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Automatic and Strategic Processing of Threat Stimuli: A Comparison Between PTSD, Panic Disorder, and Nonanxiety Controls

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This study evaluated 2 hypotheses derived from the theoretical work of A. T. Beck and D. A. Clark (1997). Two anxiety disorder groups, posttraumatic stress disorder (PTSD) and panic disorder (PD), and a nonanxiety control group participated in a modified-Stroop study. The study evaluated whether the diagnostic groups could be differentiated on the basis of responses to stimulus valence and content at different stages of information processing (IP). We found no support for the hypothesis that the diagnostic groups would be sensitive to stimulus valence at automatic stages of IP. Consistent with the second of our 2 hypotheses, the PD group showed delayed vocal responses when processing disorder-specific threat stimuli at strategic stages of IP. The PTSD group showed a generalized valence effect at strategic stages of IP, evincing delayed vocal responses to all stimuli with negative valence. The clinical implications of these findings are discussed, as are directions for future research.

KEY WORDS: PTSD; panic-disorder; information-processing; stroop.

Recent epidemiological evidence suggests that anxiety disorders are among the most common forms of psychopathology (Kessler, 1994). In a search for the etiological and maintaining factors that drive the all too common phenomenon of anxiety, investigators have employed a number of experimental methods. These methods include biological challenge tasks (Sanderson, Rapee, & Barlow, 1989), cue-reactivity psychophysiology studies (Blanchard, Hickling, et al., 1996), and information processing (IP) studies (see Williams, Mathews, & MacLeod, 1996). During the past two decades, the number of studies using IP paradigms in particular has increased dramatically, as theories of anxiety have moved away from stimulus–response models to IP models (Rachman, 1977).

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A core assumption of these IP theories of clinical anxiety is that the manner in which emotional material is processed is crucial to the etiology, maintenance, and treatment of anxiety (Beck & Clark, 1997). Although theories differ, most propose that at the very earliest stages of IP, referred to as automatic stages of processing, anxiety patients are characterized by a sensitivity to all stimuli with a negative valence (see Beck & Clark, 1997; McNally, 1995). In addition, many of these theories assert that, at latter stages of IP, anxiety patients selectively allocate attention toward environmental stimuli that are of central concern to their clinical condition relative to other classes of stimuli (Beck, Emery, & Greenberg, 1985; McNally, 1994).

This paper provides a brief overview of one such theoretical model proposed by Beck and Clark (1997). We then review a series of emotional Stroop paradigm studies that have examined automatic and strategic processing of threat stimuli among anxiety disorder populations. Finally, a study utilizing the emotional analogue of the Stroop paradigm is described, which tested two hypotheses that are generated by Beck and Clark's model, but are yet to be tested with panic disorder (PD) or posttraumatic stress disorder (PTSD) samples.

BECK AND CLARK'S INFORMATION PROCESSING THEORY OF ANXIETY

Stage I Processing

During the first stage of processing in Beck and Clark's model, automatic processing of stimuli occurs. Beck (1996) defines this as an orienting response that is characterized by automatic processing. In fact, Beck and Clark (1997) refer to this response as "automatic processing *par excellence*" (p. 51). They propose that the processing of stimuli occurs without conscious awareness, is involuntary, and requires little attention. This definition is consistent with conventional definitions of *automatic* processing in the experimental-cognitive and social-cognition literatures (Posner & Snyder, 1975). The authors take an evolutionary approach to describing the function of such automatic stimulus appraisal, stating that such processing of threat information is necessary for the survival of the organism. Thus, it has been proposed by Beck and Clark, as well as many others (e.g., Mathews & MacLeod, 1994; McNally, 1995), that at automatic stages of IP, anxiety patients may be overly sensitive to stimuli of negative valence.

Stage II and III Processing

Beck and Clark (1997) propose that after Stage I appraisal identifies a stimulus as threatening, the organism engages in the activation of elaborative processing of such threat-related material that characterizes anxiety (Stage II processing). At this second stage, attentional resources are allocated toward goal directed activities that minimize threat and enhance safety. The activation of this mode of thinking results in increased autonomic arousal, behavioral inhibition/mobilization, and a narrowing of cognitive processing onto the threatening stimulus. Thus, once this process is activated, nearly all attentional resources are allocated toward the threat stimuli and the organism's response to it.

Beck and Clark (1997) propose a third stage of processing that they term "secondary elaboration." At this stage, IP is characteristically slow, effortful, and schema driven. It is at this stage that current concerns and personal issues of the individual are activated and the individual engages in reflective consideration of his/her capacity to cope with the current demands given the context in which events are occurring. Thus, according to the model, Stage III processing is characterized by strategic IP. Their definition of Stage III processing is quite consistent with experimental and social-cognition definitions of strategic processing (Posner & Snyder, 1975).

This theory generates testable hypotheses that can be evaluated with either facilitation or interference paradigms, both of which have their origin in experimental-cognitive psychology. Facilitation paradigms show how a tendency to selectively process emotionally relevant stimuli may facilitate performance on a task that benefits from the processing of such information. Conversely, interference paradigms show how selective processing of emotional stimuli can impair performance on a task where the processing of such information would be disruptive.

Automatic Processing of Threat Stimuli and Clinical Anxiety

According to the aforementioned theoretical model, one can hypothesize that upon early registration of a stimulus with negative valence, those with anxiety disorders will show largely altered processing (as indexed by either facilitation or interference effects) relative to when they are presented with neutral or positive stimuli. Moreover, this pattern of findings should not be present in nonanxiety control groups. We refer to this valence sensitivity at the earliest stages of IP (automatic stages) as the "valence hypothesis."

