The impact of pretrauma analogue GAD and posttraumatic emotional reactivity following exposure to the September 11 terrorist attacks: A longitudinal study

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Abstract

The relation between analogue generalized anxiety disorder (GAD) assessed the day before the events of September 11, 2001 (9/11), and long-term outcome was examined in 44 young adults who were directly exposed the following day to the terrorist attacks in New York City. After controlling for high exposure to the attacks, preattack analogue GAD was associated with greater social and work disability, loss of psychosocial resources, anxiety and mood symptoms, and worry, but not symptoms of posttraumatic stress, assessed 12 months after 9/11. Fear and avoidance of emotions assessed 4 months after 9/11 statistically mediated the relation between preattack analogue GAD and social and work disability, loss of psychosocial support, mood and anxiety symptoms, and worry at 12-month follow-up. Avoidance of emotions 4 months after 9/11 also statistically mediated the relation between preattack analogue GAD and posttraumatic stress symptoms 12 months after 9/11.

Keywords: September 11, emotion regulation, disaster, generalized anxiety disorder, trauma
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On September 11, 2001 (9/11), millions watched in horror as terrorists crashed commercial aircraft into the World Trade Centers (WTC) in New York City. Nearly 3,000 people were killed in the attacks, and the lives of many more were severely affected. In the days and weeks that followed, Manhattan residents faced widespread property destruction and social and economic upheaval, generating considerable concern about the mental health consequences of a disaster of this type and scale (Fullerton, Ursano, Norwood, & Holloway, 2003; Galea et al., 2002; Schlenger et al., 2002). Indeed, among adults directly affected by terrorist events (including those on 9/11), the prevalence of posttraumatic stress disorder (PTSD) in the year following the event is estimated to be 12%-16% according to a recent meta-analysis (DiMaggio & Galea, 2006). In Manhattan, random-digit-dialing telephone surveys of adults living in households south of 110th Street conducted 1, 4, and 6 months after 9/11 found that the prevalence of current (30-day) probable posttraumatic stress disorder (PTSD) declined from 7.5% to 1.7% to 0.6%, respectively (Galea et al., 2003). Within the first 2 months, proximity to the WTC attacks, defined geographically as living south of Canal Street (several blocks from the WTC), was associated with higher prevalence (20.0%) of current probable posttraumatic stress disorder (PTSD) compared to those living between Canal Street and 110th Street (6.8%; Galea et al., 2002).

Most individuals exposed to major disaster experience symptoms of acute distress that abate within several months. Although the vast majority of individuals exposed to disaster or other trauma do not develop diagnosable PTSD (DiMaggio & Galea, 2006; Norris, Friedman, Watson, Byrne, Diaz, & Kaniasty, 2002; North & Pfefferbaum, 2002; Rubonis & Bickman, 1991), research has consistently identified several individual-level moderators of vulnerability to posttraumatic stress and global dysfunction following a traumatic event (see Norris et al., 2002, for a review). For example, predisaster functioning and psychopathology have repeatedly predicted postdisaster functioning (Katz, Gluck,
Maurizio, & DeLisi, 2002; La Greca, Silverman, & Wasserstein, 1998; Norris et al., 2002; Weems et al.,
2007).

Previous research suggests that generalized anxiety disorder (GAD) and related constructs (e.g.,
anxiety, worry, neuroticism) are associated with increased risk for posttraumatic stress and generalized
anxiety following exposure to trauma or disaster. For example, children’s pretrauma trait anxiety was a
significant predictor of symptoms of posttraumatic stress and generalized anxiety after controlling for
exposure to Hurricane Katrina (Weems et al., 2007). Combat-exposed Vietnam War veterans with
precombat GAD were nearly three times more likely to develop PTSD than were veterans without
precombat GAD (Koenen et al., 2002). A study of children exposed to Hurricane Andrew found that
preexisting anxiety was associated with higher severity of posttraumatic stress symptoms 3 to 7 months
after the hurricane after controlling for demographic and hurricane exposure variables (La Greca et al.,
1998). In addition, personality traits associated with anxiety, such as neuroticism (Holtzman, Calvin, &
Bitterman, 1952), and symptoms that are not specific to GAD, such as worry (Sexton, Norton, Walker, &
Norton, 2003), have also been associated with increased risk for the occurrence and chronicity of
postdisaster psychiatric morbidity (Carr et al., 1997; Lewin, Carr, & Webster, 1998; McFarlane, 1989).

Considerable empirical support has emerged for a model of worry as a cognitive mechanism for
avoiding distressing emotional experiences, particularly aversive imagery and somatic arousal (see
Borkovec, Alcaine, & Behar, 2004). Specifically, Borkovec and colleagues have found that worry is
followed by reductions in physiological responsiveness and aversive imagery (Borkovec & Hu, 1990;
Borkovec, Lyonfields, Wiser, & Diehl, 1993). They speculate that this operantly reinforced feedback
loop may become aversive and uncontrollable for the worrier, and thus may be a critical mechanism in the
development and maintenance of worry and GAD (Borkovec et al., 2004). Furthermore, evidence
suggests that the extent to which individuals report having difficulty controlling their worry discriminates
high worriers with GAD from high worriers without GAD (Ruscio, 2002).

Building on Borkovec and colleagues’ (2004) avoidance perspective on worry, Mennin and
colleagues (Mennin et al., 2005) have provided empirical support for an emotion dysregulation model of
GAD. Individuals with GAD report heightened emotional intensity, difficulty understanding or labeling their emotions, increased negative reactivity to emotional experience, and poor management of emotions compared to nonanxious controls both dispositionally and following a negative mood induction (Mennin et al., 2005). The negative emotional reactivity component suggested that individuals with GAD react more strongly to their emotional responses than do nonanxious controls. In this model, negative emotional reactivity is closely linked to fear of emotions and their consequences (see Williams, Chambless & Ahrens, 1997). In addition, poor management of emotions often involves the use of cognitive or behavioral strategies that impair the processing of emotional experiences (e.g., emotional avoidance). This component overlaps with the experiential avoidance construct, which involves an unwillingness to experience one’s emotions, thoughts, sensations, and other internal experiences (Hayes et al., 2004). Individuals with GAD may react negatively to their emotions, particularly to fear and anxiety, because they experience them intensely and do not understand their motivational significance (Mennin et al., 2005). These negative reactions may elicit further dysfunctional emotion regulation strategies, such as worry, that serve avoidant functions and exacerbate emotional dysfunction.

