Accounting for Posttraumatic Stress Disorder Symptom Severity With Pre- and Posttrauma Measures: A Longitudinal Study of Older Adults

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Abstract
Using data from a longitudinal study of community-dwelling older adults, we analyzed the most extensive set of known correlates of posttraumatic stress disorder (PTSD) symptoms obtained from a single sample to examine the measures' independent and combined utility in accounting for PTSD symptom severity. Fifteen measures identified as PTSD risk factors in published meta-analyses as well as 12 theoretically and empirically supported individual difference and health-related measures were included in our analysis. Individual difference measures assessed after the trauma, including insecure attachment and factors related to the current trauma memory, such as self-rated severity, event centrality, frequency of involuntary recall, and physical reactions to the memory, accounted for symptom severity better than did measures of pretrauma factors. In an analysis restricted to prospective measures assessed before the trauma, the total variance explained decreased from 56% to 16%. Results support a model of PTSD in which characteristics of the current trauma memory promote the development and maintenance of PTSD symptoms.

We know a great deal about the multiple interacting factors that affect posttraumatic stress disorder (PTSD) symptom severity from studies that have examined these factors in isolation or in the context of a few other factors. What remains unknown, however, is how these factors combine to explain PTSD symptom severity. An understanding of the relative contributions of these factors to PTSD symptom severity requires research in which these factors are measured in the same study using a single sample. Such studies ideally would include all major constructs researchers have agreed on as factors that increase PTSD symptom severity on the basis of their inclusion either in PTSD meta-analyses or in other supporting literature. The effect size for each factor could then be directly compared in the same participants, and multiple regression analyses could show how the factors relate to each other as well as how they combine to account for

PTSD symptom severity. Such information cannot be derived from meta-analyses that pool data from across multiple studies that test different participant samples. This information can be obtained only from studies in which the same factors are measured in the same participants. A longitudinal variant of such a study would indicate which correlates of PTSD symptom severity are predisposing factors that promote the development of symptoms following later trauma and which factors increase the severity of symptoms after the trauma occurs.

Studies in which researchers test comprehensive sets of factors that combine to explain PTSD symptom severity in

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a single sample would have important theoretical and clinical value. At a theoretical level, these studies would allow broad classes of factors that are central to theoretical debates concerning PTSD to be compared directly. These include factors that measure properties of the trauma on which a diagnosis is made versus factors that measure an individual's lifetime history of trauma exposure, predisposing factors measured before the trauma occurred versus factors measured during and after the trauma, and factors that are properties of the memory of the trauma versus factors that are stable properties of the individual. Because some of these factors are modifiable after a trauma, such studies would also be of clinical value in that they would provide empirical evidence of factors that are most likely to be important in treating PTSD. Their relative strength in correlating with PTSD symptom severity should indicate the most promising candidates to consider for modification in therapy. If two factors both correlate highly with PTSD severity but not with each other, then combining them in designing a therapy might also be considered.

The type of studies described thus far would not involve exploratory data fishing. The field has and is rapidly accumulating knowledge about PTSD that can be used to guide the selection of factors to be included in such studies. Such studies would also not replace meta-analyses, and each would have the limitations of any single study that includes particular measures assessed in a single sample. However, if several studies of this kind were conducted by research groups with different interests and theoretical biases, the field would have a more advanced scientific understanding of PTSD and stronger empirical knowledge of the factors that might be the most promising candidates for modification in therapy. We offer one such study here as an example of a general method for advancing cumulative knowledge that has major advantages over what currently exists; a method that could help to provide a more adequate empirical base to understand an inherently complex disorder with many causes, effects, and contributing factors.

Data were drawn from a large nonclinical longitudinal study of older adults in their mid-60s, an age-group that is underrepresented in studies of PTSD. Community-dwelling older adults are an ideal population in which to examine factors that influence the development and maintenance of PTSD symptoms because they have lived long enough to potentially be exposed to many types of trauma, they generally have intact cognitive abilities to report their experiences, and they are less likely to be suffering from chronic disease than older generations. Testing older adults also enabled us to examine PTSD symptoms associated with traumatic events experienced at different points throughout the life course, including childhood traumas. This is an important extension of previous research, given that meta-analyses of studies concerning risk factors for PTSD (Brewin, Andrews, & Valentine, 2000; Orth & Weiland, 2006; Ozer, Best, Lipsey, & Weiss, 2003) were restricted to studies of adulthood trauma despite evidence that PTSD symptoms related to early life trauma can persist for decades and cause greater distress than adulthood trauma (Cloitre et al., 2009; Colbert & Krause, 2009; Hiskey, Luckie, Davies, & Brewin, 2008; Ogle, Rubin, & Siegler, 2013a). Testing older adult members of a longitudinal study also enabled us to prospectively examine the relations between factors measured before the target trauma occurred and subsequent PTSD symptom severity, which allowed us to provide a stronger test of the direction of causality.

The factors included in our analyses were selected on the basis of the strength of empirical support for each factor in the extant PTSD literature. We included measures of nearly all constructs examined in meta-analyses of PTSD risk factors[1] (Brewin et al., 2000; Orth & Weiland, 2006; Ozer et al., 2003), including gender, annual family income, education, minority ethnic status, psychiatric history, history of childhood abuse, previous traumas, posttrauma exposure, age at trauma, family history of psychopathology, trauma severity, perceived life threat, hostility, posttrauma social support, and peritraumatic emotional responses. Because the empirical support for each of these constructs has been reviewed in detail in the published meta-analyses, we do not repeat their descriptions here. The only factor that was included in the meta-analyses but not in our study is a measure of peritraumatic dissociation examined by Ozer et al. (2003). However, given that peritraumatic dissociation is a posttrauma assessment of cognition and emotion experienced during the traumatic event, reports of peritraumatic dissociation may also reflect reconstructive processes at the time of recall. Thus, in place of peritraumatic dissociation, we included a measure of the phenomenological properties of an individual's current memory of the trauma that have been linked to elevated PTSD symptoms (reviewed in detail later). It is important to note that this measure does not make claims about what individuals thought or felt in the past. With the exception of peritraumatic dissociation, we tested all constructs that were shown to account for more than 1% of the variance in PTSD symptoms or diagnosis by Brewin et al. (2000), Orth and Wieland (2006), and Ozer et al. (2003) in a single sample.

