "My heart beats faster"). Both scales contain seven items rated on a five point Likert type scale (1-not typically experienced when anxious; 5-very much typical of anxiety experience). Reliability and validity properties are reported to be good (Schwartz, et al., 1978).

Barnes Vulcano Rationality Test. This 44 item test measures the degree to which a person holds rational beliefs. Rational beliefs are operationalized as being those beliefs which are not based upon absolutistic, dichotomous, overgeneralized, and otherwise maladaptive heuristics (e.g., "I feel that I must succeed at everything I undertake"). This operationalization is typical of Rational Emotive Therapy proponents and is based upon Ellis's (1962) irrational beliefs model. The subject responds to each item on a five point Likert type scale (1-agree strongly with statement; 5-disagree strongly with statement). The higher the subject's total score the more rational their belief system is. Validity and reliability properties are good (Barnes & Vulcano, 1982).

Agoraphobic Cognitions Questionnaire-Revised. This 14 item questionnaire measures the frequency of catastrophic thoughts reported by a subject when nervous or frightened. The measure has two scales. The physical scale measures the frequency of thoughts concerning somatic disturbance (e.g., "I will choke to death") and the social scale measures the frequency of thoughts concerning potential negative social

evaluation (e.g. "I am going to scream"). All items are responded to on a five point Likert type scale (1-thought never occurs; 5-thought always occurs when I am nervous). Higher scores are indicative of more frequent catastrophic thinking when nervous. Validity and reliability properties of the measure are good (Chambless, et al., 1984).

The Confusion Scale of the Profile of Mood States.

This seven item scale measures the degree of perceived cognitive impairment experienced in the week prior to administration. Impairment includes memory, attention, and concentration difficulties. Each item consists of a short descriptor (e.g., "uncertain about things") to which the subject indicates how descriptive the item was of their feelings on a five point Likert scale (0-not at all; 4-extremely). Higher scores are indicative of more confusion. Reliability and validity properties for the entire POMS are good (McNair, et al., 1971). Reliability and validity data on the use of a single scale are unavailable.

Anxiety Sensitivity Index. This 16 item questionnaire measures the degree of anxiety experienced in response to the perception that one is experiencing anxiety symptoms (e.g., "It scares me when I am nervous"). The subject responds to each item on five point Likert type scale (0-very little; 4-very much). Higher scores are indicative of increased anxiety sensitivity. Mean scores for Panic Disorder/Agoraphobia patients typically are in the 36-38 range (Donnel & McNally, 1989) and the normative mean is

18.4 (Peterson and Reiss, 1987). Reported validity and reliability data appear favourable (Telch, Shermis, & Lucas, 1989).

Panic Attack Questionnaire. This research instrument determines the subject's panic status (has, or has not, experienced a panic attack, as per DSM-III-R criteria) and evaluates the associated phenomena (e.g., frequency, duration, symptomatology, history, etc.). The PAQ's format consists of a variety of Likert type scales and checklists. The PAQ has good concurrent validity with structured interview measures (Norton, et al. 1986).

For the purposes of the present study the PAQ served only to identify a subject's panic status. A subject was classified as a panicker if they reported at least one attack in the three weeks prior to testing, or at least three attacks in the last year. Reported attacks did not meet inclusion criteria if either: the attacks were only in response to life threatening circumstances (e.g. car accident), if the attacks were not characterized by at least four DSM-III-R symptoms of a moderate or greater severity, or if the symptom intensity level of an attack did not reach peak within 10 min. Subjects who reported having never experienced a panic attack during their lifetime were classified as nonpanickers.

## Procedure

Permission was sought from course instructors to solicit subjects during lectures. At the appointed time the author would read aloud the document entitled, Subject Consent Form (see Appendix A), following which questionnaire packages were distributed. Subjects were instructed to read the attached consent form and indicate voluntary consent by signature. Further instruction indicated to the subjects not to indicate their name or other identifying information on the remaining questionnaires and reiterated the anonymous, confidential, and voluntary nature of their participation. Depending upon course time constraints, questionnaires were either completed in class, or were returned completed to the author's departmental mailbox at a later date.

Upon receiving each subject's questionnaires, the author separated the signed consent forms from the questionnaires. Thus, during the scoring of the measures, identifying data was not available to the investigator.

## Data Analyses

All data were analyzed by BMDP (1980) statistical software. Comparisons on measures between two groups (nonclinical panickers vs. nonpanickers) were analyzed via independent t-test procedures. Levene's tests were used to



test for homogeneity of variances. Depending upon whether the assumption of homogeneity was met, or not, pooled or separate estimates of variances were used in the calculation of the t-statistic.