Evidence that fear and avoidance of emotional experience are common to GAD and PTSD (Roemer, 1997) and predict a more chronic course of PTSD following disaster (North et al., 1999) raises important questions about the potential role of these processes in psychological dysfunction following 9/11. Both emotional avoidance and fear of emotions have been shown to be associated with distress and severity of posttraumatic symptoms (Plumb, Orsillo, & Luterek, 2004) and increased GAD severity even after accounting for variance shared with level of trauma severity (Plumb et al., 2004) or chronic worry (Roemer, Salters, Raffa, & Orsillo, 2005). For example, Plumb et al. (2004, Study 3) found that, after controlling for combat exposure, experiential avoidance accounted for 13% of the unique variance in clinician-rated PTSD symptom severity and 31% of the variance in global distress among treatment-seeking combat veterans. In addition, self-reported avoidance coping has been linked to increased severity of posttraumatic symptoms following 9/11 in a number of nonexposed or indirectly exposed samples, including a nationwide online survey (Silver et al., 2002), children in Seattle, Washington
In addition, research indicates that intolerance of uncertainty may be relevant to the symptoms and dysfunction associated with GAD (Dugas, Gagnon, Ladouceur, & Freeston, 1998). Intolerance of uncertainty is a cognitive construct reflecting the extent to which one believes that uncertainty is unacceptable. Dugas and colleagues have demonstrated that a self-report measure of intolerance of uncertainty contributed to the statistical discrimination of individuals with GAD from nonanxious controls (Dugas et al., 1998), and that it may have a causal relationship to worry (Grenier & Ladouceur, 2004; Ladouceur et al., 2000). We are not aware of prior research examining the relationship of intolerance of uncertainty to negative posttraumatic reactions. However, it seems plausible that an inability to navigate the ambiguous posttraumatic environment (particularly one as unpredictable as post-9/11 New York City, which was characterized by frequent, protracted threats of further attacks) may worsen one’s clinical profile. The terrorist attacks on 9/11 and the protracted instability that ensued in the months afterward may have confirmed beliefs that uncertainty is intolerable in individuals who had GAD prior to the attacks, prolonging their traumatic experience (Piotrkowski & Brannen, 2002) and exacerbating the severity and chronicity of their psychological dysfunction.

The aims of this study were to determine the role of preattack analogue GAD, fear and avoidance of emotional experience, and intolerance of uncertainty, on long-term post-9/11 outcome in a sample of young adults exposed to the WTC attack and collapse. We hypothesized that preattack analogue GAD would have a detrimental effect on psychological functioning (i.e., psychological symptoms, impairment, and loss of psychological resources) 12 months later after controlling for high exposure to the attacks. We also hypothesized that individual differences in fear and avoidance of emotional experience and intolerance of uncertainty 4 months after 9/11 would statistically mediate the hypothesized effect of preattack analogue GAD on negative outcome at a 12-month follow-up after controlling for high exposure to the attacks. Prospective assessment of GAD occurred one day prior to 9/11, which offered a unique opportunity to examine the role of GAD as a potential risk factor for negative psychological sequelae
GAD and September 11

arising in the context of this disaster. We are not aware of previous prospective research examining post-9/11 psychological functioning as a function of preattack psychopathology among individuals who were exposed to the WTC attacks.

Method

Participants and Procedure

An overview of the study design is presented in Figure 1. On September 10, 2001, two hundred twenty-nine students enrolled in an introductory psychology course at New York University (NYU) completed the GAD Questionnaire for DSM-IV (GADQ-IV; Newman et al., 2002) and the Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996) as part of prescreening for a study unrelated to the present study. Although that study was not conducted due to the following day’s events, we obtained approval from the Institutional Review Board at NYU to conduct the present follow-up study. To increase statistical power to detect differences in long-term outcome associated with preattack analogue GAD, we aimed to recruit an analogue GAD group and a control group of approximately equal size. Based on criteria described below, we identified and contacted all individuals from the original sample of 229 students who met criteria for analogue GAD (n = 69, 30.1%) with or without depression (BDI-II > 15); we randomly contacted a subset of the remaining 160 (69.9%) students classified as controls until the groups were approximately equal in size. Of the 69 students with analogue GAD in the original sample, 23 (33.3%) students enrolled in the study; the remaining 46 (66.7%) individuals either did not respond to or declined the invitation to participate in the longitudinal study (unfortunately, data on these responses are not available). Thirty-four (21.3%) of the 160 students in the original sample who were classified as controls enrolled and completed the first follow-up assessment; unfortunately, data on the number of individuals from the control group who were contacted or unreachable are unavailable. Thus, 57 (24.9%) of the original sample of 229 students enrolled in the present longitudinal study.

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Insert Figure 1 about here

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After providing informed consent, participants completed 4- and 12-month follow-up (FU) assessments, for which they were paid $10 and $20, respectively. Thirteen (22.8%) participants who completed the 4-mo FU did not complete the 12-mo FU, including 5 (21.7%) from the analogue GAD group and 8 (23.5%) from the control group, leaving a final sample of 44 individuals (18 analogue GAD, 25 control) who completed all assessments through the 12-mo FU. Reasons for dropout were not assessed for the 13 (23%) participants who did not complete the 12-mo FU. However, these individuals did not differ from those who completed the 12-mo FU in terms of age, \( t(54) = .80, p = .43 \); sex, \( \chi^2(1, N = 57) = .67, p = .41 \); GAD status, \( \chi^2(1, N = 57) = .03, p = .87 \); or exposure group, \( \chi^2(1, N = 57) = .64, p = .42 \). Participants in the 4-mo FU sample did not differ from the original sample in age, \( t(212) = .80, p = .43 \) (\( M = 18.9 \) yrs, \( M = 19.0 \) yrs), and gender was differentially associated with membership in the 4-mo FU sample, \( \chi^2(1, N = 57) = 5.11, p = .02, \phi = .15 \), with females being more likely than males to complete the 12-mo FU assessment. Likewise, participants in the final sample (\( N = 44 \)) did not differ from the original sample (\( N = 229 \)) in age, \( t(255) = .29, p = .77 \) (\( M = 18.9 \) yrs, \( M = 19.0 \) yrs), but females in the original sample were more likely than males to complete the 12-mo FU assessment, \( \chi^2(1, N = 44) = 5.16, p = .02, \phi = .15 \). Analyses reported below were conducted on the sample of 44 participants who completed all follow-up assessments.

Insert Table 1 about here

Table 1 presents demographic, exposure, and clinical data on study participants. Participants ranged in age from 17.8 to 21.2 years (\( M = 18.9, SD = 0.8 \)). The ethnic distribution was 63% Caucasian-American, 21% Asian-American, 7% Latino-American, 2% African-American, and 7% of mixed or other ethnicity. Eleven (25.0%) participants reported that they were diagnosed with a psychological disorder.
within the year prior to 9/11 (depression, n = 7; anxiety n = 3; other/missing n = 2), and 12 (27.3%) participants reported that they had received psychological therapy within the same time frame.