Our analyses also include measures of several individual difference, trauma memory–related, and behavioral health–related factors that are associated with an elevated risk of PTSD but were not included in the meta-analyses of Brewin et al. (2000), Orth and Weiland (2006), and Ozer et al. (2003) largely because empirical support for these measures has grown substantially in the years

since the meta-analyses were completed. Consistent with our goal of developing more comprehensive models of PTSD that take into account accumulating evidence concerning factors that contribute to the disorder, we included 12 of these theoretically and empirically supported measures in our analyses. The addition of these measures to models that also include the risk factors identified in previous meta-analyses allowed us to compare their explanatory power with other factors known to influence the development or maintenance of PTSD symptoms.

Support for the first of these measures, insecure attachment, is drawn from research with diverse clinical and nonclinical samples of trauma-exposed adults, including military veterans (Dieperink, Leskela, Thuras, & Engdahl, 2001; Pietrzak & Cook, 2013; Solomon, Ginzburg, Mikulincer, Neria, & Ohry, 1998), political prisoners (Kanninen, Punamaki, & Quota, 2003), adults with histories of child abuse (Alexander et al., 1998; Muller, Sicoli, & Lemieux, 2000), and survivors of terrorist attacks (Fraley, Fazzari, Bonanno, & Dekel, 2006), in which strong associations have emerged between insecure attachment and elevated PTSD symptom severity. *Attachment anxiety*, one dimension of insecure attachment, is associated with hyperactivating coping strategies that result in the amplification of negative emotional reactions to stressful events and the exaggeration of threats and related distress (Mikulincer & Shaver, 2007; Mikulincer, Shaver, & Pereg, 2003). *Attachment avoidance*, the second dimension of insecure attachment, is characterized by discomfort with intimacy and interpersonal emotional involvement as well as deactivating emotion-regulation strategies that involve efforts to inhibit or minimize perceptions of threat and feelings of distress (e.g., Fraley & Shaver, 1997; Maunder, Lancee, Nolan, Hunter, & Tannenbaum, 2006). Overall, insecure attachment and the maladaptive emotion-regulation strategies associated with it are thought to undermine individuals' abilities to psychologically adapt to traumatic events and increase vulnerability to PTSD symptoms (Mikulincer & Shaver, 2007; Mikulincer, Shaver, & Horesh, 2006). In the present study, we expected both dimensions of insecure attachment to predict greater PTSD symptom severity.

In related research on personality and PTSD, individuals who score higher on measures of neuroticism have also been shown to report elevated levels of PTSD symptoms after exposure to traumatic events (Breslau & Schultz, 2013; Cox, MacPherson, Enns, & McWilliams, 2004; Holeva & Tarrier, 2001; Ogle, Rubin, & Siegler, 2013b; Parslow, Jorm, & Christensen, 2006; Rubin, Boals, & Berntsen, 2008; Rubin, Boals, & Hoyle, 2014; Rubin, Dennis, & Beckham, 2011). Neuroticism, an enduring disposition characterized by high negative affect, is thought

to increase the availability of memory for stressful events (Rubin, Berntsen, & Bohni, 2008) and the tendency to interpret neutral or ambiguous stimuli in a negative or threatening manner (Costa & McCrae, 1990). Such information-processing biases may, in turn, promote the development and maintenance of PTSD symptoms (Frazier et al., 2011). In the present study, higher neuroticism was expected to predict greater PTSD symptom severity.

Research concerning individuals' evaluations of their ability to cope with stress has suggested that self-rated coping efficacy is another important source of variability in posttraumatic adjustment. Negative self-evaluations of coping ability have been shown to predict greater posttraumatic distress after a wide range of traumas, including natural disasters (Benight & Harper, 2002), terrorist attacks (Benight et al., 2000), and motor vehicle accidents (Benight, Cieslak, Molton, & Johnson, 2008). In the present study, we expected individuals with a lower perceived ability to cope with stress to report more severe PTSD symptoms.

Event centrality, or the extent to which individuals perceive traumas to be important to their identity and life story, has also emerged as a robust predictor of PTSD symptoms in a wide range of participant samples (Berntsen & Rubin, 2006; Berntsen, Rubin, & Sieger, 2011; Blix, Solberg, & Heir, 2014; Boals, Hayslip, Knowles, & Banks, 2012; Robinaugh & McNally, 2011), even with potentially confounding factors, such as personality, dissociation, anxiety, depression, and type of trauma controlled (Berntsen & Rubin, 2007; Brown, Antonius, Kramer, Root, & Hirst, 2010; Rubin et al., 2014). Greater event centrality is thought to increase PTSD severity by enhancing the emotional salience of the trauma memory as well as the frequency and ease with which the memory comes to mind (Berntsen & Rubin, 2006). Repeatedly reexperiencing the trauma through vivid intrusive memories or flashbacks that are typical of PTSD may also promote integration of the trauma memory into one's autobiographical narrative, thereby increasing the likelihood that the event will be perceived as a highly salient turning point in the life story (Thomsen & Berntsen, 2008).