A series of studies have used the emotional analogue of the Stroop task (Stroop, 1935) with extremely short stimuli presentation times (e.g., 16 ms) and backwards visual masking to evaluate such hypotheses. Presenting the stimuli in such a short period of time in conjunction with a backwards visual mask precludes strategic processing of stimulus-content by the participant and allows for inferences regarding "automatic" processing of stimuli (Williams et al., 1996). The Beck and Clark theoretical model predicts that anxiety disorder patients will show slower vocal response latencies (i.e., reaction times) to threat material relative to neutral material. By way of contrast, nonanxiety control groups should not show differential vocal response latencies as a function of stimulus valence.

To examine automatic processing of threat cues in PTSD, McNally, Amir, and Lipke (1996) compared the vocal response latencies of Vietnam combat veterans with PTSD to veterans without PTSD for trauma words, positive words, neutral words, and color words presented at automatic stages of IP in a masked Stroop task. They found no evidence for differential processing of threat cues as a function of diagnostic group. In contrast, in a study with a motor vehicle accident (MVA) related PTSD sample, Harvey, Bryant, and Rapee (1996) found evidence for differential automatic processing of threat material, using a masked Stroop paradigm.

Two studies with specific phobia have found conflicting evidence, one supporting such effects (van den Hout, Tenney, Huygens, & DeJong, 1997) and one that did not (Thorpe & Salkovskis, 1997). Mogg and colleagues found evidence that individuals

with generalized anxiety disorder or who are high in trait anxiety differentially process threat stimuli relative to neutral or positively valenced stimuli at automatic stages of IP. Such effects were not found for nonanxiety control groups in their studies (e.g., Mogg, Bradley, Williams, & Mathews, 1993; Mogg, Kentish, & Bradley, 1993). This effect has also been obtained in populations high in trait anxiety relative to low trait anxiety under conditions of situational distress (e.g., MacLeod & Rutherford, 1992).

Although not all of the aforementioned studies support the valence hypothesis, most provide some support for the hypothesis that anxiety disorder samples differentially process negatively valenced stimuli at automatic stages of IP relative to nonanxiety comparison samples. However, there are methodological limitations with these studies that make such an interpretation of these results less than definitive. According to the valence hypothesis of Beck and Clark (1997) model, anxiety disorder patients should show this automatic processing bias for all negatively valenced stimuli. Therefore, one would expect all anxiety disorder groups to show similar interference effects for negatively valenced information, regardless of the content of that negative information. Based on the masked presentation studies conducted to date, it is not possible to know whether such automatic processing bias is circumscribed to negative stimuli that are disorder-specific or for all classes of negatively valenced stimuli.

Strategic Processing of Threat Stimuli and Clinical Anxiety

The most widely used experimental paradigm to study the IP mechanisms associated with anxiety disorders is the modified-Stroop paradigm with unmasked presentation times (Williams et al., 1996). This paradigm presents stimuli without a mask. Because the stimuli are not degraded upon presentation, this paradigm is thought to tap both automatic and strategic aspects of IP, whereas the masked presentation studies are thought to tap automatic processing exclusively.

If anxiety patients selectively process disorder-specific threatening stimuli to a greater extent than nonspecific threat stimuli at strategic stages of IP, two predictions can be made when using the Stroop paradigm with anxiety patients and control groups. First, when asked to name the color of disorder-specific threat words, anxiety patients will respond slower than when asked to name the color of nondisorder threat or neutral words. Second, this effect should be disorder-specific. For example, patients suffering from PTSD should display this interference effect with trauma-threat words in contrast to traumatized non-PTSD populations and other anxiety disorder groups (e.g., panic disorder), who should not show such a marked effect for PTSD-related threat stimuli. We refer to this sensitivity to stimulus content (independent of valence) at later stages of IP as the "content-specificity" hypothesis.

Foa, Feske, Murdock, Kozak, and McCarthy (1991) found support for this hypothesis by presenting rape victims with PTSD, rape victims without PTSD, and nontraumatized controls with four classes of stimuli. Results showed that rape victims with PTSD took longer to respond to rape-specific threat words relative to the other three stimulus categories. The two control groups did not show differential responding as a function of word category. Cassiday, McNally, and Zeitlin (1992) replicated this effect with a sample of rape victims. Utilizing virtually identical paradigms,

other investigators have replicated these effects with accident-related PTSD samples (Bryant & Harvey, 1995; Harvey et al., 1996) as well as with samples of combat veterans suffering from PTSD (e.g., McNally, Kaspi, Riemann, & Zeitlin, 1990). Virtually identical findings have been found with PD samples when stimulus words included those that are of central concern to panic patients (e.g., Carter, Maddock, & Magliozzi, 1992; Ehlers, Margraf, Davies, & Roth, 1988; McNally et al., 1994).

These studies suggest that individuals with PTSD and PD evince delayed vocal response times for disorder-associated stimuli at strategic stages of IP. According to the aforementioned theoretical model, this tendency to process disorder-specific information (e.g., somatic concerns for panic disorder patients vs. trauma threat words for PTSD patients) should vary across diagnostic categories in higher-order processing. Similar to the masked Stroop studies, which have examined automatic information processing, many of these studies lacked an anxiety disorder comparison group and/or included only one type of threat word category, disorder-specific threat words. A more stringent test of both the *valence* and *content-specificity* hypotheses would involve a study design with multiple anxiety disorder groups and multiple classes of negative stimuli that are content-specific to each disorder. One would then be able to present the stimuli at different speeds to examine the effect of both stimulus valence and content at different stages of processing. According to the aforementioned model, one would expect the anxiety disorder groups to look similar at automatic stages of IP by showing a processing bias toward all stimuli of negative valence relative to neutral stimuli. At slower presentation rates, one would expect a cross over interaction between the diagnostic groups when processing stimuli that are content-specific to their disorder. Nonanxiety control groups should not show marked variation in vocal reaction time as a function of stimulus valence. No such study has been conducted with PTSD and PD groups.