Physical and psychological exposure to the terrorist attacks on 9/11 were assessed at 4-mo FU (see Table 1) using a modified version of a self-report measure of exposure (described below) developed by the National Institute of Mental Health for this purpose. All participants reported that they were in Manhattan during the WTC attacks. Typically, one’s fear or belief that one’s safety or well-being is in jeopardy is considered within high exposure demarcations (see Norris et al., 2002). However, this variable was not included among the high exposure criteria given its high rate in the total sample (91%). The remaining items assessed whether participants a) were south of Canal St. when the attacks occurred (i.e., within a short distance of the WTC towers), b) were seriously physically injured (e.g., were burned or harmed by debris), c) lost a loved one or close friend in the attacks, or d) were exposed to the dead or dying as a first hand witness or as part of the recovery efforts. Given that few participants endorsed more than one item, and items other than physical proximity to the attacks were endorsed infrequently (see Table 1), we coded individuals as highly exposed if they endorsed at least one of the items. Nineteen (43%) participants met this criterion for high exposure to the WTC attacks, including 7 (27%) participants in the control group and 12 (67%) participants in the analogue GAD group. Preattack analogue GAD status was significantly associated with high exposure based on self-report at 4-mo FU, \( \chi^2(1, N = 44) = 6.85, p = .01, \phi = .39. \)

**Baseline Selection Measures (September 10, 2001)**

The *GADQ-IV* (Newman et al., 2002) is a 9-item self-report questionnaire that was designed to provide an analogue diagnosis of GAD based on the DSM-IV (American Psychiatric Association, 1994) criteria for GAD. The GADQ-IV is primarily composed of dichotomous items concerning the excessive and uncontrollable nature of worry as experienced by persons with GAD (e.g., “During the last six months, have you been bothered by excessive worries more days than not?”) and physical symptoms including restlessness, sleep difficulties, difficulty concentrating, irritability, fatigue, and muscle tension. One open-ended item asks individuals to list up to six of their most frequent worry topics, and two items
ask them to rate their functional impairment and subjective distress on a scale of 0 (“None”) to 8 (“Very Severe”). Item scores are combined to create a total dimensional score ranging from 0 to 13. However, Newman et al. (2002) cautioned that this score violates classic psychometric assumptions due to the presence of a skip-out item; they recommended instead that investigators apply a cutoff to the total score to classify analogue GAD as present or absent. Newman et al. (2002) reported that a 5.7 cutoff score on the GADQ-IV had excellent sensitivity and specificity for DSM-IV diagnosis of GAD in a college student sample. High rates of sensitivity and specificity for this cutoff score have also been reported using the Anxiety Disorders Interview Schedule for DSM-IV (ADIS-IV; Brown, Anthony, & Barlow, 1994) as a criterion in treatment-seeking populations (Luterek et al., 2002). Following these findings, individuals scoring 5.7 or greater were classified as having an analogue diagnosis of GAD and those scoring below this cutoff were classified as control participants in the present study.

Descriptive and Meditational Measures (4-month follow-up)

Assessment of Exposure to the Events (AEE; Norris, 2002) is a self-report measure that was designed to capture multiple dimensions of individuals’ exposure to the events of 9/11. Items include questions about physical proximity to the WTC attacks (e.g., “Were you directly exposed to the disaster of September 11? By directly, I mean that you were in the vicinity of the World Trade Center or the Pentagon at the time the disaster occurred?”); injury or physical impairment because of the attacks (e.g., “Were you injured physically in any way, that is [a] were you burned or harmed by debris or [b] your ability to breathe, see hear, or move about was impaired in more than a transient way?”); loss of a loved one or close friend; exposure to the dead and dying as part of recovery efforts; evacuation from one’s building or place of work; and the extent to which one experienced fear for one’s safety, well-being, and acceptance due to one’s ethnic or religious background or others’ perceptions of one’s ethnic or religious background. We modified the AEE by adding items asking participants to report their specific location during the WTC attacks on 9/11 (i.e., whether they were south of the New York street designations of 14th or Canal). A subset of the modified AEE was used to create the high exposure grouping variable as described above.
The *Affective Control Scale* (ACS; Williams et al., 1997) is a 42-item self-report measure assessing fear of emotions and their consequences. Subscales include 1) fear of anxiety (e.g., “It scares me when I am nervous”); 2) fear of depression (e.g., “When I get ‘the blues’, I worry that they will pull me down too far”); 3) fear of anger (e.g., “I am afraid that letting myself feel really angry about something could lead me into an unending rage”); and 4) fear of positive emotions (e.g., “I worry about losing self-control when I am on cloud nine”). Items are scored on a 7-point Likert-type scale (i.e., “Strongly Agree” to “Strongly Disagree”). The subscales have demonstrated satisfactory internal consistency (Berg, Shapiro, & Chambless, 1998; Williams et al., 1997). The ACS total score, which was analyzed in the present study, is highly correlated with neuroticism and emotional control and minimally correlated with social desirability (Berg et al., 1998; Williams et al., 1997). Internal consistency was good in the present study (Cronbach’s $\alpha = .83$).

The *Acceptance and Action Questionnaire* (AAQ; Hayes et al., 2004) is a 9-item self-report questionnaire designed to assess several facets of the experiential avoidance construct. Individuals are asked to rate each item in terms of how true it is when applied to themselves on a Likert-type scale from 1 (“never true”) to 7 (“always true”). Items query individuals’ avoidance of distressing private events (e.g., “I’m not afraid of my feelings” [reverse scored]), high need for control over emotions and cognitions (e.g., “I rarely worry about getting my anxieties, worries, and feelings under control” [reverse scored]), difficulty taking important action in the face of uncertainty or negative private events (e.g., “I am able to take action on a problem even if I am uncertain what is the right thing to do”), and negative evaluations of internal experiences (e.g., “Anxiety is bad”). The AAQ has been shown to have weak internal consistency (Cronbach’s $\alpha = .70$), which was comparable to that obtained in the present study (Cronbach’s $\alpha = .67$). The AAQ correlates moderately with related constructs such as thought suppression, thought control, and behavioral/emotional avoidance ($r$s between .26 and .50), and it does not correlate with unrelated constructs such as social desirability (Hayes et al., 2004). The total score was used in the present study.
The Intolerance of Uncertainty Scale (IUS; Freeston, Rheaume, Letarte, Dugas, & Ladouceur, 1994) was designed to measure an individual’s intolerance of uncertainty, particularly their beliefs that uncertainty is unacceptable, reflects badly on a person, leads to frustration and stress, and leads to the inability to take action. The scale is composed of 27 items (e.g., “Uncertainty makes me uneasy, anxious, or stressed”) and is scored on a 1 (“not at all characteristic of me”) to 5 (“entirely characteristic of me”) ordinal scale. The IUS has shown excellent internal consistency (Cronbach’s α = .94), good test-retest reliability over a five-week period (r = .78), and highly significant correlations with measures of worry and depression (Buhr & Dugas, 2002). In the present study the IUS demonstrated excellent internal consistency (Cronbach’s α = .93), and the total score was used in the analyses.

Outcome Measures (1-year follow-up)

The Sheehan Disability Scale (SDS; Sheehan, 1983) is a 4-item scale on which individuals rate their current level of disability in work, social life, and family life on a scale from 0 (“not at all”) to 10 (“very severe”). The first three items are summed to determine current disability; however, the fourth item, on which individuals rate their work and social disability on scale from 1 (“no complaints, normal activity”) to 5 (“symptoms radically change or prevent normal work or social activities”) scale, may also be used (Hambrick, Turk, Heimberg, Schneier, & Liebowitz, 2003, 2004). Internal consistency is weak given the small number of items in the scale (e.g., Cronbach’s α = .55; Hambrick et al., 2004). However, the SDS has been shown to correlate well with other criterion measures of current disability, symptoms, and quality of life in patient populations (Kennedy, Lin, & Schwab, 2002; Leon, Shear, Portera, & Klerman, 1992), and to respond to changes in treatment among primary care patients with mood and anxiety disorders (Olfson et al., 1997).