The remaining trauma memory–related measures included in our analyses concern phenomenological properties of trauma memories. According to the autobiographical memory theory of PTSD (Rubin, Berntsen, & Bohni, 2008), particular properties of trauma memories, including the intensity of individuals' emotional and physical reactions to memories of their trauma as well as the frequency of involuntary recall and voluntary rehearsal, are the central mechanisms that promote the development and maintenance of PTSD symptoms. Support for this theoretical model of PTSD has been

found in numerous empirical studies (Berntsen, Willert, & Rubin, 2003; Ogle, Rubin, & Siegler, 2015; Rubin et al., 2011; Rubin, Feldman, & Beckham, 2004; Talarico & Rubin, 2003). On the basis of these findings, we included the following measures in our prediction models: participant ratings of the intensity of their emotional reactions while remembering the trauma (emotional intensity), the extent of their visceral reactions while remembering the trauma (physical reactions), and the frequency of voluntary rehearsal and involuntary recall of their trauma memories.

Three behavioral-health measures were also included in our prediction models on the basis of studies in which researchers have documented their association with PTSD symptoms or diagnosis. The first of these measures, sleep disturbances, has been implicated in both the development and the maintenance of PTSD symptoms. Subjective reports of sleep disturbances after trauma exposure have been shown to predict greater symptom severity (Fairholme et al., 2013) and diagnosis (Harvey & Bryant, 1998; Koren, Amon, Lavie, & Klein, 2002). In addition, prospective longitudinal studies have indicated that sleep disturbances prior to experiencing a traumatic event increase the risk of subsequent PTSD (Koffel, Polusny, Arbisi, & Erbes, 2013; van Liempt, van Zuiden, Westenberg, Super, & Vermetten, 2013; for a review, see Germain, 2013). Collectively, these findings suggest that sleep disturbances may act as a predisposing factor for PTSD, as opposed to their current status as a secondary symptom, perhaps as a result of the impact of impaired sleep on emotion regulation (Harvey, Murray, Chandler, & Soehner, 2011) or fear conditioning (Pace-Schott et al., 2009).

A second behavioral-health measure, lifetime smoking history, was included in our prediction models on the basis of substantial empirical evidence of the frequent co-occurrence of nicotine use and PTSD symptoms and diagnosis (e.g., Fu et al., 2007; McClernon, Calhoun, Hertzberg, Dedert, & Beckham, 2013). Smoking has also been shown to prospectively predict greater PTSD symptom severity (van der Velden, Grievink, Olff, Gersons, & Kleber, 2007). For instance, in a prospective study of emergency rescue workers, smoking independently predicted PTSD symptoms after pretrauma and posttrauma confounding variables, including alcohol consumption and previous trauma exposure, were controlled (van der Velden, Kieber, & Koenen, 2008). Genetic research has further suggested that trauma-exposed individuals with histories of nicotine dependence may be at an increased risk of PTSD (Koenen et al., 2005).

The third behavioral-health measure examined in our prediction models, subjective physical health, was selected on the basis of research that has shown that poor subjective physical health is associated with PTSD symptoms in older adults (Pietrzak, Goldstein, Southwick,

& Grant, 2012; van Zelst, de Beurs, Beekham, Deeg, & van Dyck, 2003) and that declines in physical health status can trigger symptoms or precipitate the reemergence of posttraumatic distress (Hiskey et al., 2008; Kaup, Ruskin, & Nyman, 1994). We expected lower subjective physical health to predict greater PTSD symptom severity in the present study. Overall, our ability to examine the associations between PTSD symptoms and health-related variables in relation to other known psychosocial factors associated with PTSD allows us to test a unique and relatively comprehensive model of factors related to PTSD.

In summary, in this study, we examined an extensive set of factors associated with PTSD symptoms or diagnosis to determine the relative contribution of each factor in explaining PTSD symptom severity. Our experimental design enabled us to extend previous meta-analytic research on PTSD risk factors (Brewin et al., 2000; Orth & Wieland, 2006; Ozer et al., 2003) and related studies (e.g., Ehring, Ehlers, & Glucksman, 2008; Kleim, Ehlers, & Glucksman, 2007) in several ways. First, because data for each factor were drawn from a single sample, we were able to examine the isolated contribution of each factor in univariate analyses in the same sample, as well as how the factors combine to predict PTSD symptom severity in multiple regression analyses. Second, consistent with the goal of developing more comprehensive models of PTSD symptom severity that take into account recent accumulating evidence concerning a wide range of factors, our prediction models included 15 factors shown to correlate with PTSD symptom severity in the published PTSD meta-analyses (Brewin et al., 2000; Orth & Wieland, 2006; Ozer et al., 2003), in addition to 12 theoretically and empirically supported individual difference, trauma memory–related, and behavioral-health factors that have received substantial empirical support since the PTSD meta-analyses were published. Third, in contrast to previous researchers who have primarily focused on PTSD symptoms related to specific types of traumas encountered in adulthood (e.g., motor vehicle accidents, assault; Ehring et al., 2008; Kleim et al., 2007), we examined the severity of symptoms linked to a broad range of traumatic events experienced throughout the life course.

Finally, because most of the factors examined in the PTSD meta-analyses (Brewin et al., 2000; Orth & Wieland, 2006; Ozer et al., 2003) were measured after the traumas occurred, the reported associations between the identified risk factors and elevated PTSD symptoms support their role as correlates of PTSD symptoms rather than as causal factors. To address this limitation and to provide a stronger test of the direction of causality between contributing factors and PTSD symptom severity, we used longitudinal data in a second analysis to prospectively examine the relations between a subset of constructs (drawn from the full set of constructs described earlier) measured before

the target trauma occurred and subsequent PTSD symptoms. Pretrauma factors shown to have consistent effects on the risk of PTSD by Brewin et al. (2000) and Ozer et al. (2003) include a family history of psychopathology and a history of childhood abuse. In addition to these factors, our prospective analysis included pretrauma assessments of hostility, neuroticism, smoking history, subjective physical health, previous traumas, history of depression diagnosis, and depressive symptoms.