Comparisons involving three groups (nonclinical panickers vs. non-anxious nonpanickers vs. anxious nonpanickers) were conducted via an analysis of variance (ANOVA) for each measure. When Levene's tests revealed non-homogeneity of variances, Welch and Brown-Forsythe tests were conducted.

Planned pairwise comparisons between the nonclinical panickers and the anxious nonpanickers on each measure were calculated using a Bonneferoni t-test procedure. Homogeneity of variance assumptions were investigated with Levene's tests, and appropriate estimates of variance were incorporated.

To control for possible familywise error rates in each set of comparisons, alpha was corrected using a Bonneferoni correction. Alpha was set to alpha divided by the number of comparisons.

## CHAPTER III

### RESULTS

#### Sample Characteristics

Of the 289 respondents, 113 subjects reported having experienced at least one panic attack during their lifetime. However, four subjects who indicated that they had experienced panic attacks failed to endorse a sufficient symptom profile and were excluded from all further report. It should be noted that an examination of the symptom profile was possible only for the 88 subjects who reported an attack within the last year. Thus, there were 21 subjects who reported attacks, but for whom no symptomatological data were collected. This may have resulted in an inflation of the lifetime prevalence rate estimate of panic.

Overall sample estimates of prevalence rates for panic were 37.7 % for lifetime, 30.4% for the past year, and 11.1% for the three weeks prior to testing.

Examination of panic attack incidence in the preceding year, indicated that of the 109 self-reported panickers, 21 (19.3%) had not had a panic attack, 54 (68.8%) had one or two attacks, 17 (15.6%) had three or four attacks, six (5.5%) had five or six attacks, five (4.6%) had seven or eight attacks, two (1.8%) had nine or ten attacks, and four (3.7%) had 11 or more attacks.

Examination of panic attack frequency in the three weeks prior to testing (response sample equals 102 due to incomplete reports) indicated that 70 (68.6%) had not had an attack, 14 (13.7%) had one attack, eight (7.8%) had two attacks, one (1.0%) had three attacks, and nine (8.8%) had four or more attacks.

Thirty four subjects (12.1%) from the overall sample of 289 subjects reported a sufficient number of panic attacks and/or an attack within the three weeks prior to testing to be classified as a panicker in the present study. Of these 34 panickers, only two subjects did not report an attack in the last three weeks. All panickers reported experiencing at least four DSM-III-R symptoms at a moderate or greater severity.

## Comparisons of Panickers and Nonpanickers

As previous research had indicated that sex and age may mediate responses on the psychometrics, two sets of two way ANOVAs were conducted for each psychometric. For the first set of ANOVAs, panic status and sex were grouping factors, and for the second set of ANOVAs, panic status and age (based upon a median split) were the grouping factors. To correct for family-wise error rates Alpha was set to .005.

The results indicated no significant interactions between either age or sex with panic status . The lack of significant interactions indicates that neither sex nor age mediated any possible differences between nonpanickers and panickers. All subsequent analyses will be based upon a combined sample.

Panickers (Ps) were compared to nonpanickers (NPs) on each psychometric measure and the variable age using t-tests. Alpha was set at .005 to protect against family-wise error rates. No Levene tests for homogeneity of variances were significant. These results are presented in Table 1.

Panickers were significantly more dysfunctional on all psychometrics compared to nonpanickers. Panickers scored higher on the STAI-T [ $\underline{M}(\text{Ps}) = 45.4$  vs  $\underline{M}(\text{NPs}) = 39.0$ ,  $\underline{t}(208) = 3.90$ ,  $p < .0001$ ]; the CSAQ-T somatic scale [ $\underline{M}(\text{Ps}) = 18.4$  vs  $\underline{M}(\text{NPs}) = 14.2$ ,  $\underline{t}(205) = 4.69$ ,  $p < .0001$ ]; the CSAQ-T cognitive scale [ $\underline{M}(\text{Ps}) = 19.7$  vs  $\underline{M}(\text{NPs}) = 15.1$ ,  $\underline{t}(1, 206) = 4.72$ ,

$p < .0001$ ]; the ASI [ $M(Ps) = 28.5$  vs  $M(NPs) = 17.0$ ,  $t(208) = 5.86$ ,  $p < .0001$ ]; the ACQ-R physical scale [ $M(Ps) = 1.77$  vs  $M(NPs) = 1.43$ ,  $t(186) = 3.23$ ,  $p < .0015$ ]; the ACQ-R social scale [ $M(Ps) = 2.04$  vs  $M(NPs) = 1.57$ ,  $t(186) = 4.27$ ,  $p < .0001$ ]; and the POMS confusion scale [ $M(Ps) = 12.6$  vs  $M(NPs) = 10.1$ ,  $t(208) = 4.41$ ,  $p < .0001$ ]. On the BVRT, panickers scored lower ( $M = 133.2$ ) than nonpanickers ( $M = 150.5$ ) [ $t(207) = 4.15$ ,  $p < .0001$ ]. On the variable of age, panickers ( $M = 24.3$ ) did not differ significantly from nonpanickers ( $M = 25.2$ ) ( $t(207) = 0.53$ ,  $p < .5978$ ).