In the current study we compared two anxiety disorder groups (PTSD & PD) and a nonanxiety control group on vocal response latencies for three types of stimuli: categorized neutral words, PTSD-specific threat words, and panic-specific threat words. The stimuli were presented in both a masked and unmasked condition. This allowed us to test the hypothesis that these two anxiety disorder groups are sensitive to stimulus valence at the earliest stages of IP. The design also allowed us to assess for content-specific effects at later stages (strategic) of IP.

Hypothesis #1

We predicted that the two anxiety disorder groups would show delayed vocal response latencies for all stimuli with negative valence ratings relative to neutral stimuli in the masked presentation condition and that the control group would not.

Hypothesis #2

We hypothesized that in the unmasked condition, the PTSD group would show the greatest delays in vocal response latencies when asked to name the color of trauma-specific threat stimuli relative to neutral words and panic-specific threat stimuli. Likewise, we expected that the panic disorder group would demonstrate the greatest delay in vocal response latencies for panic-specific threat stimuli. The

control group was not expected to show significant differences in vocal response latencies as a function of word type.

METHOD

Design

The design of this study was a 3 (group: PTSD, PD, nonanxiety control) × 3 (word-type: PTSD-specific, panic-specific, categorized neutral-1/categorized neutral-2) × 2 (presentation-time: 16 ms masked vs. unmasked) mixed-factorial, with repeated measures on the last two factors. The primary dependent variable was vocal response time, measured in milliseconds.

Participants

PTSD Group

This group consisted of 30 survivors of severe MVAs who were 6–24 months post-MVA. We defined severe MVAs as those in which at least one passenger in the vehicle required professional medical attention as a direct result of the accident. This group was recruited through self-referral, response to local media advertising, and referral from the medical community. Accident victims who suffered a closed head injury (evidenced by loss of consciousness) as a result of their MVA were excluded from the study. In addition, MVA survivors with comorbid panic disorder, substance abuse, or psychotic disorders were excluded.

Panic Disorder (PD) Group

The PD group ($n = 30$) was recruited through local media advertising. Panic disorder patients with a history of PTSD subsequent to MVAs were excluded. In addition, participants whose panic attacks were situationally predisposed to motor vehicles or other driving-related situations were excluded. Individuals with a history of serious head injury, current substance abuse, and/or a current psychotic disorder diagnoses were also excluded.

Nonanxiety Control Group

The nonanxiety control group ($n = 30$) was recruited through local media advertising. Participants with any current anxiety disorder, history of panic disorder, and/or MVA-related PTSD were excluded from this group. Participants with a history of serious head injury, current substance, and/or a current psychotic disorder diagnosis were also excluded.

Measures

The Structured Clinical Interview for *DSM-IV* (SCID-I; First, Spitzer, Gibbon, & Williams, 1996) was used to determine the presence of all lifetime and current

Axis-I diagnoses for all three groups. The MVA group received The Clinician Administered PTSD Scale (CAPS; Blake et al., 1997) in order to determine if they met *DSM-IV* diagnostic criteria for PTSD. Previous work from our laboratory found interrater reliability on the CAPS, as assessed by the Kappa statistic, to be .84 (Blanchard, Jones-Alexander, Buckley, & Forneris, 1996).

To assess the overall level of PTSD symptoms, the MVA group was administered the PTSD Checklist (PCL; Weathers, Litz, Herman, Huska, & Keane, 1993) and the Impact of Event Scale (IES; Horowitz, Wilmer, & Alvarez, 1979). All three groups were administered the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961), State-Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, & Lushene, 1970), and the Brief Symptom Inventory (BSI; Derogatis & Melisaratos, 1983) to quantify depressive symptoms, anxiety symptoms, and overall level of psychopathology respectively. The National Adult Reading Test-Revised (NART-R; Blair & Spreen, 1989) was administered to all participants in order to determine the verbal ability of the patients in each group. We also gathered basic demographic information on all individuals including age, gender, ethnicity, and highest level of educational achievement. The demographic information on the diagnostic subgroups can be found in Table I.

Stimulus Materials

There were four categories of words. There were two sets of categorized neutral stimuli, each consisting of eight semantically related words [tools (Screwdriver, Hammer, Wrench, Toolbox, Pliers, Saw, Crowbar, Nail) and musical instruments (Rattle, Cornet, Bagpipe, Piano, Harp, Banjo, Clarinet, Guitar)]. Using semantically related words for the neutral word categories controlled for semantic priming effects. Using two sets of categorized neutral words allowed the frequency of neutral and threat stimuli presentation to be identical. The panic-specific threat stimuli included words that previous studies have demonstrated to be of central concern to panic disorder patients (Heart attack, Faintness, Breathless, Shaky, Dizzy, Collapse, Insane, Heartbeat). These stimuli have produced delayed vocal response times relative to neutral words with panic populations in previous studies. Similarly,

Table I. Demographic Characteristics of Subsamples

Variable	Diagnostic group		
	PTSD ($n = 30$)	Panic disorder ($n = 30$)	Nonanxiety ($n = 30$)
Age[a]	40.0[a] (12.6)	39.5[a] (12.4)	31.4[b] (11.9)
Years of education[a]	14.2[a] (2.2)	13.7[a] (2.4)	15.9[b] (2.0)
NART-R[a]	38.2[a] (11.6)	31.0[b] (12.9)	39.4[a] (10.3)
Gender (% M/F)	23/77[a]	33/67[a]	33/67[a]
Ethnicity (% Caucasian/ minority)	87/13[a]	90/10[a]	90/10[a]