Method

Participants and procedure

Data were drawn from the University of North Carolina Alumni Heart Study (UNCAHS), a longitudinal mail survey study of students who entered the University of North Carolina, Chapel Hill, in 1964 to 1966 (Siegler et al., 1992). With 13 points of data collection now complete, the UNCAHS contains an extensive archive of data concerning personality, well-being, and disease outcomes. The recruitment procedures, participation rates, and measures of the UNCAHS are detailed elsewhere (Hooker, Hoppmann, & Siegler, 2010) and briefly described here. All waves of the UNCAHS were approved by the Duke University Medical Center institutional review board. Relevant to the present report, on the Wave 13 questionnaire, participants were asked to describe three traumatic events that currently bothered them most, to select a category that best described each traumatic event from the list of events included in the Traumatic Life Events Questionnaire (TLEQ; Kubany et al., 2000), and to report their age at the time of each event. For each of the three traumatic events, participants also completed measures of PTSD symptom severity, event centrality, and the phenomenological properties of the trauma memory.

Participants were selected to be included in the primary analysis sample if they completed the measure of PTSD symptoms at Wave 13 ($n = 1,186$; 76.42% of respondents who participated in Wave 13). The final sample was 61.30% male and 38.70% female with a mean age of 63.43 years ($SD = 2.78$) and a mean annual household income of $70,000 to $99,999. The sample was also predominantly Caucasian (98.23%). Five participants were African American (0.42%), 2 American Indian (0.17%), 1 Hispanic (0.08%), and 13 (1.10%) declined to report their ethnicity. Approximately 9% had less than a college degree, 20% had bachelor's degrees, 23% had bachelor's degrees plus additional training, 27% had master's degrees, and 22% had advanced degrees. Comparisons of respondents included in the primary analysis sample with those who were excluded from analysis because they declined to provide data on PTSD symptoms indicated that the groups did not differ on demographic characteristics, including education, ethnicity, or income. Participants in the primary analysis sample were slightly older and included more females. (Table S1 in the Supplemental Material available online includes comparisons of included versus excluded respondents on all study variables.)

The prospective analysis was limited to a subset of participants ($n = 645$) whose traumas occurred after Wave 7 (1997–1998) and to data from measures assessed up to and including Wave 7. We chose this cutoff because it retained the maximum number of participants in the analysis while enabling us to examine key constructs. The demographic characteristics of the prospective analysis subsample were nearly identical to those reported for participants in the primary analysis (see Table S1 in the Supplemental Material).

Measures

Table S1 in the Supplemental Material provides the dates when each construct was measured in the primary sample and the prospective analysis subsample.

Adult attachment. Attachment anxiety and avoidance were assessed at Wave 13 using the 12-item short form of the Experiences in Close Relationships Inventory (ECR-S; Wei, Russell, Mallinckrodt, & Vogel, 2007). Participants rated the extent to which each item generally describes their feelings in close relationships on a 7-point scale ranging from 1 (*disagree strongly*) to 7 (*agree strongly*). Higher scores on the Anxiety and Avoidance subscales indicate greater levels of attachment anxiety and avoidance, respectively. The ECR-S has been shown to have strong psychometric properties, including high test-retest reliability (*r*s at or above .82) and high construct validity (Wei et al., 2007).

Childhood abuse. Participants' histories of childhood abuse were assessed using items from the TLEQ (Kubany et al., 2000), a measure of lifetime exposure to potentially traumatic events included in Wave 12. On the TLEQ, participants reported how often they experienced a broad spectrum of events capable of producing PTSD symptoms, with responses provided on a 7-point scale from 0 (*never*) to 6 (*more than 5 times*), and their age at the worst occurrence of each event type. The TLEQ has strong psychometric properties and high convergent validity with structured clinical interviews (Kubany et al., 2000). Items concerning childhood physical abuse and sexual assault were combined to create a dichotomous measure of childhood abuse.

Coping. Perceived coping ability was assessed at Wave 13 using a measure of health and well-being in late and

middle age (Siegler, 2004). Respondents rated their "current ability to cope with stress" on a 4-point scale ranging from 1 (*poor*) to 4 (*excellent*).

Demographic characteristics. Education was coded using an 8-point scale from 1 (*less than a high school diploma*) to 8 (*doctorate/law/medical degree*). Minority ethnic status (1 = ethnic minority, 0 = Caucasian) and female gender (1 = female, 0 = male) were dichotomously coded.

Depression. Depressive symptoms were measured in the primary analysis sample at Wave 11 using the Prime MD (Kroenke, Spitzer, & Williams, 2001), a commonly used screening tool for depression. Nine symptoms during the past 2 weeks are rated on 4-point scales ranging from 0 (*not at all*) to 3 (*nearly every day*). Total scores higher than 11 indicate major depressive disorder. Depressive symptoms in the prospective analysis subsample were measured at Wave 6 by the Center for Epidemiological Studies Depression Scale (CES-D; Radloff, 1977). Respondents rated how often they experienced 20 depressive symptoms in the previous week on 4-point scales from 0 (*rarely or none of the time*) to 3 (*most or all of the time*). Total scores higher than 21 indicate possible major depressive disorder.

Participants reported their history of depression diagnosis at every wave of the UNCAHS except Wave 9. Participants were dichotomously classified as with (= 1) or without (= 0) a history of depression diagnosis on the basis of these cumulative reports. Reports of depression diagnoses prior to Wave 8 were included in the prospective analysis.

Event centrality. The Centrality of Event Scale (CES; Berntsen & Rubin, 2006) assesses the extent to which a trauma forms a central component of personal identity, a turning point in the life story, and a reference point for everyday inferences. Items are rated on 5-point scales ranging from 1 (*totally disagree*) to 5 (*totally agree*). Respondents completed the seven-item short version of the CES at Wave 13. Mean scores are reported.