Thus, panickers reported higher levels of trait anxiety, more cognitive and somatic symptoms when anxious, more catastrophic fears of adverse social and physical consequences of anxiety, less rational beliefs, greater anxiety sensitivity, and more subjective cognitive dysfunction (confusion).

Table 1

Descriptive Statistics: Nonpanickers vs Panickers.

| Measure   | Nonpanickers |       |      | Panickers |       |      | t    | p**   |
|-----------|--------------|-------|------|-----------|-------|------|------|-------|
|           | N*           | M     | sd   | N         | M     | sd   |      |       |
| STAI      |              |       |      |           |       |      |      |       |
| trait     | 176          | 39.0  | 8.7  | 34        | 45.4  | 9.4  | 3.90 | .0001 |
| CSAQ-T    |              |       |      |           |       |      |      |       |
| somatic   | 174          | 14.2  | 4.7  | 33        | 18.4  | 4.5  | 4.69 | .0001 |
| cognitive | 174          | 15.1  | 5.2  | 34        | 19.7  | 5.1  | 4.72 | .0001 |
| ASI       | 176          | 17.0  | 10.8 | 34        | 28.5  | 8.8  | 5.86 | .0001 |
| ACQ-R     |              |       |      |           |       |      |      |       |
| physical  | 159          | 1.43  | 0.51 | 29        | 1.77  | 0.55 | 3.23 | .0015 |
| social    | 159          | 1.57  | 0.54 | 29        | 2.04  | 0.62 | 4.27 | .0001 |
| BVRT      | 175          | 150.5 | 22.0 | 34        | 133.2 | 23.0 | 4.15 | .0001 |
| POMS      |              |       |      |           |       |      |      |       |
| confusion | 176          | 8.8   | 4.6  | 34        | 12.6  | 4.6  | 4.41 | .0001 |
| AGE       | 176          | 25.2  | 9.0  | 33        | 24.3  | 7.7  | 0.53 | ns    |

\* Group sizes vary due to incomplete questionnaires.

\*\* Alpha was set to .005 to correct for family-wise error rates.

Comparisons of Nonanxious Nonpanickers, Anxious  
Nonpanickers, and Panickers

Group membership in the non-anxious non-panicking (NANP) or the anxious non-panicking (ANP) groups was determined using a median split on STAI-T scores (Median= 39). Prior to investigation of differences between the panicking and non-panicking groups on the psychometrics, possible sex and age interactions were investigated. Two sets of two way ANOVAs were conducted to investigate possible interactions of sex or age with panic status. Using an Alpha set to .005 (to correct for family-wise error), no significant interactions of age or sex with panic status were found. Thus, sex and age did not mediate any potential differences attributable to panic status. All further analyses are based upon a combined sample, collapsed across age and sex.

To investigate differences between panickers, non-anxious nonpanickers, and anxious nonpanickers, a series of ANOVAs, using panic status as the grouping variable, were conducted on the psychometric measures. Levene tests variances were conducted for each ANOVA. Some Levene tests were significant and Brown-Forsythe statistics and Welch statistics (which approximate an F distribution) were calculated for these ANOVAs. Significance levels yielded for these tests did not appreciably differ from calculated F statistics. Thus, for simplicity of presentation, only the F statistics will be reported.

These results are presented in Table 2. The analyses yielded significant main effects of panic status for all psychometric measures: 1) STAI-T;  $F(2, 207) = 144.35$ ,  $p < .0001$ ; 2) CSAQ-T (cognitive);  $F(2, 205) = 49.75$ ,  $p < .0001$ ; 3) CSAQ-T (somatic);  $F(2, 204) = 18.31$ ,  $p < .0001$ ; 4) ACQ-R (physical);  $F(2, 186) = 6.82$ ,  $p < .002$ ; 5) ACQ-R (social);  $F(2, 186) = 17.13$ ,  $p < .0001$ ; 6) ASI;  $F(2, 207) = 40.89$ ,  $p < .0001$ ; 7) BVRT;  $F(2, 206) = 57.29$ ,  $p < .0001$ ; and 8) POMS (confusion);  $F(2, 207) = 31.40$ ,  $p < .0001$ .