Note. Means that do not share a superscript (within rows) are statistically different based on Tukey-HSD or chi-square tests (i.e., $p < .05$). NART-R = National Adult Reading Test-Revised. PTSD = Posttraumatic stress disorder.
[a]Values represent *M* (*SD*).

the MVA-PTSD stimuli (Highway, Accident, Smash, Scream, Accelerate, Mutilated, Emergency, Trapped) included words from previous studies that have demonstrated that these threat words are specific MVA-related PTSD concerns and have produced delayed vocal response times relative to other classes of stimuli on modified Stroop tasks with MVA-related PTSD populations (Bryant & Harvey, 1995; Harvey et al., 1996). A one-way analysis of variance (ANOVA) on a frequency of occurrence measure indicated that these word groups do not differ in their frequency of usage in the English language, $F(3, 28) = .62$, $p = .61$ (Carroll, Davies, & Richman, 1971). A one-way ANOVA revealed that the word groups did not differ in number of characters per word, $F(3, 28) = 1.97$, $p = .14$.

Each word appeared eight times, twice in each of four colors: green, blue, white, and red. The words appeared an equal number of times in both the masked and unmasked conditions, yielding 256 total trials. The words were presented in a fixed-randomized format with the following constraints: no two words from the same category appeared consecutively, nor did a color appear twice on consecutive trials. The order of presentation was determined through use of a random number table. The valence for each word stimulus was rated by participants with a series of 100 mm visual analogue scales with anchor points of 0 (*extremely negative*), 50 (*neutral*), and 100 mm (*extremely positive*).

Apparatus

An IBM computer with a 100 MHz Pentium processor presented the stimuli to the participants and recorded their vocal response latency on each trial. MEL Professional software (version 2.0d) was used to program the experiment. The stimuli appeared in 1-in. capitalized block letters in the center of a 14-in. Magnavox color monitor. A voice-activated relay connected to a Software Tools 200A-Psychology serial response box was used to detect the onset of vocal responses.

Procedure

When prospective participants contacted the clinic, a brief phone interview was conducted. After a phone screen contact indicated that subjects might be eligible for the study, they were mailed the self-report questionnaires prior to their first appointment for the structured clinical interviews. Following the structured interview session, the subjects were asked to report back to the clinic approximately 1 week later for Stroop testing. In this way, semantic and emotional priming effects for disorder-specific threat words were minimized (as opposed to running the IP task immediately after the clinical interview). The Stroop task was described as one that tests concentration. Participants were seated in a sound attenuated room approximately 18 in. from the computer monitor. All participants wore a headset microphone to activate the vocal response relay. The experimenter sat approximately 3 ft. behind the subjects so that errors in responding could be recorded.

Stimulus words were preceded by a white fixation cross, which appeared in the middle of the computer screen at the same spot where the word stimuli appeared. The words in the unmasked condition were presented until a vocal response was

recorded. In the masked condition, words were presented for 16 ms and followed by a backward visual mask of letter strings, of the same character length and color. This mask remained on the screen until a vocal response was recorded. The interstimulus interval was 2 s.

Prior to the experimental trials, subjects responded to 10 practice trials with words unrelated to the experimental word categories (e.g., One, Two, Three). Provided the participants understood the task, the 256 experimental trials were conducted with a short break after trial number 128. The purpose of the break was to counteract fatigue effects.

Data Analytic Plan

The omnibus statistical analysis used to test the hypotheses involved a 3 (group) × 3 (word-type) × 2 (presentation time) mixed-factorial analysis of variance (ANOVA) with repeated measures on the last two factors. Because there were no theoretical reasons to predict differences in response latencies among the neutral word groups, the mean vocal response latencies for the two neutral word groups were averaged into a single value (recall two neutral word groups were used only to control for frequency of occurrence of neutral and threat stimuli). These two neutral word groups did not differ on valence ratings or error responses (see Results). The primary dependent variable was mean vocal response time. Consistent with previous research in this area, vocal response times of less than 300 ms were excluded from the analyses (indicative of instances when the voice-activated relay was tripped prior to emission of a vocal response). In addition, vocal response times of greater than 2000 ms were excluded (indicative of vocal responses that were too soft to be detected by the voice activated relay). Mean latency scores were computed for each word category by taking the average value of the response time for all words in that category. These word group means were computed separately for both the unmasked and masked presentation conditions. Because reaction time data are typically skewed in a positive direction (Heathcote, Popiel, & Mewhort, 1991), the data were log-transformed to approximate a normal distribution.

The omnibus test was followed by a set of a priori split-plot ANOVAs. To test our first hypothesis, we conducted a 3 (group) × 3 (word-type) split-plot ANOVA on vocal response latencies within the masked presentation condition. A statistically significant interaction effect was predicted resultant from group mean differences as outlined by Hypothesis #1. In order to test our second hypothesis, the following test was conducted within the unmasked condition: a 3 (group) × 3 (word-type) split-plot ANOVA on vocal response latencies. A statistically significant interaction effect was predicted resultant from group mean differences as outlined by Hypothesis #2.

RESULTS

Demographic Variables

Demographic variable values and a synopsis of the corresponding analyses can be found in Table I. Chi-square analyses were conducted on nominal demographic

Table II. Group Mean Values on Self-Report Measures of Psychopathology

Variable	Diagnostic group: PTSD ($n = 30$)	Panic disorder ($n = 30$)	Nonanxiety ($n = 30$)	ANOVA statistics: df^a	F
BDI	23.7^a (9.9)	16.2^b (9.4)	3.8^c (3.4)	2, 87	46.3***
State Anxiety	57.6^a (12.3)	49.8^b (13.8)	31.0^c (8.9)	2, 85	39.0***
Trait Anxiety	55.0^a (10.9)	53.0^a (11.0)	33.3^b (8.5)	2, 87	41.7***
BSI-PSI	0.28^a (0.14)	0.29^a (0.15)	0.07^b (0.06)	2, 85	29.8***

Note. Means that do not share a superscript (within rows) are statistically different based on Tukey-HSD tests (i.e., $p < .05$). PTSD = Posttraumatic stress disorder. BDI = Beck Depression Inventory. BSI-GSI = Brief Symptom Inventory-Positive Symptom Index.
[a]Degrees of freedom vary because of missing data.
***$p < .001$.

variables and one-way ANOVA analyses were conducted on continuous demographic variables. Statistically significant ANOVAs were followed by Tukey-HSD follow-up tests.