Hostility. Hostility was measured at Wave 11 using a 37-item version of the Cook-Medley Hostility Scale (Cook & Medley, 1954) that included four subscales (i.e., Cynicism, Hostile Attitude, Hostile Attribution, Aggressive) derived by Barefoot, Dodge, Peterson, Dahlstrom, and Williams (1989). The prospective analysis included hostility data collected at Wave 2 for original UNCAHS members and at study enrollment (1992) for spouses.

Maternal and paternal psychopathology. Data concerning maternal and paternal psychiatric history were drawn from Wave 10. Participants reported whether their mother and father were ever diagnosed with depression or anxiety.

Neuroticism. Neuroticism measured using the NEO Personality Inventory (NEO-PI; Costa & McCrae, 1992) at Wave 7 was analyzed. Raw scores were converted to *t* scores based on adult combined-sex norms. The NEO-PI has strong psychometric properties (Cronbach's α = .92) and is well validated (Costa & McCrae, 1992).

Phenomenological properties of trauma memories. Phenomenological properties of trauma memories were assessed at Wave 13 using the Autobiographical Memory Questionnaire (AMQ; Rubin, Schrauf, & Greenberg, 2003, 2004). Questions concerning processes involved in remembering an event are rated on 7-point scales from 1 (*not at all*) to 7 (*completely*). In the UNCAHS, participants were asked to complete the AMQ while they recalled their target trauma. Our analyses included four items from the AMQ that correlate consistently with PTSD symptom severity (Rubin et al., 2011), including emotional intensity ("While remembering the event, the emotions that I feel are extremely intense"), physical reaction ("While remembering the event, I had a physical reaction [laughed, felt tense, felt sweaty, felt cramps or butterflies in my stomach, my heart pounded or raced, etc.]"), voluntary rehearsal ("Since it happened, I have willfully thought back to the event in my mind and thought about it or talked about it"), and involuntary recall ("Has the memory of the event suddenly popped in your thoughts by itself—that is, without having attempted to remember it?").

Previous traumas and posttrauma exposure. The TLEQ (Kubany et al., 2000) was used to assess lifetime exposure to potentially traumatic events at Wave 12. Events that occurred before and after the target trauma (excluding childhood abuse) were summed separately to create indices of previous traumas and posttrauma exposure.

PTSD symptom severity. The PTSD Checklist–Stressor Specific Version (PCL-S; Weathers, Litz, Huska, & Keane, 1994), a 17-item PTSD screening instrument, was used to measure PTSD symptom severity at Wave 13. Using 5-point scales from 1 (*not at all*) to 5 (*extremely*), respondents indicated the extent to which a specific event produced each of the B, C, and D *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev.; *DSM–IV–TR*; American Psychiatric Association, 2000) PTSD symptoms during the previous month. The PCL-S has strong psychometric properties (Blanchard, Jones-Alexander, Buckley, & Forneris, 1996) and high diagnostic agreement with the Clinician-Administered PTSD Scale (Blake et al., 1990).

Self-reported **DSM–IV–TR** ***A1 and A2 PTSD criteria.*** Participants rated their trauma using the *DSM–IV–TR* A1 ("Did the event involve actual or threatened death, serious injury, or threat to the physical integrity of yourself or others?") and A2 ("Did you experience intense fear, helplessness, or horror when it happened?") PTSD criteria at Wave 13. These measures are comparable with the indices of perceived life threat and peritraumatic emotional response examined by Ozer et al. (2003).

Self-reported event severity. Event severity was assessed using the Four Kinds of Damages Scale (Rubin & Feeling, 2013) at Wave 13. Using 7-point scales ranging from 1 (*negligible*) to 7 (*as much as any event I could imagine*), participants answered questions concerning the extent of physical, emotional, financial, and future damage caused by the trauma. Total scores were analyzed.

Sleep disturbances. The frequency of various types of sleep disturbances was assessed at Wave 12. On 3-point scales ranging from 0 (*never*) to 2 (*almost all of the time*), participants rated how often they experienced trouble falling asleep, waking during the night, waking too early, restless sleep, and daytime drowsiness during the past month. Summed scores were analyzed.

Smoking history. Lifetime smoking history was assessed at Waves 1, 3, 6, 9, and 13 of the UNCAHS. Participants were dichotomously coded as ever smoked (= 1) or never smoked (= 0) on the basis of these cumulative reports. Smoking history up to and including Wave 6 was included in the prospective analysis.

Social support. Posttrauma social support was assessed at Wave 13 using a measure of health and well-being in late and middle age (Siegler, 2004). Respondents rated the support they receive from persons close to them on a 4-point scale from 1 (*poor*) to 4 (*excellent*).

Subjective physical health. Respondents rated their current health on a 4-point scale ranging from 1 (*poor*) to 4 (*excellent*) at Wave 13. The prospective analysis included ratings of subjective physical health collected at Wave 6.

Data analysis

We used multiple imputation (Rubin, 1987) to manage missing data (see Table S1 in the Supplemental Material for frequencies of missing values for each study variable). Results from each of 10 imputed data sets were combined to produce a single set of estimates and standard errors.

Our analyses focused on the traumatic event with the highest PCL-S total severity score for each participant. We first report zero-order correlations between each construct and PTSD symptom severity to provide measures of effect size that can be compared with existing PTSD meta-analyses. Next, predictors were tested in a stepwise multiple regression model to identify those that explain unique variance in PTSD symptom severity. Multiple regression was selected as our analysis strategy because it best fits the limits of our data set, which contains only one measure or standardized scale for most of the PTSD-related factors included in the present study. Given that no overarching theory currently exists to explain how these factors combine to account for variability in posttraumatic stress reactions, multiple regression also best fits the limits of our current empirical knowledge. In the prospective analysis, predictors were restricted to pretrauma measures among a subset of participants whose traumas occurred after Wave 7 to provide a stronger test of the direction of causality between predictors and PTSD symptom severity. In each model, all variables significant in the univariate analyses at $p \leq .05$ were entered and deleted using backward elimination with probability-to-remove set to .05. The level of multicollinearity in the models was low (variance inflation factors at or below 1.79).