Table 2

Descriptive Statistics and Anova Comparisons: Nonanxious  
Nonpanickers vs Anxious Nonpanickers vs Panickers.

| Measure   | Nonanxious |       |      | Anxious |       |      | n  | M     | sd   | F       |
|-----------|------------|-------|------|---------|-------|------|----|-------|------|---------|
|           | n          | M     | sd   | n       | M     | sd   |    |       |      |         |
| STAI      |            |       |      |         |       |      |    |       |      |         |
| trait     | 87         | 31.7  | 4.2  | 89      | 46.0  | 5.6  | 34 | 45.4  | 9.4  | 144.35* |
| CSAQ-T    |            |       |      |         |       |      |    |       |      |         |
| cognitive | 86         | 12.2  | 3.3  | 88      | 17.9  | 5.2  | 34 | 19.7  | 5.1  | 49.75   |
| somatic   | 86         | 13.0  | 3.8  | 88      | 15.5  | 5.1  | 33 | 18.4  | 4.5  | 18.31   |
| ACQ-R     |            |       |      |         |       |      |    |       |      |         |
| physical  | 84         | 1.37  | 0.54 | 75      | 1.51  | 0.47 | 29 | 1.77  | 0.55 | 6.82    |
| social    | 84         | 1.42  | 0.48 | 75      | 1.74  | 0.55 | 29 | 2.04  | 0.63 | 17.13   |
| ASI       | 87         | 12.3  | 8.2  | 89      | 21.5  | 11.1 | 34 | 28.5  | 8.8  | 40.89   |
| BVRT      | 87         | 163.9 | 18.5 | 88      | 137.3 | 16.6 | 34 | 133.2 | 23.0 | 57.29   |
| POMS      |            |       |      |         |       |      |    |       |      |         |
| confusion | 87         | 6.7   | 3.3  | 89      | 10.8  | 4.8  | 34 | 12.6  | 4.6  | 31.40   |

\*All overall F-tests were significant at  $p < .0001$ , with the exception of the overall F-test for the ACQPHYS which was significant at  $p < .002$ .

### Pairwise Comparisons: Anxious Nonpanickers and Panickers

Since the comparisons of primary interest were between the panicker and the anxious nonpanicker groups, planned pairwise comparisons were conducted for each psychometric. Bonferroni t-test statistics were calculated for each comparison. The mean square error terms used for these calculations were those obtained from the overall three group ANOVAs. These error terms provided better estimates of error than those which could be obtained from ANOVAs based upon only the panicker and anxious nonpanicker groups. Levene tests were conducted for each of these comparisons. Only the comparisons on the STAI (trait) and the BVRT yielded a significant Levene. Therefore, the t statistic for these comparisons was based upon separate estimates of group variances. Alpha was set to .005 to protect against familywise error rates.

The results from the pairwise comparisons are presented in Table 3. The results indicated that the panicker and anxious nonpanicker groups did not differ significantly on STAI (trait) scores [ $\underline{M}(P) = 45.4$  vs.  $\underline{M}(ANP) = 46.0$ ,  $\underline{t}(42.41) = 0.35$ ,  $p < .7294$ ]; CSAQ-T (cognitive) [ $\underline{M}(P) = 19.8$  vs  $\underline{M}(ANP) = 17.9$ ,  $\underline{t}(205) = 1.96$ ,  $p < .0508$ ]; ACQ-R (physical) [ $\underline{M}(P) = 1.77$  vs.  $\underline{M}(ANP) = 1.51$ ,  $\underline{t}(185) = 2.32$ ,  $p < .0212$ ]; ACQ-R (social) [ $\underline{M}(P) = 2.04$  vs.  $\underline{M}(ANP) = 1.74$ ,  $\underline{t}(185) = 2.62$ ,  $p < .0096$ ]; BVRT scores [ $\underline{M}(P) = 133.2$  vs.  $\underline{M}(ANP) = 137.3$ ,  $\underline{t}(46.96) = 0.94$ ,

$p < .3532$ ] and POMS (confusion) [ $M(P) = 12.6$  vs.  $M(ANP) = 10.8$ ,  $t(207) = 2.13$ ,  $p < .0344$ ].

Thus, the panicker and anxious nonpanicker groups did not significantly differ on trait anxiety levels, the amount of cognitive anxiety, the frequency of catastrophic fears regarding social and physical consequences of being anxious, the amount of cognitive disability typically experienced, or on the amount of rational beliefs.