Self-Report Psychopathology Measures

The self-report psychopathology measures were examined for group differences by way of a multivariate ANOVA (MANOVA). The four measures that went into the MANOVA analysis were the BDI, State-Anxiety Inventory, Trait-Anxiety Inventory, and BSI-Global Severity Index. The Wilks' Lambda statistic value (.34) was statistically significant, $F(8, 160) = 14.4$, $p < .01$, indicating group mean differences on at least one of the measures. To determine the nature of the differences, this test was followed by one-way ANOVAs with Tukey-HSD follow-up tests for each of the aforementioned measures. The results of the one-way ANOVAs are presented in Table II.

Stimulus Valence Rating Analyses

The valence ratings for each word category were examined as a function of diagnostic group and are presented in Table III. A group (3) × word-type (4) split-plot ANOVA analysis with word-type as the repeated measure was conducted. Based on Mauchly's Test of Sphericity, the sphericity assumption for the repeated measure

Table III. Valence Ratings for Word Stimuli as a Function of Word-Type and Diagnostic Status[a]

Diagnostic group	Word stimulus category: Neutral-1	Neutral-2	PTSD-specific	Panic-specific	Group marginal means
Control	71	62	37	39	52^a
PD	62	55	30	35	46^b
PTSD	67	55	30	23	44^b
Word marginal means	67^a	57^b	32^c	33^c	—

Note. Means that do not share a superscript (within rows or columns) are statistically different (i.e., $p < .05$). PTSD = Posttraumatic stress disorder.
[a]Values have been rounded to nearest whole number.

was violated, $W = .247$, $\chi^2(5) = 117$, $p < .01$. Therefore, Huynh-Feldt Epsilon corrected degrees of freedom were used to examine repeated-measure effects (Glass & Hopkins, 1996). There was a main effect of word, $F(1.7, 143) = 117.4$, $p < .001$. There was also a main effect of group, $F(2, 85) = 5.49$, $p = .006$. Importantly, the Group × Word-Type interaction was not statistically significant, $F(3.36, 143) = 1.7$, $p = .12$.

Vocal Response Reaction Time and Error Analyses

Errors for vocal responses were examined using a 3 (group) × 3 (word-type) × 2 (masking condition) mixed-factorial ANOVA with repeated measures on the last two factors. Because of sphericity violations, Huynh-Feldt Epsilon corrected degrees of freedom were used to evaluate all repeated measure effects. The main effects of group, word-type, and masking condition were all nonsignificant. In addition, all two-way and three-way interactions for errors were nonsignificant.

The main analysis of this study involved a 3 (group) × 3 (word-type, with the two neutral word categories collapsed) × 2 (masking condition) mixed-factorial ANOVA on log-transformed vocal reaction time scores. A chi-square evaluation of Mauchly's test of sphericity indicated that both the *word* and *word by mask* variables met the assumption of sphericity. Therefore, Huynh-Feldt corrected degrees of freedom were *not* used for these analyses. There were main effects of masking condition, $F(1, 87) = 75.3$, $p < .01$, word type, $F(2, 174) = 9.3$, $p < .01$, and group, $F(2, 87) = 8.6$, $p < .01$. There was also a two-way interaction between group and masking condition, $F(2, 87) = 5.8$, $p < .01$, and a marginally significant interaction between word-type and diagnostic group, $F(4, 174) = 2.2$, $p = .07$. The three-way interaction between group, word-type, and masking condition approached statistical significance, $F(4, 174) = 2.0$, $p = .10$.

The groups varied on years of education (see Table I). Thus, we repeated the omnibus 3 × 3 × 2 ANOVA with years of education as a covariate in an analysis of covariance (ANCOVA) to ensure that our results involving the between-group variable (i.e., diagnostic group membership) were not due to an artifact of group mean differences on this variable. In accordance with the findings above, there continued to be a main effect of group, $F(2, 85) = 8.7$, $p < .001$. In addition, there was a Group × Word-Type interaction, $F(4, 170) = 2.4$, $p = .05$, a Masking Condition × Group interaction, $F(2, 85) = 4.6$, $p = .01$, and a marginally significant three-way interaction between word, masking condition, and group, $F(4, 170) = 2.1$, $p = .09$. For similar reasons, we ran an ANCOVA with age as the covariate. In accordance with the above findings, there continued to be a main effect of group, $F(2, 85) = 7.0$, $p < .001$. In addition, there was a Group × Word-Type interaction, $F(4, 170) = 2.5$, $p = .04$, a Masking Condition × Group interaction, $F(2, 86) = 3.9$, $p = .02$, and a marginally significant three-way interaction between word-type, masking condition, and group, $F(4, 172) = 2.3$, $p = .10$. Given the uniformity of effects between the omnibus ANOVA and ANCOVA, we proceeded to examine the nature of the interactions with planned contrasts as outlined by our hypotheses.