Results

The events included in the analyses represented a wide range of potentially traumatic events. Prevalence rates were highest for unexpected death of a loved one (24.87%), followed by life-threatening personal illness (10.46%), life-threatening or disabling accident or illness of a loved one (10.12%), accident that badly injured self or killed someone (6.07%), motor vehicle accident that badly injured self or killed someone (5.82%), natural disaster that badly injured self or killed someone (3.96%), warfare or combat (2.11%), death threat (1.43%), childhood physical abuse (1.35%), childhood sexual abuse (1.18%), and witnessing childhood family violence (1.01%). Less than 1% of the sample reported each of the following events: experiencing or witnessing an armed robbery, physical assault by a stranger, witnessing assault or murder, physical assault by a partner, adulthood sexual assault, being stalked, and non-live-birth pregnancy. In addition, 23.61% of participants reported a life-threatening or highly disturbing event that did not fit these categories, 1.26% reported an experience they did not wish to name, and 3.1% of respondents did not select a category for their trauma. The mean PCL-S total severity score was 30.40 (*SD* = 12.77, range = 17–85). Prior research with the UNCAHS study sample has indicated that the distribution of potentially traumatic

events in the UNCAHS is typical of what might be expected for a population of mixed ages and includes frequencies of canonical traumatic stressors that are comparable with results from studies of nationally representative samples (e.g., Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). For example, 9.73% of the UNCAHS study sample reported sexual assault, 8.28% reported warfare or combat, and 17.32% reported a form of interpersonal violence (i.e., physical assault by partner, physical assault by a stranger, childhood physical abuse; Ogle, Rubin, Berntsen, & Siegler, 2013).

Descriptive information and correlations between PTSD symptom severity and factors that account for it are reported in Table 1 (see Table S2 in the Supplemental Material for correlations among all study variables). Significant associations emerged between PTSD symptom severity and all factors with the exception of demographic characteristics, including age, education, and ethnicity, as well as age at trauma, smoking history, and the self-reported *DSM–IV–TR* A1 criterion. Predictors that explained unique variance in PTSD symptom severity in the final model of the multiple regression analysis are shown in the right-most column. Overall, the model explained 56% of the variance in PTSD symptom severity, $F(12, 1185) = 125.69$, $p < .001$.[2] Because involuntary recall, physical reactions to the trauma memory, the emotional intensity of the trauma memory, and hostility can be viewed as related to items on the PCL-S, we tested a regression model with the four PCL-S items related to these predictors removed from the total PCL-S score to ensure that our results were not being driven by potential content overlap. Removal of these items did not substantially decrease the percentage of variance explained ($R^2 = .52$). Thus, the predictive value of the phenomenological characteristics of the trauma memory and hostility remained strong after we accounted for content overlap.

Table 2 contains descriptive information and correlations between PTSD symptom severity and each factor included in the prospective analysis. Results from the final model of the regression are shown in the right-most column. Overall, the model explained 16% of the variance in PTSD symptom severity, $F(4, 644) = 29.47$, $p < .001$.[3]

Discussion

In the present study, we examined the relations between current PTSD symptom severity and an extensive set of factors associated with PTSD symptoms in a nonclinical sample of older adults. Because the longitudinal study from which our data were drawn contained measures of nearly all of the constructs identified as PTSD risk factors in previous meta-analytic research in addition to several individual difference and health-related variables shown to predict symptoms and diagnosis in the adult PTSD

literature, we were able to test a relatively comprehensive set of factors in a single model to determine their unique and combined utility in explaining PTSD symptom severity. Overall, individual difference measures assessed after the target trauma occurred, including insecure attachment, depressive symptoms, and a lower perceived ability to cope with stress, in addition to measures related to the current trauma memory, including self-rated severity, centrality to identity, frequency of involuntary recall, and the strength of visceral reactions to the memory, emerged as strong statistical predictors of PTSD symptom severity, whereas pretrauma factors, such as sociodemographic characteristics and parental history of psychopathology, did not. These findings are consistent with results from meta-analyses of PTSD risk factors in showing that factors that are more temporally proximal to the traumatic exposure better account for variance in PTSD symptom severity than do factors that are more distal to the trauma. For instance, Ozer et al. (2003) found that posttrauma risk factors, including reports of event severity and peritraumatic emotionality, had larger effect sizes than did pretrauma risk factors, including previous trauma exposure and prior psychological adjustment. Likewise, Brewin et al. (2000) found that posttrauma social support and life stress encountered after the trauma were the strongest correlates of PTSD severity. Of the 12 factors that explained unique variance in PTSD symptom severity in the present study, only one—history of depression diagnosis—could be classified as a characteristic of the individual's life that may have predated the target trauma.

Our findings also support memory-based theories of PTSD in which characteristics of the individual's current memory of the trauma promote the development and maintenance of PTSD symptoms (Rubin, Berntsen, & Bohni, 2008; Rubin, Boals, & Berntsen, 2008; Rubin et al., 2011). Specifically, the autobiographical memory theory of PTSD contends that the intensity of the trauma memory, the frequency of voluntary and involuntary recall, and the centrality of the memory to the person's identity combine to account for PTSD symptoms by increasing the rehearsal and maintenance of the trauma memory and by reciprocally promoting each other. Consistent with this theoretical model, results from the present study showed that the frequency of involuntary recall, the intensity of physical reactions to the trauma memory, and event centrality emerged as unique predictors of PTSD symptoms in the multivariate analyses. Notably, none of these constructs were examined in the extant meta-analyses of PTSD risk factors, in large part because earlier studies did not include them. Furthermore, in contrast to many of the risk factors identified by Brewin et al. (2000), Orth and Weiland (2006), and Ozer et al. (2003), such as general childhood adversity and