Loss of Psychosocial Resources (LPR; Norris, 2002) is a 12-item measure assessing psychological reactions to the September 11th terrorist attacks. It is the second part of a two-part questionnaire (the AEE is the first part) commissioned by the National Institute of Mental Health (NIMH) to examine individuals’ exposure and distress following the attacks (Norris, 2002). The LPR asks individuals to rate each item in terms of how true it is about themselves on an ordinal scale from 1 (“not
true at all”) to 5 (“extremely true”). Two 6-item subscales comprise the LPR: 1) loss of agency, which concerns the perception of a loss of psychosocial resources, such as control, hope, and optimism, after the attacks (e.g., “Compared to how you felt before the disaster, you feel less able to control the forces that will influence your life”); and 2) loss of support, whose items reflect disappointment in one’s own or others’ actions after the attacks (e.g., “You were disappointed to find that someone you thought you could count on for support or assistance was not helpful to you when you needed him or her”). The LPR demonstrated adequate internal consistency (Cronbach’s α = .78) in a preliminary sample of 278 non-directly-exposed undergraduate students in Georgia who completed the measure 5 weeks after the 9/11 attacks (Norris, 2002). In the present study, internal consistency for the measure was excellent (Cronbach’s α = .86), and the total score was used.

The Mood and Anxiety Symptom Questionnaire-Short Form (MASQ-SF; Watson & Clark, 1991) is a 62-item measure assessing symptoms that commonly occur in the mood and anxiety disorders. The MASQ-SF consists of five subscales: General Distress: Anxious Symptoms (GDA); General Distress: Depressive Symptoms; General Distress: Mixed Symptoms (GDM); Anxious Arousal; and Anhedonic Depression. Each item is rated on a 1 (“not at all”) to 5 (“extremely likely”) Likert-type scale. In the present study, correlations among the MASQ-SF subscales at 12-mo FU were high (all Rs .46-.84); accordingly, the total score was used.

The Posttraumatic Stress Disorder Diagnostic Scale (PDS; Foa, Cashman, Jaycox, & Perry, 1997) is a 49-item self-report measure designed to provide an analogue diagnosis of PTSD according to DSM-IV (APA, 1994) diagnostic criteria. The first 12 items comprise a checklist of various kinds of traumatic events. Individuals who endorse experiencing more than one traumatic event are asked to indicate which of the endorsed events bothers them most and to refer to that event for the remaining questions. In the present study, the items were rephrased to instruct participants to answer the questions as they pertain only to the 9/11 terrorist attacks. The next 17 items comprise the DSM-IV PTSD symptom criteria: Reexperiencing (5 items; Criterion B), Avoidance (7 items; Criterion C), and Arousal (5 items; Criterion D). Individuals are asked to rate the frequency with which they have experienced each
symptom in the past month on a 4-point scale from 0 (“not at all or only one time”) to 3 (“five or more times a week/almost always”). These 17 items are summed to create a symptom severity score, which was used in the present study. The final nine items assess individuals’ impairment in different life areas (e.g., work, family, life satisfaction) in the past month in a yes-no checklist. Foa et al. (1997) found the symptom severity score to have excellent internal consistency (Cronbach’s $\alpha = .92$) and good test-retest reliability ($\kappa = .74$) in a sample of individuals reporting a wide variety of recent trauma. Furthermore, they reported that the PDS demonstrated satisfactory agreement with the SCID-IV diagnosis of PTSD ($\kappa = .65$, sensitivity = .89, and specificity = .75). In the present study, the PDS symptom severity items exhibited excellent internal consistency (Cronbach’s $\alpha = .87$).

The Penn State Worry Questionnaire-Past Month (PSWQ-PM) is a 15-item self-report questionnaire designed by the second author to measure the propensity to worry uncontrollably (e.g., “I know I shouldn’t worry about things, but I just can’t help it”), excessively (e.g., “I am always worrying about something”), and pervasively (e.g., “Many situations make me worry”), regardless of worry topic, during the past month. The questionnaire instructs individuals to indicate how often each statement was characteristic of them in the past month on a 5-point ordinal scale from 1 (“Not at all typical”) to 5 (“Very typical”). The PSWQ-PM was adapted from a prior validation study of the Penn State Worry Questionnaire-Past Week (PSWQ-PW; Stöber & Bittencourt, 1998), which was designed to measure weekly changes in worry. As with the PSWQ-PW, the PSWQ-PM dropped one item (i.e., “I’ve been a worrier all my life”) from the original 16-item PSWQ (Meyer, Miller, Metzger, & Borkovec, 1990) because the item did not match with the instructions to answer based on the past month. In the present study, the PSWQ-PM demonstrated weak internal consistency (Cronbach’s $\alpha = .61$). The total score was used in the analyses.

Data Analytic Plan

Figure 2 illustrates the conceptual and data analytic models described below. Two analytic models were examined within our overarching aim of testing whether fear and avoidance of emotions and intolerance of uncertainty statistically mediated the effect of preattack analogue GAD on psychological
outcome at 12-mo FU. The first model (Fig. 2, Model 1) examined the total effect of analogue GAD on each outcome measure after controlling for high exposure to the attacks. The second model (Fig. 2, Model 2) examined the indirect effect of analogue GAD on outcome through each intervening variable (i.e., ACS, AAQ, or IUS) after controlling for high exposure. Both models included high exposure as a covariate to examine the extent to which statistical mediation could be inferred beyond the effect of proximal contact with the WTC attacks.

Following recent recommendations for statistical mediation analysis (Preacher & Hayes, 2004; Shrout & Bolger, 2002), we first examined the strength of the paths in the models via simple correlation and multiple regression analysis to examine the plausibility of statistical mediation. First, each outcome was separately regressed onto both high exposure and analogue GAD to obtain the total effect \( c \) of analogue GAD on outcome. Next, components of the indirect effect, \( a \) and \( b \), were examined in two regression models. Path \( a \) was obtained from the partial coefficient for analogue GAD after regressing the outcome onto both high exposure and analogue GAD, and Path \( b \) from the partial coefficient for the hypothesized mediator after regressing the outcome at 12-mo FU onto high exposure, analogue GAD, and the hypothesized mediator. To protect the overall familywise Type I error rate and statistical power, hypothesized mediators that were not significantly associated with either analogue GAD or outcome were not tested for statistical mediation.

After examining the plausibility of a mediator model for each combination of candidate mediator and outcome, we used nonparametric bootstrapping methods (Efron & Tibshirani, 1993; Fox, 2002) to conduct a formal test of the null hypothesis that there was no indirect effect of analogue GAD on outcome (e.g., work and social disability) involving fear or avoidance of emotion. Bootstrapping procedures involve randomly resampling with replacement from the observed data to generate an empirical sampling distribution for the statistic of interest (e.g., the indirect effect). When sample size is small, as in the
present study, there are strong reasons to use bootstrapping over traditional analytic procedures to examine statistical mediation (e.g., Baron & Kenny, 1986), including the superior statistical power, coverage, and reliability of bootstrapped confidence intervals (for excellent discussions, see MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002; Shrout & Bolger, 2002). For technical reasons, we report bias-corrected-and-accelerated (BCa) CI’s, a modified form of bootstrapped confidence intervals that have been recommended by experts on statistical mediation (e.g, MacKinnon et al., 2002).