To examine the nature of the interactions, the a priori 3 (group) × 3 (word-type) split-plot ANOVAs with repeated measures on the word-type factor were

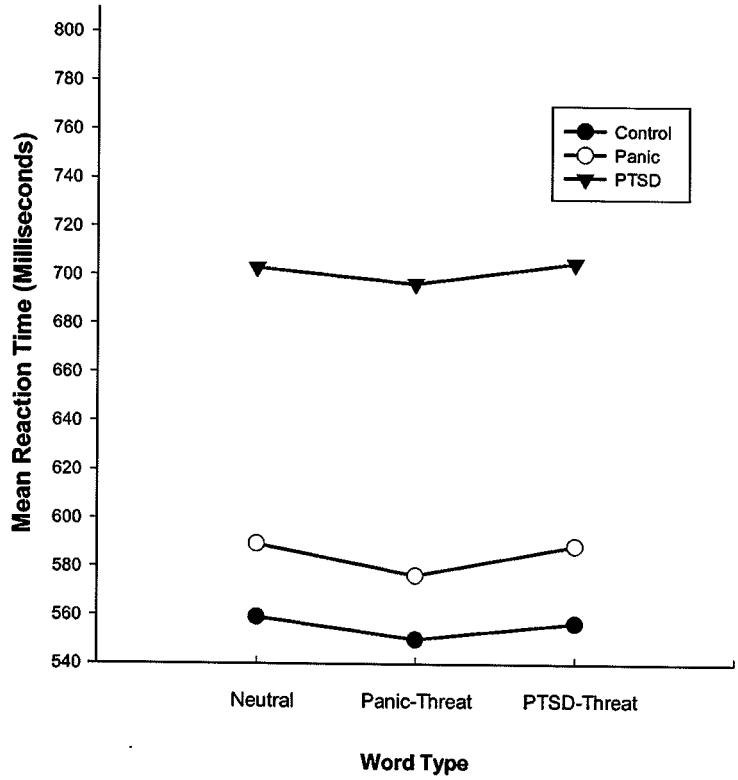

Fig. 1. Reaction time data for masked presentation condition.

conducted within each level of the presentation condition (masked vs. unmasked). The group × word-type ANOVA in the masked presentation condition revealed a main effect of group, $F(2, 87) = 7.7$, $p = .001$. The vocal response latencies in the masked condition are presented in Fig. 1 as a function of diagnostic group status and word-type (values in figure are untransformed data). Pairwise comparisons revealed that the PTSD group was slower in overall reaction time than both the panic disorder and nonanxiety control groups, who did not differ from each other. There was also a main effect of word, $F(2, 174) = 7.9$, $p = .001$. Pairwise comparisons revealed that across all groups, subjects generally showed quicker reaction times to the panic words, relative to the other word categories, which did not differ from each other. Contrary to our prediction, the Group × Word-Type interaction test was not statistically significant in the masked presentation condition, $F(4, 174) = .38$, $p = .82$. Therefore, no simple main effects or interaction contrasts with the masked condition data were conducted.

The group (3) × word-type (3) split-plot ANOVA within the unmasked presentation condition revealed a different pattern of results than that in the masked condition. The vocal response latencies in the unmasked condition are presented in Fig. 2 as a function of diagnostic group status and word-type. There was a main effect of group, $F(2, 87) = 9.27$, $p < .001$. There was also a main effect of word-type,

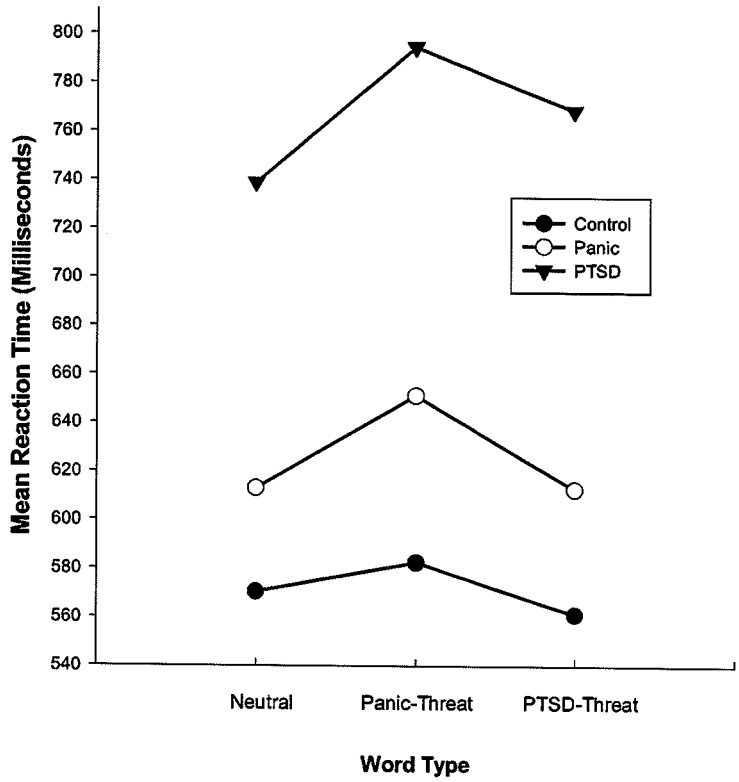

Fig. 2. Reaction time data for unmasked presentation condition.