Table 1. Descriptive Statistics and Results From Multiple Regression Analysis Predicting Posttraumatic Stress Disorder Symptom Severity ($n = 1,186$)

Predictors	M	r with PCL-S	β
Demographic measures			
Age	63.43 (2.78)	.01	
Education	6.27 (1.39)	−.05	
Ethnic minority status (%)	0.67	.05	
Female gender (%)	38.70	.08**	
Annual household income	$70,000–$99,999	−.09**	
Health and trauma history measures			
Maternal psychopathology (%)	17.03	.09**	
Paternal psychopathology (%)	8.52	.08*	
Childhood abuse (%)	15.01	.13***	
Age at trauma	48.32 (16.68)	.05	
Previous traumas	3.42 (4.04)	.15***	
Posttrauma exposure	1.10 (2.76)	.07**	0.04*
Sleep disturbances	3.73 (1.76)	.25***	
History of smoking (%)	54.05	.03	
Subjective physical health	3.30 (0.66)	−.30***	−0.09***
History of depression diagnosis (%)	23.02	.29***	0.07***
Depressive symptoms	2.51 (3.07)	.34***	0.09***
Individual difference measures			
Attachment anxiety	14.45 (5.38)	.39***	0.16***
Attachment avoidance	14.93 (5.67)	.19***	0.08***
Hostility	10.60 (5.99)	.25***	
Neuroticism	48.76 (10.61)	.31***	
Coping ability	3.14 (0.70)	−.32***	−0.06**
Social support	3.34 (0.77)	−.25***	
Memory-related measures			
Self-reported *DSM–IV–TR* A1 (%)	65.26	.02	
Self-reported *DSM–IV–TR* A2 (%)	68.80	.26***	0.05*
Self-rated event severity	15.52 (5.58)	.51***	0.19***
Emotional intensity of memory	5.32 (1.62)	.40***	
Physical reaction to memory	3.65 (2.10)	.49***	0.22***
Voluntary rehearsal	4.98 (1.54)	.42***	
Involuntary recall	4.36 (1.81)	.52***	0.20***
Event centrality	3.27 (1.09)	.52***	0.13***

Note: Standard deviations are shown in parentheses. PCL-S = PTSD Checklist–Stressor Specific Version symptom severity scores; *DSM–IV–TR* = *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev.; American Psychiatric Association, 2000).
*$p \leq .05$. **$p \leq .01$. ***$p \leq .001$.

family history of psychopathology, our findings indicate that the constructs that account for the most variance in PTSD symptoms are amenable to change in therapy. In particular, our findings suggest that intervention and treatment programs targeted at altering particular properties of individuals' trauma memories, such as diminishing the physiological impact of the trauma memory and the centrality of the memory to one's personal identity, may show promise in reducing the severity of PTSD symptoms.

The longitudinal design of the UNCAHS also provided the opportunity to prospectively examine relations between a subset of factors assessed prior to the trauma and subsequent PTSD symptoms. Results from this prospective analysis indicated that childhood abuse, depressive symptoms, hostility, and neuroticism predicted greater PTSD symptom severity. Our focus on factors assessed before the target trauma ensured that measurement of the factors was not influenced by exposure to the target trauma itself or by posttraumatic symptoms. This aspect of our design strengthened the support for the status of depressive symptoms, hostility, and neuroticism as predisposing factors for PTSD symptoms rather than as correlates of trauma exposure or posttraumatic

Table 2. Descriptive Statistics and Results From Multiple Regression Analysis of Prospective Measures of Posttraumatic Stress Disorder Symptom Severity ($n = 645$)

Predictors	M	r with PCL-S	β
Demographic measures			
Age	63.34 (2.92)	−.01	
Education	6.27 (1.31)	−.03	
Ethnic minority status (%)	0.78	.07	
Female gender (%)	39.53	.09*	
Annual household income	$60,000–$69,999	−.07	
Health and trauma history measures			
Maternal psychopathology (%)	16.90	.10**	
Paternal psychopathology (%)	7.13	.08	
Childhood abuse (%)	13.18	.12***	0.08*
Previous traumas	1.96 (1.61)	.12**	
History of smoking (%)	13.33	.04	
Subjective physical health	3.49 (0.58)	−.18***	
History of depression diagnosis (%)	9.15	.15***	
Depressive symptoms	7.74 (7.42)	.33***	0.20***
Individual difference measures			
Hostility	13.34 (7.06)	.19***	0.09*
Neuroticism	48.41 (10.56)	.32***	0.19***

Note: Standard deviations are shown in parentheses. PCL = PTSD Checklist–Stressor Specific Version symptom severity scores.
*$p \le .05$. **$p \le .01$. ***$p \le .001$.

symptoms. Although our finding that depressive symptoms was the only factor to explain unique variance in PTSD symptom severity in both the primary analysis sample and the prospective analysis subsample was somewhat surprising, it should be noted that these two samples varied with respect to both the age distribution of the traumatic events and the types of traumatic events examined. The primary analysis model predicted current PTSD symptoms related to traumas experienced throughout the life course, including categories of traumatic events that can occur only in childhood and adolescence (e.g., childhood abuse), whereas the prospective analysis investigated PTSD symptoms linked to traumas that occurred during a 15-year period in midlife and older adulthood. The differences in the results obtained from these two models might be expected if statistical predictors of PTSD symptoms related to early life traumas are different from predictors of symptoms related to adulthood traumas. Consistent with this idea, results found by Breslau, Davis, and Andrewski (1995) showed that risk factors for exposure to traumatic events varied as a function of whether the trauma occurred in childhood or in adulthood. Additional research is needed to clarify potential differences in predictors of PTSD symptoms related to early life versus adulthood traumas.