Statistical mediation analyses were conducted with the *boot* package (Version 1.2-27; Canty, 2006) in R (Version 2.5.0; R Core Development Team, 2007). For each analysis, we generated 10,000 independent bootstrap samples of size $N = 44$ through random resampling with replacement of cases from the original sample. For each bootstrap sample, the indirect effect and its effect size ($\text{Cohen’s } f^2$) were calculated from the product of unstandardized regression coefficients for paths $a$ and $b$ estimated from that sample’s data (MacKinnon & Dwyer, 1993). The resultant empirical sampling distribution was used to derive bootstrapped estimates of the indirect effect ($ab^*$; the distribution mean), BCa 99% CI’s around $ab^*$, and Cohen’s $f^2*$. The null hypothesis of no indirect effect was rejected when the BCa 99% CI* did not overlap with 0. Alpha was set at .01 per mediational hypothesis test to keep the probability of at least one Type I error under .10 across all statistical mediator analyses.¹

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Insert Table 2 about here

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Results

Table 2 presents the zero-order correlations among all covariates, predictors, hypothesized mediators, and outcome variables. High exposure was not significantly associated with any other model variables except analogue GAD, ACS, and IUS. Analogue GAD, however, had strong positive correlations with all outcomes variables except posttraumatic stress symptom severity. Analogue GAD was strongly positively related to ACS and AAQ, but not to IUS. Correlations between hypothesized mediators (i.e., ACS, AAQ, IUS) were positive and large. ACS and AAQ exhibited positive correlations
ranging from moderate to strong with all outcome variables, but IUS was significantly related only to PSWQ-PM. High exposure did not significantly predict any of the outcome variables at 12-mo FU nor IUS at 4-mo FU; however, high exposure was significantly and positively associated with ACS at 4-mo FU.

Multiple regression analyses were used to describe the relationships among model variables after controlling for high exposure and to assess whether any of the planned statistical mediation analyses were implausible. The results of these analyses are presented in the first two columns of Table 3. The relationship between high exposure and analogue GAD and each outcome was examined (i.e., the total effect; see Fig. 2, Model 1, Path c). After controlling for high exposure, analogue GAD significantly predicted all outcome measures except for PDS symptom severity. Next, the direct effect of analogue GAD on each hypothesized mediator was assessed (see Fig. 2, Model 2, Path a). ACS, AAQ, and IUS were found to be significantly predicted by analogue GAD after controlling for high exposure (see Table 3). Finally, we estimated the direct effect of each hypothesized mediator on the outcome variables (see Fig. 2, Model 2, Path b), which involved regressing each out outcome onto high exposure, analogue GAD, and the hypothesized mediator. Both ACS and AAQ significantly predicted all outcome measures in this analysis (see Table 3), but IUS did not predict any outcomes except PSWQ-PM, $\beta = .42, t(40) = 3.12, p < .01$. Given that IUS exhibited low and nonsignificant associations with preattack analogue GAD (i.e., Path a) and with nearly all outcome measures at 12-mo FU (i.e., Path b), we did not include IUS in the statistical mediation analysis.

Finally, nonparametric bootstrap procedures were used to conduct the statistical mediation analyses, presented in the last four columns of Table 3. Indirect effects and their 99% CI’s and effect sizes were used to examine whether the association between preattack analogue GAD and psychological functioning at 12-mo FU was statistically mediated by ACS, AAQ, or IUS assessed at 4-mo FU, after
controlling for high exposure (see Figure 2, Model 2). All indirect effects were medium to large in size (all Cohen’s $f^2$'s = .16-.43). ACS statistically mediated the relation between analogue GAD and all outcomes except LPR and PDS; AAQ statistically mediated all outcomes. However, the lower bound of the confidence intervals for the indirect effect of the relation between analogue GAD and LPR was very close to zero for ACS and AAQ, with medium (Cohen’s $f^2 = .16$) and medium-to-large (Cohen’s $f^2 = .27$) effect sizes, respectively. Because LPR is composed two subscales, Loss of Agency and Loss of Support, we conducted exploratory analyses to assess the extent to which ACS statistically mediated the effect of analogue GAD on either of these subscales. The relation between analogue GAD and Loss of Agency was not statistically mediated by ACS, $ab^* = .10$ (BC$_a$ 99% CI*: -.20, .63), Cohen’s $f^2 = .04$, nor by AAQ, $ab^* = .17$ (BC$_a$ 99% CI*: -.10, .79), Cohen’s $f^2 = .08$. However, the relation between analogue GAD and Loss of Support was statistically mediated by ACS, $ab^* = .33$ (BC$_a$ 99% CI*: .02, .83), Cohen’s $f^2 = .28$, as well as by AAQ, $ab^* = .34$ (BC$_a$ 99% CI*: .02, .87), Cohen’s $f^2 = .37$.

Discussion

The present study examined longitudinal relationships between preattack analogue GAD and a range of negative psychological outcomes 12 months after 9/11 in a convenience sample of young adults with and without analogue GAD who were exposed to the WTC attacks in New York City on 9/11. Results were consistent with our first hypothesis that preattack analogue GAD would predict a range of negative outcomes 12 months after the attack, including greater functional impairment, loss of psychosocial resources, mood and anxiety symptoms, and worry symptoms. Preattack analogue GAD did not predict the severity of posttraumatic stress symptoms. However, it is important to note that, because these outcomes were not also assessed prior to 9/11, an alternative explanation for the findings is that people with preattack analogue GAD exhibit greater levels of functional impairment, loss of resources, and symptoms of mood, anxiety, and worry than those without analogue GAD, regardless of traumatic exposure.

The findings also were largely consistent with our second hypothesis that fear of emotion, emotional avoidance, and intolerance of uncertainty assessed four months after 9/11 would statistically
mediate the relationship between preattack analogue GAD and a range of negative outcomes 12 months after 9/11. Specifically, the effects of preattack analogue GAD on measures of functional impairment, mood and anxiety symptoms, and worry symptoms were statistically mediated by measures of both fear of emotions and emotional avoidance; the relations with loss of psychosocial resources and symptoms of posttraumatic stress were statistically mediated by emotional avoidance but not by fear of emotions. Follow-up analyses of subscales of loss of psychosocial resources revealed large indirect effects involving both fear and avoidance of emotion and loss of support, but not loss of agency. Finally, because intolerance of uncertainty measured 4 months after 9/11 was not associated with preattack analogue GAD nor with most of the outcomes, it was not submitted to statistical mediation analysis.

Our findings suggest that both fear of emotions and emotional avoidance are important to consider in the relationship between pretrauma GAD and posttraumatic negative outcome in the aftermath of a major urban disaster. As we discuss in the limitations section below, because temporal precedence cannot be established, the results could be due to higher levels of fear and avoidance of emotions preceding the development of GAD, or various other permutations of causal and temporal ordering of variables in our model. However, the present study was not designed to distinguish amongst these alternative models, but rather to examine whether the data from this convenience sample are consistent with a theory-driven conceptualization of fear and avoidance of emotion and intolerance of uncertainty as processes by which pre-9/11 GAD affects outcome. Indeed, the present study presents an opportunity to generate hypotheses about the outcome variables, such as disability and psychosocial resources, which rarely are examined in traumatized samples. To our knowledge, this is the first study to examine a prospectively assessed risk factor for negative psychological functioning 9/11 in a sample of individuals who were exposed to the WTC attacks. The prospective assessment of analogue GAD and depression is a unique and important feature of this study because it allows us to be certain that the events of 9/11 did not influence participants’ status on these constructs.