Significant differences were found for CSAQ-T (somatic) [ $M(P) = 18.4$  vs.  $M(ANP) = 15.5$ ,  $t(204) = 3.15$ ,  $p < .0019$ ] and ASI [ $M(P) = 28.5$  vs.  $M(ANP) = 21.5$ ,  $t(207) = 3.58$ ,  $p < .0005$ ]. Panickers compared to anxious nonpanickers typically experienced more somatic symptoms of anxiety and were more fearful of symptoms of anxiety (sensitivity).

Table 3

Planned Pairwise Comparisons: Anxious Nonpanickers (ANPs)  
vs. Panickers (Ps).

| Measure   | Anxious<br>Nonpanickers |       | Panickers |       | MS Error | t    | p**     |
|-----------|-------------------------|-------|-----------|-------|----------|------|---------|
|           | N*                      | M     | N         | M     |          |      |         |
| STAI      |                         |       |           |       |          |      |         |
| trait     | 89                      | 46.0  | 34        | 45.4  | 35.17    | 0.35 | .7294   |
| CSAQ-T    |                         |       |           |       |          |      |         |
| cognitive | 88                      | 17.9  | 34        | 19.7  | 20.26    | 1.96 | .0508   |
| somatic   | 88                      | 15.5  | 33        | 18.4  | 20.20    | 3.15 | .0019** |
| ACQ-R     |                         |       |           |       |          |      |         |
| physical  | 75                      | 1.51  | 29        | 1.77  | 0.26     | 2.32 | .0212   |
| social    | 75                      | 1.74  | 29        | 2.04  | 0.28     | 2.62 | .0096   |
| ASI       | 89                      | 21.5  | 34        | 28.5  | 92.26    | 3.58 | .0005** |
| BVRT      | 88                      | 137.3 | 34        | 133.2 | 344.06   | 1.08 | .3532   |
| POMS      |                         |       |           |       |          |      |         |
| confusion | 89                      | 10.8  | 34        | 12.6  | 22.45    | 2.13 | .0344   |

\* Sample sizes vary due to incomplete questionnaires.

\*\* Alpha was set to .005 to correct for family-wise error rates. All p-values with double asterisk are considered significant.

## DISCUSSION

The primary purposes of the present research were: 1) to identify with the PAQ a sample of nonclinical subjects who indicate that they have experienced recent and/or frequent panic attacks meeting DSM-III-R criteria; 2) to investigate psychometric differences between subjects with self-reported panic attacks and subjects without panic attacks; and 3) to investigate psychometric differences between subjects with self-reported panic attacks and subjects without panic attacks, but who were comparable to the panicking subjects on a trait anxiety measure.

Additional implicit purposes included: 1) an evaluation of any differences found regarding their potential relation to theories of panic attack maintenance and etiology; and 2) an examination of the utility of nonclinical research as a potential analogue methodology in the investigation of clinical anxiety states.

### Prevalence of Panic Attacks in a Nonclinical Sample

From a sample of 289 university and college students, a lifetime prevalence rate for panic attacks of 37.7% was obtained. This lifetime prevalence rate is much higher than the 14% prevalence rate reported by Salge et al. (1988). However, the one year incidence rate obtained from the

present sample was 30.4% which is similar to the 34.4 and 34.0% rates reported by Norton, et al. (1985; 1986) respectively. These apparently disparate sets of data may be partly explained by the recent views of Norton, Cox, & Malan (1990) who noted that prevalence rates of panic attacks were significantly affected by the type of assessment employed.

In their review of 23 nonclinical panicker studies it was found that the average one year prevalence of self-reported panic attacks was 27.6%. However, the use of a questionnaire method yielded a higher mean incidence (30.4%), whereas the use of a structured interview yielded a lower estimate (12.8%). Additionally, the use of symptomological inclusion criteria was also found to deflate reported incidence rates, thus perhaps explaining the slightly lower estimate obtained compared to Norton et al. (1985; 1986) Thus, the present data approximates estimates obtained with a similar methodology.

Another potential reason for the higher prevalence rate of the present study than that reported by Salge et al. (1988) is the use of students in the present study instead of the broader range of subjects used in large community surveys. Norton et al. (1990) noted that studies using students have a mean one year incidence rate of 32.1% compared to 6.8% for community surveys. Whether these differences are real in terms of actual panic prevalence, or

a reflection of reactivity on the part of students remains unclear.