$F(2, 174) = 27.74$, $p < .001$. The Group × Word-Type interaction was statistically significant, $F(4, 174) = 2.72$, $p = .03$. In order to examine whether the Group × Word-Type interaction was consistent with our hypotheses, we conducted one-way repeated measures ANOVAs on word-type within each diagnostic group (simple-main effect of word-type). The effect of word-type was statistically significant within the panic disorder group, $F(2, 58) = 19.1$, $p < .001$. The differences in vocal response times across word-types in the panic disorder group were in the predicted directions. Follow-up paired t tests indicated that this group was slower to respond to panic-threat stimuli relative to both neutral word, $t(29) = 4.5$, $p < .01$, and PTSD threat stimuli, $t(29) = 7.0$, $p < .01$. This group did not differ on the latter two word-types, $t(29) = .54$, $p = .59$. There was also a statistically significant effect of word-type within the PTSD group, $F(2, 58) = 8.7$, $p = .001$. Follow-up paired sample t tests indicated that this group was slower to respond to PTSD-threat stimuli than to neutral stimuli, $t(29) = 1.9$, $p = .07$. They were also slower to respond to the panic-threat stimuli relative to both the neutral, $t(29) = 3.7$, $p < .001$, and PTSD-threat stimuli, $t(29) = 2.5$, $p = .017$. Thus, the PTSD group showed delayed vocal response times to both threat-word categories and did not evince the expected content-specificity effect. The effect of word-type was significant within the control group,

$F(2, 58) = 7.89$, $p < .001$. Follow-up paired sample t tests indicated that the control group evidenced faster responses when presented with PTSD threat words relative to the panic-threat words, $t(29) = 3.5$, $p < .01$, and neutral words, $t(29) = 2.4$, $p < .02$.

Because the diagnostic groups were widely discrepant on measures of depression, which is known to influence appraisal of threat stimuli (see Williams et al., 1996), we reran some of the aforementioned analyses with the BDI as a covariate. The two planned interaction tests of group (3) × word (3) within each level of the masking condition factor were repeated with the BDI as a covariate. Similar to the initial ANOVA analyses, the Group (3) × Word-Type (3) interaction was nonsignificant in the masked condition when the BDI was used as a covariate, $F(4, 172) = .95$, $p = .44$. Also consistent with the preliminary set of ANOVA analyses, the Group (3) × Word-Type (3) interaction within the unmasked condition remained marginally significant with the BDI utilized as a covariate, $F(4, 172) = 2.0$, $p = .09$. To follow-up this Group × Word-Type interaction within unmasked condition, simple main effect of word-type was examined within each diagnostic group, utilizing the BDI as covariate. The main effect of word-type in the PTSD group remained in the same direction, but the effect was slightly attenuated, $F(2, 56) = 2.6$, $p = .08$. Likewise, the main effect of word-type remained significant within the panic disorder group after covarying out BDI scores, $F(2, 56) = 6.8$, $p < .01$. For the control group, the word-type main effect was not statistically significant. Thus, the main effects of the ANCOVA analyses revealed similar, though slightly smaller, effects relative to the ANOVA examinations of reaction time data.

In a final examination of the effect of depression on the vocal reaction time within the PTSD group, we split the PTSD group as a function of whether they met the *DSM-IV* diagnostic criteria for depression ($n = 16$) versus did not meet criteria ($n = 14$). We ran a 2 (group: depressed vs. nondepressed) × 3 (word-type) ANOVA on reaction time scores. The main effect of word-type was significant, $F(2, 56) = 8.24$, $p = .001$, as both groups took longer to respond to threat words than to neutral words. The main effect of group did not reach statistical significance, $F(1, 28) = 2.26$, $p = .14$, nor did the Group × Word-Type interaction, $F(2, 56) = .18$, $p = .65$.

DISCUSSION

Hypothesis #1

We found no evidence that PD or PTSD subjects show a sensitivity to stimulus valence at automatic stages of IP. One might conjecture that our lack of findings was due to our "threat" stimuli not being perceived by subjects as negatively valenced. However, the stimuli used to test this first hypothesis have been shown to produce interference effects at later stages of IP in other studies (e.g., Bryant & Harvey, 1995). These studies found that the clinical groups under question rate the disorder-specific threat stimuli as threatening and negative relative to other word stimuli. In addition, our ANOVA examinations of the valence ratings for the stimuli indicate that the valence manipulation worked for each group in the study (see Table III).

Furthermore, the lack of a Group × Word-Type interaction on valence ratings indicated that the hedonic valence for each stimulus category did not vary across diagnostic group. Taken together, these findings suggest that the lack of a Group × Word-Type interaction in the masked presentation condition is not likely due to a failure of the subjects to perceive the "threat" stimuli as negative relative to the "neutral" stimuli.

There is the possibility that our results were due to low statistical power. However, our sample was larger than that employed by most emotional-Stroop studies (see Williams et al., 1996). We also employed a repeated-measure design to increase power. Although low power remains a plausible explanation for our null results, our review of the literature would suggest that the sample size and repeated-measure design should have been adequate to detect an automatic processing effect. In total, we conclude that this study does not support our first hypothesis that PTSD and panic disorder samples are sensitive to stimulus valence at the earliest stages of IP.

It is worth noting that the majority of studies that *have* found evidence of an automatic processing bias toward negatively valenced stimuli have done so with populations who were suffering from GAD, or high trait anxiety. Moreover, subjects in these studies often performed the Stroop task under elevated state anxiety from environmental or laboratory induced stress (e.g., MacLeod & Rutherford, 1992). Given that GAD patients are, by definition, characterized by elevated levels of basal physiological arousal, one might argue that individuals are sensitive to the predicted valence effects at automatic stages of IP only during states of increased autonomic nervous system arousal. We took care to ensure that our subjects were not emotionally primed prior to completing the Stroop task. Our results may have been in the predicted direction had we increased autonomic arousal by exposing participants to fear stimuli prior to the IP task (e.g., imagery-based exposure, biological challenge, etc.). It may be that cue-reactive samples such as PTSD and panic disorder only show the predicted valence effects under conditions of stimulus-cued autonomic arousal. Indeed, there is a substantial amount of evidence that elevations in autonomic arousal potentiate "automatic" peripheral and central reactivity to acoustic startle tones in both animal and human populations (Lang, 1995; LeDoux, 1990). Thus, it may also be the case that automatic IP is moderated by basal differences in central and peripheral nervous system arousal across diagnostic subtypes. We revisit this point shortly.