The results of the present study are consistent with prior research indicating that GAD and related trait constructs increase risk for negative postdisaster outcome (e.g., Koenen et al., 2002, Weems et al.,
However, the finding that preattack analogue GAD was not associated with increased posttraumatic stress symptom severity 12 months after 9/11 appears to be at odds with prior longitudinal studies of large-scale disaster (e.g., La Greca et al., 1998). If this null finding reflects a true nonrelationship, it could suggest that negative emotional reactivity is an independent risk factor for the severity of posttraumatic stress symptoms (Kraemer et al., 2001). However, our sample was too small to license inferences about this null finding.

Our hypothesis that intolerance of uncertainty statistically mediates the relationship between preattack analogue GAD and negative outcome was not supported. We had predicted that intolerance of uncertainty would statistically mediate this relationship on the basis of prior research demonstrating its strong, potentially causal relationship both to worry (Grenier & Ladouceur, 2004; Ladouceur et al., 2000) and to biased processing of ambiguous information (Dugas, Hedayati, Karavidas, Buhr, Francis, & Phillips, 2005). Surprisingly, we found that preattack analogue GAD predicted almost no variance in IUS at 4-mo FU after controlling for high exposure, and IUS did not predict any of the outcome measures except worry. Individuals who met criteria for high exposure also tended to receive higher scores on the IUS; however, the strength of this relationship was not statistically significant. One speculation is that the events of 9/11 and the months that followed elicited a high degree of uncertainty, creating a ceiling effect for the role of preattack analogue GAD on intolerance of uncertainty at 4-mo FU. Prospective measurement of preattack intolerance of uncertainty within a larger sample would be necessary to evaluate this speculation empirically.

Our finding that fear and avoidance of emotions statistically mediated the relations between preattack analogue GAD and negative outcomes after 9/11 converges with recent conceptualizations of GAD within an emotion dysregulation framework (Mennin, Heimberg, Turk, & Fresco, 2002; Mennin et al., 2005; Roemer et al., 2005). Preliminary support has emerged for the contribution of four factors to dysregulatory emotional processes in individuals with GAD: 1) heightened emotional intensity, 2) difficulty understanding one’s emotional experience, 3) negative reactivity to emotions (e.g., negative beliefs regarding one’s emotional experiences), and 4) difficulty managing one’s emotional experiences.
If our theoretical model was specified correctly, the results of the present study could provide preliminary support for the predictive validity of the negative emotional reactivity and emotional management components of this model. Additional factors, such as heightened emotional intensity, also may be important to understanding the pathways to increased impairment and symptom severity following trauma and prolonged stress. Furthermore, the present findings are consistent with evidence that greater emotional avoidance is associated with poor post-event functioning and stress (Plumb et al., 2004), lower levels of functioning and well-being, and higher levels of mood and anxiety symptoms (Hayes et al., 2004; Marx & Sloan, 2002). Further research will be needed to determine whether negative emotional reactivity and poor management of emotional experience are, in fact, mechanisms by which pretrauma psychopathology such as analogue GAD increases risk for negative posttraumatic outcomes.

Although we cannot conclude that our theoretical model is correct on the basis of the present study, the present findings suggest several avenues for future research. For example, future research should explore whether changes in fear of emotions or emotional avoidance among trauma-exposed individuals impacts subsequent psychological functioning and distress, especially with anxious individuals. Considerable research documents the beneficial effects of written emotional disclosure following a stressful life event (Pennebaker, 1993; Pennebaker, Mayne, & Francis, 1997). Furthermore, linguistic analysis of daily entries in an online journal revealed a higher frequency of linguistic markers of psychological distancing (e.g., more frequent use of long abstract words and passive voice and less frequent use of first-person, present-tense voice) among preoccupied than nonpreoccupied individuals during the two months after 9/11 (Cohn, Mehl, & Pennebaker, 2004). Future research could examine whether written emotional disclosure exercises targeted at increasing emotional engagement and emotional processing (Foa & Kozak, 1986) would reduce posttraumatic psychological distress associated with GAD. Emotional approach coping has also been shown in prospective studies to predict women’s positive adjustment to breast cancer diagnosis (Stanton, Danoff-Burg, Cameron, & Ellis, 1994; Stanton, Kirk, Cameron, & Danoff-Burg, 2000). Finally, the ability to up- and down-regulate one’s emotional expression in a flexible, context-appropriate manner may be critical in helping individuals adjust
following a traumatic event such as the terrorist attacks on 9/11 (Bonanno, Papa, Lalande, Westphal, Coifman, 2004).

limitations

Several important limitations to this study should be noted. First, despite the longitudinal nature of the present study, we cannot establish the temporal ordering of changes in one variable relative to another because each construct was assessed at a unique time. Because temporal precedence—a critical prerequisite for inferences about cause and effect, mediation, and risk factors (Kraemer et al., 2001)—could not be established on the basis of our design, our findings should be interpreted as hypothesis generators rather than as confirmation of our theoretical mediation model. For example, it is possible that our results emerged because individuals with analogue GAD exhibit lower psychological functioning and higher levels of mood and anxiety symptoms than individuals without analogue GAD. Rival explanations such as this could be rendered less plausible by demonstrating that pretrauma analogue GAD predicts negative posttraumatic psychological sequelae after controlling for initial levels of these outcomes. However, this analysis depends on the availability of baseline data on the outcome variables, which often is not the case in disaster research. Further, for obvious ethical and practical reasons, we could not manipulate our variables or randomly assign individuals to analogue GAD or high exposure status to obtain strong evidence of cause and effect.

A second limitation concerns the weak internal consistency of our measures of emotional avoidance (AAQ), functional impairment (SDS), and worry (PSWQ-PM). The AAQ and SDS often are found to have weak internal consistency in part because they contain few items (9 and 4 items, respectively), which limits scale reliability. Despite this issue, both scales have demonstrated convergent and discriminant validity with more reliable measures, which has helped clarify the constructs purportedly tapped by these scales. The AAQ, in particular, has been found repeatedly to predict considerable variance in psychological outcomes beyond the contribution of trait constructs such as negative affectivity (e.g., Roemer et al., 2005; Salters-Pedneault, Roemer, Tull, Rucker, & Mennin, 2006). It was more surprising that the PSWQ-PM did not exhibit higher internal consistency, as similar versions have
obtained Cronbach’s alphas as high as .91 in other studies (Stöber & Bittencourt, 1998). Despite its low internal consistency in the present study, however, the PSWQ-PM exhibited strong effects in all analyses.

Third, our sample was small for a longitudinal study examining multiple potential risk factors, limiting both the conclusions we can draw from this study as well as the complexity of the models we could examine. Notwithstanding low power, many significant and medium-to-large effects were found and should, thus, encourage follow-up research on the role of fear and avoidance of emotion in GAD in future disaster research.