Gotlib (1984) has suggested that university students experience a degree of "general distress" which typically results in students appearing more dysfunctional on self-report measures. This may explain the present finding that four subjects (out of a possible 88) indicated that they had experienced a panic attack, but did not endorse a symptomatological profile consistent with panic attacks. However, the previously reported similarities of student panickers to clinical panickers (e.g. Norton et al., 1986; 1990) would perhaps suggest that for most student panickers the "general distress" is manifesting itself in a manner consistent with clinical panic research.

For comparisons to nonpanickers, the present study used student panickers who had at least one panic attack in the three weeks prior to testing, or who had at least three attacks in the past year. The reported attacks could not be in response to a life-threatening situation, had to be characterized by at least four DSM-III-R symptoms of a moderate or greater severity, and had to have an onset of maximum symptom severity within 10 min. In the Norton et al. (1990) review, frequency, recency, and symptomatological criteria all led to more conservative estimates of prevalence. Therefore the 34 students classified as panickers in the present study (12.1% of the total sample)

could be presumed to be panickers using conservative criteria.

### The Psychometric Responses of Panickers and Nonpanickers

As predicted, subjects who met inclusion criteria to be classified as panickers were found to achieve scores indicating more dysfunction on all psychometrics compared to non-panicking subjects. Panickers compared to nonpanickers were found to: 1) have higher levels of trait anxiety; 2) have higher levels of somatic anxiety; 3) have higher levels of cognitive anxiety; 4) have more frequent fears concerning the potentially catastrophic physical consequences of anxiety; 5) have more frequent fears concerning the potentially catastrophic social consequences of anxiety; 6) be more sensitive or fearful of anxiety symptoms; 7) have less rational beliefs; and 8) experience more subjective cognitive dysfunction.

Subjects with panic attacks compared to non-panicking subjects have been consistently reported to achieve scores indicating more dysfunction on anxiety symptomatology measures. For example, Norton et al. (1986) found panickers to score higher on the trait scale of the STAI. Norton et al. (1986) reported panicker and nonpanicker group means of 46.8 and 39.2 respectively, whereas the present study's obtained were 45.4 and 39.0 respectively. Additionally, the present finding that panickers experienced more cognitive



and somatic symptoms of anxiety parallels the findings of Borden & Turner (1989) who found Panic Disordered patients to achieve scores indicating more dysfunction than other anxiety disordered patients on the Revised Hopkins Symptom Checklist, a measure of various anxiety symptoms.

However, the relationship between higher levels of trait anxiety (and its associated symptomatology) and the existence of panic attacks is unclear. It remains to be seen if higher trait anxiety predisposes one to later develop panic attacks, or if the experience of panic attacks creates an increase in trait anxiety. Both possibilities would appear plausible given the available evidence. For example, the subsequent development of anticipatory anxiety between panic attacks in some Panic Disordered patients (A.P.A., 1987) and the finding that panic attacks can be precipitated by biological challenge in some subjects with high levels of trait anxiety, but no previous panic history (Margraf, et al., 1986), would serve to suggest that panic may act as a predisposition towards or as a consequence of high trait anxiety.

The present finding that panicking subjects compared to nonpanickers scored more dysfunctional on the BVRT, a measure of rational beliefs, is consistent with previous research. Argyle (1988) reported that cognitive errors were characteristic of the anxious subject.

The finding that panickers reported more sensitivity/fearfulness of anxiety symptoms compared to

nonpanickers is consistent with previous research. Higher anxiety sensitivity scores have been reported for Panic Disordered patients compared to Generalized Anxiety Disordered patients (Sartory & Olajide, 1988). Additionally, nonclinical panickers have also been found to score higher than non-panicking subjects. Telch et al. (1989) found that nonclinical panickers typically scored in the 21-27 range on the ASI, compared to nonpanickers who typically scored under 20. Likewise, Rapee, Ancis, & Barlow (1988) report means of 25.3 and 17.7. These data are comparable to those of the present study (panickers:  $M=28.5$ ; nonpanickers:  $M=17.0$ ).

The finding that panickers reported more catastrophic fearfulness concerning the possible physical and social consequences of anxiety compared to nonpanickers is consistent with the findings of research with clinical patients. Chambless et al. (1984) found that Agoraphobic patients scored higher than other anxiety disordered patients on a scale which combined both the physical and social items of the ACQ-R. Reported total scores were 2.32 for Agoraphobics and 1.52 for controls. The present study's scores for panickers were 1.77 for the physical scale and 2.04 for the social scale, again suggesting that nonclinical panickers fall in the midrange of a clinical-normative continuum.