Hypothesis #2

Consistent with previous research, the predicted pattern of content-specific results was obtained within the PD group in the unmasked condition. They did not show a generalized delay in vocal response times to all negatively valenced stimuli, only those that were of central concern to their clinical condition. This pattern of findings is quite consistent with previous research that has examined strategic stages of IP of threat stimuli among panic populations (Ehlers et al., 1988; McNally et al., 1994).

The PTSD group also showed delayed vocal response times for disorder-specific threat words relative to neutral words. However, unlike the PD group, the PTSD group also showed delayed vocal response times for non-PTSD threat words. Thus,

the PTSD group showed a more generalized effect than did the PD group, in that processing any negatively valenced stimulus impaired their performance on the primary task, naming the color of word stimuli.

One possible interpretation of this pattern of results is that at strategic levels of IP, individuals with PD are characterized by cue-specific reactivity to threat stimuli. By way of contrast, individuals with PTSD might be characterized by a more generalized tendency to selectively focus on any negatively valenced stimuli regardless of its direct relevance to their clinical condition. A possible alternative explanation to that offered above is that some of the PD words were tapping trauma-related content. Given that many of the words had a "somatic" theme, it may have been the case that individuals who were severely injured in their accident and were dealing with the medical sequelae of injuries were responding to such words in a slower fashion. Alternatively, the tendency of the PTSD group to show biased processing toward stimuli with somatic content may be reflective of that group being high on the underlying trait of *anxiety sensitivity*, a phenomenon that has been noted by other investigators who have studied PTSD samples (see Taylor, Koch, & McNally, 1992).

There is one major limitation with our sample that may confound the above interpretations of our study, specifically, the differences in depression between the groups. As can be seen in Table II, the PTSD group has high levels of depression relative to the two comparison groups, as evidenced by scores on the BDI. They score almost a full standard deviation higher than the PD group. There is substantial evidence that individuals suffering from major depression selectively process negatively valenced information relative to neutral or positively valenced information at strategic stages of IP (Gotlib & McCann, 1984; see also Williams et al., 1996). Therefore, it is possible that the effect we see in our PTSD group is due to the presence of comorbid depression rather than something unique to PTSD. However, our post hoc analyses of vocal response latencies with depression as a covariate were not markedly different than any of the ANOVA analyses suggesting that depression could not account for our findings.

Because the aforementioned effects were found in the unmasked, but not masked, condition the data are consistent with the notion that the Stroop interference effects seen in both PD and PTSD populations are at least partly due to postrecognition cognitive processes that interfere with the response execution of the primary task of naming the color of stimuli. To the extent that this conclusion is correct, there are clinical implications for the treatment of PTSD and PD. It has been suggested that if the cognitive processes involved in stimulus appraisal are automatic in nature, then cognitive therapy, which is designed to help patients assume control over voluntary (strategic) cognitive processes, may not be up to task for the treatment of anxiety (McNally, 1995). Such a condition would necessitate exposure-based methods of treatment that are designed to extinguish automatic responses to threat stimuli. Our data suggest that the Stroop interference effects seen with these two clinical populations are at least, in part, mediated by strategic processing that may be amenable to cognitive interventions (Beck & Clark, 1997). This would be consistent with recent data that suggest that cognitive strategies devoid of in-vivo exposure alleviate the symptoms of both PD patients (Bouchard et al., 1996) and PTSD patients (Calhoun & Resick, 1993). We should note, however, that our sample was

disproportionately female and Caucasian. Thus, the generalizability of our conclusions to other samples awaits further investigation.

Areas for Future Investigation

Interpretive problems can arise from the use of single paradigms within a study because most, if not all, IP paradigms involve the use of both automatic and strategic processing by study participants (Jacoby, 1991). However, much of the research reviewed here has been conducted under the assumption that particular tasks are pure measures of a particular process (e.g., automatic processing). Because it is assumed that the measures are "process pure," performance on those measures is taken to be a true indicator of either automatic or strategic processing. This assumption may be erroneous and we suggest that future studies employ multiple paradigms under varying degrees of emotional priming and divided attention. This will more effectively disentangle the relative contributions of automatic and strategic processes by examining differences in process (automatic vs. strategic) rather than differences among IP tasks (Jacoby, 1991).

The effect of emotional priming on automatic processing of threat stimuli in PTSD or PD populations is an area that warrants future investigation. We speculate that if subjects were indeed in a state of autonomic arousal prior to undergoing tests of automatic processing, they may show the predicted patterns of results. The studies that have found evidence for automatic processing of negatively valenced stimuli have done so with clinical groups that were characterized by elevated autonomic arousal (Bradley, Mogg, Millar, & White, 1995; Mogg, Bradley, et al., 1993). Indeed there is a substantial amount of empirical literature that would suggest that priming normal or clinical populations into a state of negative affect with exposure to negatively valenced stimuli potentiates automatic responses such as the startle reflex (Lang, 1995). Studying the effects of fear-induced patterns of arousal on IP may reveal different patterns of results for automatic and strategic IP mechanisms. For example, recent studies have shown that fear-induced arousal attentuates the Stroop effect in unmasked presentation studies (Mathews & Sebastian, 1993).

The experimental paradigms reviewed here have been shown useful in studying the IP mechanisms associated with PTSD, PD, and other Axis-I conditions. The literature provides provisional evidence to support some aspects of the current IP theories of anxiety but not others. The hypotheses regarding automatic processing of threat stimuli did not receive support in this study. However, the hypotheses regarding strategic processing received partial support. The findings of the summarized literature address some interesting theoretical questions; however, the clinical utility of these findings and the application of these paradigms to clinical settings await future investigation.

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