Fourth, our conclusions in this study are limited to the population of college students with and without analogue GAD who were exposed to the 9/11 attacks. It would be premature to generalize our findings to other populations, including children, community samples, and individuals living far from the attacks on 9/11. As one example, consider that individuals in community samples, compared with individuals in college student samples, are likely to have fewer available, less affordable, and less accessible social and mental health support services. In light of these differences as well as ample evidence of marked distress in community samples following 9/11 (e.g., Galea et al., 2003), exploring how GAD and depression and emotion regulatory processes relate to clinically relevant outcomes following disaster is clearly an important direction for future research.

Fifth, we found that preattack analogue GAD and report of high exposure were not independent in the full and completer samples. We offer several speculations about why this should or should not have occurred. First, exposure items queried individuals about specific behavioral indices of exposure, such as whether they were south of Canal St. during the collapse of the WTC, which should have attenuated any retrospective reporting bias favoring high exposure. However, individuals with GAD still may have been more likely than non-GAD individuals to inflate their level of exposure to the attacks in their retrospective reports. Second, analysis of participants’ responses on the individual items comprising high exposure status did not reveal differential patterns of response by GAD group. Third, because the attacks on the WTC were completely unexpected, proximity to the attacks arguably should not have been related systematically to diagnostic status. However, given that this relationship was found in the sample
that completed the 4-month follow-up ($N = 57$) as well as in the subsample that also completed the 12-month follow-up ($N = 44$), a genuine effect of differential rates of high exposure across the groups may be the most parsimonious explanation.

Finally, most of the participants who met criteria for analog GAD also met criteria for depression by the BDI-II. Analog GAD status was significantly correlated with BDI-II total score. Although this is not surprising given the high rate of 30-day and lifetime comorbidity of the disorders in epidemiological studies (e.g., Wittchen, Zhao, Kessler, & Eaton, 1994), the overlap between analogue GAD and BDI-II scores in our sample suggests that depression may comprise part of the preattack construct examined in our study. Prior analyses showed a relationship between preattack depression and a similar negative outcome profile, but this relationship was not statistically mediated by fear and avoidance of emotions or by intolerance of uncertainty (Farach & Mennin, 2004). Additionally, power was too low in the present study to examine additive or interactive effects of comorbid depression in the model. Given these findings, statistical limitations, and the theoretical focus on analogue GAD in the present study, the relationship of preattack depression to emotion dysregulation and outcome was not further examined. At least one prospective study (Nolen-Hoeksema & Morrow, 1991) in the disaster literature has shown a relationship between predisaster dysregulatory processes (i.e., rumination) and levels of postdisaster depression among young adults exposed to a major disaster (i.e., the Loma Prieta earthquake). The role of preattack depression in predicting outcome will be important to examine in future research given its links to poor functioning (e.g., Kessler, DuPont, Berglund, & Wittchen, 1999) and to emotion dysregulation (e.g., Rottenberg, Gross, Wilhelm, Najmi, & Gotlib, 2002; Rottenberg, Kasch, Gross, & Gotlib, 2002).

**Conclusion and Implications**

We found that young adults exposed to the terrorist attacks in New York City on September 11, 2001, were more likely to have increased levels of impairment; to perceive greater loss of psychological resources; and to report greater symptoms of mood, anxiety, and worry 12 months later if they met criteria for analogue GAD the day before the attacks. Moreover, four months after the attacks, the extent
to which participants reported that their emotions are to be feared or avoided statistically mediated the relationship between GAD and negative outcome after accounting for the effect of high exposure to the attacks. Though tentative, our findings suggest that both fear of emotions and avoidance of emotional experience may be important in understanding how GAD may contribute to long-term negative outcome following a major urban terrorist incident such as the 9/11 attacks.

Understanding how negative emotional reactivity and other emotion regulation deficits may mediate between GAD and negative outcome may be critical to the development and refinement of treatments for GAD. Several treatments already in existence or in development explicitly target fear and avoidance of emotional experience. For example, a main goal of Acceptance and Commitment Therapy (ACT; Hayes, Strosahl, & Wilson, 1999) is to increase clients’ acceptance of their emotional experience. In addition, mindfulness meditation techniques (Segal, Williams, & Teasdale, 2002), which focus on increasing clients’ awareness of their moment-to-moment experience and contingencies, have been integrated into cognitive-behavioral treatments for GAD with preliminary success (Roemer & Orsillo, 2003). Fear and avoidance of emotions are also explicit intervention targets in an interpersonal, emotion-focused, treatment for GAD (Newman, Castonguay, Borkovec, & Molnar, 2004), as well as an emotion regulation treatment for GAD currently being developed by Mennin and colleagues (Mennin, 2004). Unpacking the mechanisms by which fear and avoidance of unwanted emotional experiences may hinder emotional processing may be critical to the identification of effective targets for intervention and improving treatment efficacy for GAD.
GAD and September 11

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Rita Smith is now at Department of Psychology, University of Hawaii, and Matthew Mandelbaum is now at the Bank Street College of Education.

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References


Footnotes

1 We performed ten hypothesis tests of the indirect effect. The familywise Type I error rate would be approximately .10 for this study under the assumption that the tests are independent. However, examination of simple correlations between outcomes and between hypothesized mediators and outcomes in Table 2 does not support the assumption of independence. For example, intercorrelations between the five outcome variables range from $r = .46-.76$, and ACS and AAQ, the two hypothesized mediators that ultimately were tested with these outcomes, are very highly correlated ($r = .76$). Given the nonindependence among the hypothesis tests as well as the need to balance against the substantial risk of a Type II error in such a small sample, we opted to use a modest correction of per comparison alpha to .01.

2 Readers may wonder, as our reviewers did, whether a moderator model may have been more appropriate than a mediator model. Specifically, is the relation between preattack analogue GAD and negative psychological sequelae significantly stronger among people reporting higher levels of fear of emotions or experiential avoidance than among people reporting lower levels of these constructs? We did not plan to examine this moderator model because the literature on emotion regulation in GAD led us to conceptualize the constructs of fear and avoidance of emotions as processes that may lead to negative outcomes rather than as modifiers of risk for negative posttraumatic outcomes associated with preattack analogue GAD status. Moreover, we did not suspect the presence of moderation due to the reasonably strong correlations between analogue GAD and fear and avoidance of emotion ($rs = .51$ and $.42$, respectively). However, we conducted additional analyses to address this question; a moderator model was not supported (analyses are available upon request from the corresponding author).