The finding that panickers reported more subjective cognitive dysfunction on the confusion scale of the POMS is

in disagreement with the results reported by Norton et al. (1986) who found no differences between panickers and nonpanickers. Norton et al. (1986) report mean scores of 5.9 and 4.0 for panickers and nonpanickers respectively, whereas the present study yielded scores of 12.6 and 8.8 respectively. These discrepancies in scores between the two studies remains to be explained given the similar methodologies employed (e.g. similar sampling procedures and the use of the PAQ to classify subjects). Although, the present finding of more subjective cognitive dysfunction reported by the panicking group would not appear anomalous given the well known deleterious effects of high anxiety upon cognitive functioning. The present finding is also consistent with McNair, et al. (1971) who reported means of 12.9 and 11.0 for samples of Anxiety Disordered patients and college students.

In summary, the above findings are clearly supportive of previous research which has consistently shown that subjects who experience panic attacks typically achieve scores indicating more dysfunction on several psychometrics theoretically related to anxiety. Furthermore, the present data confirms the findings and conclusions from previous research that nonclinical panickers evidence psychometric responses which are similar in pattern to those of clinical panickers, but do so at a less dysfunctional level.

## The Panicker Compared to the Anxious Nonpanicker

When panickers were compared to nonpanickers with comparable trait anxiety levels, the pattern of differences observed was different from that found when trait anxiety is not methodologically controlled for. As noted in the previous section of the discussion, panickers were more dysfunctional than nonpanickers on all psychometrics. When an equally anxious nonpanicker comparison group is employed, panickers differ significantly only on the somatic subscale of the CSAQ-T and the ASI. As predicted, panickers were found to report more somatic symptoms of anxiety and were more fearful of experiencing anxiety symptoms.

Additionally, several of the comparisons on the remaining measures were significant until a family-wise correction was used. Specifically, the differences on the ACQ-R subscales and the confusion scale of the POMS were both in a similar direction, with the panicker group means being indicative of greater dysfunction. Although, it was predicted that panickers would appear more dysfunctional on the ACQ-R scales, the present results cannot be viewed as supportive. It may be that catastrophic fears of physical and social consequences are not salient to the nonclinical panicker, unlike the Panic Disorder patient. This distinction may even prove important in the etiology of clinical syndromes. These particular differences may warrant further investigation.

It would appear from the obtained results that the experiencing of anxiety related greater somatic distress and a concomitant fear of that distress (i.e. anxiety sensitivity) differentiates the panicker from the merely anxious. These conclusions parallel the findings in clinical samples that Panic Disordered patients compared to Generalized Anxiety Disordered patients report more severe somatic symptoms of anxiety (e.g. Hoehn-Saric, 1982; Thyer & Himle; 1987) and have higher ASI scores (Sartory & Olijade, 1988).

Regarding the debate as to whether panic is qualitatively or quantitatively distinct from high anxiety, the present study's data is not particularly helpful. Important differences were found between the anxious nonpanicker and panicker groups when there were no differences on a measure of trait anxiety. This would suggest that a qualitative distinction would be supportable. However, the panicking group also were found to experience more somatic arousal symptoms of anxiety, suggesting a quantitative distinction. It may be the case that had a measure of trait anxiety with a greater somatic component been employed that the results may have been different. Additionally, the notion of resolving a debate between a qualitative and a quantitative distinction via quantitative data seems ill advised. The point at which one concludes a qualitative difference, as opposed to a quantitative difference, is arbitrary.

The present data also serves to indicate the importance of considering trait anxiety as a potential confound when studying panic attacks and associated characteristics. Previous findings suggest that Panic Disordered patients have comparable levels of trait anxiety to Generalized Anxiety Disordered patients (Borden & Turner, 1989). Within the last five years there has been a growing realization in the clinical research that trait anxiety levels must be controlled for. One of the main purposes of this study was to extend this methodological consideration to nonclinical research. Indeed, the different pattern of results obtained with an anxious non-panicking contrast group as opposed to the results obtained by simply using a non-panicking contrast group, underlines the importance of considering trait anxiety levels. Panickers were found to be high on the STAI trait scale, and it would appear that differences between them and nonpanickers can be attributed to both their panic status and their high trait anxiety levels.

#### A Further Examination of the Cognitive Model of Panic

The present findings also are clearly supportive of the contemporary cognitive models of panic (i.e. Ley, 1985; Clark, 1986). The basic tenet of these models is that fear of anxiety symptoms further exacerbates somatic arousal, which further increases the fear, which increases arousal, and so on until panic is experienced. Although, the present

study did not explicitly examine this hypothesis, panickers were found to evidence both increased somatic arousal and the fear of that arousal. Furthermore, an equally anxious comparison group did not evidence comparable characteristics. In other words, the anxious person might not experience panic if their somatic arousal isn't high and they are not particularly fearful of being anxious. Of course, this conclusion begs the question of what creates the high somatic anxiety and the increased anxiety sensitivity. In a three component system it is possible that each may by itself, or in combination with each other be either antecedent or sequelae. Further research should attempt to investigate whether, or not, the existence of high somatic anxiety and high anxiety sensitivity is a precursor of, or a consequence, of panic attacks.

More specifically, future studies may wish to contrast a panic sample with nonpanickers who have comparable somatic anxiety symptoms. If cognitive models of panic are valid, then panickers would be expected to have higher ASI scores even when levels of somatic symptoms are similar. The Beitman, et al. (1987) findings that heart patients experience similar somatic symptoms, but don't panic would suggest that the cognitions, not the somatic distress, are more panic-specific. A potential advantage of using nonclinical subjects with high somatic anxiety in a similar fashion would be that they would not have an available

schemata to explain their symptoms, unlike heart patients (i.e. "the palpitations are expected").

### The Utility of Nonclinical Panic Research

The consistent finding that nonclinical panickers evidence a psychometric profile similar to their clinical counterparts (albeit less severe) was replicated in the present study. Furthermore, the cognitive model of panic attacks borne of clinical research, was substantiated using a nonclinical sample. This consistency would suggest that nonclinical panickers may have utility as an analogue for clinical panickers. However, the nonclinical panicker has some features that limit this proposition. First, the nonclinical panicker has typically not sought therapeutic intervention. Thus, there are potential differences that remain to be clearly understood. Second, previous research (e.g. Norton, et al., 1986) has demonstrated that nonclinical panickers do not evidence significant avoidance behaviour. This would imply that nonclinical panickers are distinct from Agoraphobics in important ways.

The above caveats concerning the limits of the nonclinical panicker as an analogue, lead to the assertion that nonclinical panickers may be appropriate analogues for research which is focussed upon panic specific phenomena. Nonclinical panickers would be inappropriate analogues of clinically disordered patients if the variables under study



were potentially related to degree of avoidance, or the degree of impairment experienced (which one would assume to be significant predictors of clinical status). In other words, if, and only if, the variables of interest are related to panic attacks then nonclinical panickers could serve as a more accessible subject pool. This proposal is substantiated by clinical research which has demonstrated that panic symptomatology and panic frequency are not significantly related to degree of avoidance (Craske & Barlow, 1988; Mavisakalian, 1988).

Perhaps more interesting is the possibility of using nonclinical panickers as a comparison group for clinical panic patients. If the panic-specific phenomena are similar, then any potential differences may prove to be important in the etiological development of the clinical presentation.

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## APPENDIX A

### SUBJECT CONSENT FORM

Simon Fraser University and those conducting this research subscribe to the ethical conduct of research and to the protection at all times of the interests and comfort of subjects. This form contains important information regarding the present project. Please read carefully.

Psychologists have been interested in the phenomena of anxiety for quite some time. Recently, the focus of much research has been directed towards the experience of non-clinical subjects rather than persons with anxiety disorders. Since anxiety is experienced by all, this has been a welcomed change. The present study is designed to allow an evaluation of different forms of anxiety in an university population. Of particular interest are situational anxiety, trait anxiety levels, and panic anxiety. You will be asked to fill out a series of questionnaires dealing with various aspects of the anxiety experience. These questionnaires are not of a clinical nature, and have all been used often with college samples. If you agree to participate, please read the remainder of this form and complete the three lines at the bottom of this page.

Having read the above description of research entitled:

#### THE PHENOMENA OF PANIC

I understand that all of the information that is collected will be kept strictly confidential, and that my name will not be used in the discussion of the data.

I also understand that I MAY REFUSE TO PARTICIPATE or discontinue participation at any point. Furthermore, I reserve the right to withdraw my responses upon completion.

In addition, I may register a complaint about the present research to the principle researcher, John Dorward, university staff, or Dr. Roger Blackman, Chairman of the Department of Psychology, Simon Fraser University.

Copies of the results of this study when completed, may be obtained by contacting John Dorward, Department of Psychology, Simon Fraser University.

I AGREE TO PARTICIPATE IN THE PRESENT STUDY BY COMPLETING THE QUESTIONNAIRES.

Name: \_\_\_\_\_

Signature: \_\_\_\_\_

Date: \_\_\_\_\_