3 We thank an anonymous reviewer for suggesting this exploratory analysis.

4 We thank an anonymous reviewer for suggesting this example.
Table 1

Demographic, clinical, and September 11 exposure characteristics of participants present at both 4-month and 12-month follow-up assessments (N = 44)

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female</td>
<td>38</td>
<td>86</td>
</tr>
<tr>
<td>GAD (GADQ-IV &gt; 5.7)</td>
<td>18</td>
<td>41</td>
</tr>
<tr>
<td>In NYC on 9/11</td>
<td>44</td>
<td>100</td>
</tr>
<tr>
<td>Personally witnessed 9/11 attack</td>
<td>21</td>
<td>48</td>
</tr>
<tr>
<td>High Exposure</td>
<td>19</td>
<td>43</td>
</tr>
<tr>
<td>Proximity (below Canal St.)</td>
<td>12</td>
<td>27</td>
</tr>
<tr>
<td>Burned/injured/impaired</td>
<td>5</td>
<td>11</td>
</tr>
<tr>
<td>Loss of loved one</td>
<td>4</td>
<td>9</td>
</tr>
<tr>
<td>Loved one injured/impaired</td>
<td>4</td>
<td>9</td>
</tr>
<tr>
<td>Exposure to dead or dying</td>
<td>1</td>
<td>2</td>
</tr>
</tbody>
</table>

Note. GAD = Generalized Anxiety Disorder; GADQ-IV = Generalized Anxiety Disorder Questionnaire-IV.
Table 2

*Intercorrelations between predictors, mediators, and outcome variables (N = 44)*

<table>
<thead>
<tr>
<th>Measure</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. High Exposure</td>
<td>--</td>
<td>.39**</td>
<td>.31*</td>
<td>.13</td>
<td>.39**</td>
<td>.10</td>
<td>.15</td>
<td>.22</td>
<td>.15</td>
<td>.13</td>
</tr>
<tr>
<td>2. GADQ-IV</td>
<td>--</td>
<td>.51***</td>
<td>.42**</td>
<td>.25</td>
<td>.34*</td>
<td>.46**</td>
<td>.38*</td>
<td>.21</td>
<td>.51***</td>
<td></td>
</tr>
<tr>
<td>3. ACS</td>
<td>--</td>
<td>.76***</td>
<td>.60***</td>
<td>.51**</td>
<td>.48***</td>
<td>.61***</td>
<td>.38*</td>
<td>.67***</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. AAQ</td>
<td>--</td>
<td>.58***</td>
<td>.51**</td>
<td>.54***</td>
<td>.50***</td>
<td>.49***</td>
<td>.62***</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. IUS</td>
<td>--</td>
<td>.28</td>
<td>.20</td>
<td>.27</td>
<td>.12</td>
<td>.45**</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. SDS</td>
<td>--</td>
<td>.49***</td>
<td>.67***</td>
<td>.58***</td>
<td>.46**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. LPR</td>
<td>--</td>
<td>.55***</td>
<td>.73***</td>
<td>.57***</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. MASQ-SF</td>
<td>--</td>
<td>.67***</td>
<td>.76***</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>9. PDS</td>
<td>--</td>
<td>.53***</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. PSWQ-PM</td>
<td></td>
<td></td>
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</tbody>
</table>

*Note. GADQ-IV = Generalized Anxiety Disorder Questionnaire-IV; ACS = Affective Control Scale; AAQ = Acceptance and Action Questionnaire; IUS = Intolerance of Uncertainty Scale; SDS = Sheehan Disability Scale; LPR = Loss of Psychosocial Resources; MASQ-SF = Mood and Anxiety Symptom Questionnaire; PDS = Posttraumatic Stress Diagnostic Scale (Symptom Severity); PSWQ-PM = Penn State Worry Questionnaire-Past Month.

*p < .05. **p < .01. ***p < .001.*
Table 3

*Standardized path estimates and bootstrapped estimates of the indirect effect.*

<table>
<thead>
<tr>
<th>Criterion</th>
<th>Sample Paths</th>
<th>Bootstrapped Indirect Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$\beta$</td>
<td>$t$</td>
</tr>
<tr>
<td><strong>Predictor: GADQ-IV</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SDS</td>
<td>.37</td>
<td>2.23*</td>
</tr>
<tr>
<td>LPR</td>
<td>.49</td>
<td>3.18**</td>
</tr>
<tr>
<td>PDS</td>
<td>.15</td>
<td>1.05</td>
</tr>
<tr>
<td>MASQ-SF</td>
<td>.32</td>
<td>2.23*</td>
</tr>
<tr>
<td>PSWQ-PM</td>
<td>.56</td>
<td>3.70**</td>
</tr>
<tr>
<td>ACS</td>
<td>.41</td>
<td>3.12**</td>
</tr>
<tr>
<td>AAQ</td>
<td>.44</td>
<td>2.80**</td>
</tr>
<tr>
<td>IUS</td>
<td>.12</td>
<td>.74</td>
</tr>
<tr>
<td><strong>Predictor: ACS</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SDS</td>
<td>.48</td>
<td>2.99**</td>
</tr>
<tr>
<td>LPR</td>
<td>.40</td>
<td>2.23*</td>
</tr>
<tr>
<td>PDS</td>
<td>.37</td>
<td>2.10*</td>
</tr>
<tr>
<td>MASQ-SF</td>
<td>.58</td>
<td>3.81***</td>
</tr>
<tr>
<td>PSWQ-PM</td>
<td>.60</td>
<td>4.37***</td>
</tr>
<tr>
<td><strong>Predictor: AAQ</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SDS</td>
<td>.47</td>
<td>3.05**</td>
</tr>
<tr>
<td>LPR</td>
<td>.46</td>
<td>2.99**</td>
</tr>
<tr>
<td>PDS</td>
<td>.50</td>
<td>3.28**</td>
</tr>
<tr>
<td>MASQ-SF</td>
<td>.42</td>
<td>2.85**</td>
</tr>
<tr>
<td>PSWQ-PM</td>
<td>.53</td>
<td>3.86**</td>
</tr>
</tbody>
</table>
Note. $AB^* =$ indirect effect (standardized); $ab^* =$ indirect effect (unstandardized); $BC_a$ 99% CI$^*$ = bias-corrected-and-accelerated 99% confidence interval around $ab^*$; ACS = Affective Control Scale; AAQ = Acceptance and Action Questionnaire; SDS = Sheehan Disability Scale; LPR = Loss of Psychosocial Resources; Agency = LPR Loss of Agency subscale; LPR Support = LPR Loss of Support Subscale; PDS = Posttraumatic Stress Diagnostic Scale (Symptom Severity); MASQ-SF = Mood and Anxiety Symptom Questionnaire; PSWQ-PM = Penn State Worry Questionnaire-Past Month; GADQ-IV = Generalized Anxiety Disorder Questionnaire-IV.

* $p < .05$. ** $p < .01$. *** $p < .001$. 
Figure Captions

Figure 1. Assessment schedule and sample sizes.

Note. AEE = Assessment of Exposure to the Events; ACS = Affective Control Scale; AAQ = Acceptance and Action Questionnaire; IUS = Intolerance of Uncertainty Scale; SDS = Sheehan Disability Scale; LPR = Loss of Psychosocial Resources; PDS = Posttraumatic Stress Diagnostic Scale (Symptom Severity); MASQ-SF = Mood and Anxiety Symptom Questionnaire; PSWQ-PM = Penn State Worry Questionnaire-Past Month; GADQ-IV = Generalized Anxiety Disorder Questionnaire-IV; BDI-II = Beck Depression Inventory-II.

Figure 2. Two conceptual models for predicting negative posttraumatic outcome from preattack analogue generalized anxiety disorder and hypothesized mediators, controlling for high exposure to the 9/11 attacks.

Note. a, b, and c, and c’ represent the unstandardized regression coefficients for their respective direct paths after controlling for high exposure. GADQ-IV = GAD Questionnaire for DSM-IV.