Although hostility and neuroticism were both positively associated with PTSD symptom severity in the bivariate correlations, contrary to our expectations,

hostility and neuroticism each explained unique variance in PTSD symptoms in the prospective analysis model but not in the primary analysis model. The reduction in the explanatory power of these personality factors in the primary analysis may have occurred because they were competing with posttrauma memory–related factors that were more strongly associated with PTSD symptoms. Another possible explanation of the differences in the explanatory power of neuroticism and hostility in the primary versus the prospective analysis is that these personality factors may be more strongly related to the development of PTSD symptoms and less implicated in the persistence of PTSD symptomology. This hypothesis is consistent with results from previous research in which differences were found among risk factors for the development of PTSD symptoms compared with risk factors for chronic trajectories of the disorder (Koenen, Stellman, Stellman, & Sommer, 2003; Schnurr, Lunney, & Sengupta, 2004). Future research is needed to systematically examine the role of neuroticism and hostility in the development compared with the maintenance of PTSD.

Strengths and limitations

Strengths of the current study include a large sample, a longitudinal design, and our use of many well-validated measures, including the NEO-PI, the Cook-Medley

Hostility Scale, the CES-D, and the Prime MD. Compared with previous studies of PTSD risk factors, we also tested a more extensive set of factors in a single model, which allowed us to assess the relative predictive utility of each factor in explaining PTSD symptom severity. Another unique aspect of our design was our examination of current PTSD symptoms related to a broad range of traumatic events experienced across the life course, rather than an exclusive focus on adulthood traumas or a single type of traumatic event. Thus, we provide the most comprehensive analysis to date of factors related to PTSD symptoms associated with traumatic events experienced throughout the life course.

Several limitations of the study should also be noted. First, our use of retrospective self-report data concerning traumatic events may have resulted in biased reporting. However, research regarding the validity of retrospective reports indicates that biases typically result in underreporting, at least for childhood traumas (Hardt & Rutter, 2004). Second, the relatively limited educational, ethnocultural, and socioeconomic diversity of the sample restricts the generalizability of our findings to broader community samples. Third, although cross-sectional and prospective research has suggested that low intelligence increases the risk of exposure to traumatic events and the development of PTSD symptoms after exposure (Breslau, Lucia, & Alvarado, 2006; Macklin et al., 1998; McNally & Robinaugh, 2011; McNally & Shin, 1995), intelligence is not directly assessed in the UNCAHS and, therefore, could not be included in the present analysis. It is unlikely that this omission substantially reduced the predictive power of our model, given that low intelligence was among the predictors with the smallest effect sizes in previous meta-analytic research (Brewin et al., 2000) and that the range of intelligence in our study sample may be limited as suggested by the high overall level of education in the UNCAHS. Furthermore, education—which may be considered a proxy measure of intelligence—was not significantly correlated with PTSD symptom severity in the primary analysis or the prospective analysis subsample. Despite these findings, future research should include standardized measures of intelligence.

Fourth, our examination of an older adult sample may limit the generalizability of our results to younger adults, given that different types of traumatic events are more likely to occur during particular periods of the life course. Previous research with the UNCAHS sample has shown that the frequency of unexpected deaths of loved ones increases with age starting in middle adulthood (Ogle et al., 2013). Consistent with this finding, results showed that many older adults in the present study reported that the traumatic event that caused the most severe PTSD symptoms involved the unexpected death of a loved one. Given the symptom overlap between grief and depression, depressive symptom scores included in the present

study may also reflect symptoms of grief. Future research on posttraumatic stress in older adults should incorporate standardized measures of bereavement to distinguish grief symptoms from potential overlapping symptoms of other posttraumatic outcomes, including PTSD and depression.

Fifth, replication of our findings is needed as a result of our analysis of factors that were assessed with few or single items (e.g., coping ability, social support). Although brief assessments are common in epidemiological research because they make it possible to measure a large number of constructs within a single longitudinal cohort study, the unknown validity of these measures may have compromised the reliability of the assessments and underestimated the influence of these factors. Other single-item subjective measures (e.g., self-rated health), however, are well-established predictors of psychological outcomes, including well-being and depression (e.g., Ambresin, Dowrick, Lierrman, & Gunn, 2014; Sneed, Kasen, & Cohen, 2007).

Within the context of these limitations, the present study provides important information on how factors associated with posttraumatic stress combine to explain PTSD symptom severity. Our results indicate that PTSD symptoms are primarily driven by a combination of phenomenological properties of the current trauma memory as well as posttrauma measures of insecure adult attachment, subjective appraisals of event severity, event centrality, and depressive symptoms. Future research should be devoted to identifying the specific processes through which these factors influence the development and persistence of PTSD symptoms.

Author Contributions

All authors contributed to the study design as well as the design and data collection of Wave 13 of the University of North Carolina Alumni Heart Study from which the posttraumatic stress disorder data were drawn. C. M. Ogle analyzed the data and drafted the manuscript. D. C. Rubin and I. C. Siegler provided critical interpretations and revisions. All authors approved the final version of the manuscript.

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Supplemental Material

Additional supporting information may be found at http://cpx .sagepub.com/content/by/supplemental-data

Notes

1. In these meta-analyses, the term *risk factor* was used to refer to constructs that correlate with the development or maintenance of PTSD symptom severity or diagnosis. In the present study, to avoid the implication of causality, we refer to measures that correlate with PTSD symptom severity and that were chosen to be independent variables in our analyses as *factors*. We use *prospective measures* to refer to measures assessed before the target trauma occurred. We use *predictor* in the statistical sense in multiple regression and correlational analyses.

2. Results based on individuals with complete data for all study variables (n = 866) were not substantially different. The total variance explained was 57%. Compared to the results reported in Table 1, the results from this model indicated that sleep disturbances and gender explained unique variance in PTSD symptom severity, whereas posttrauma exposure did not.

3. Results based on individuals with complete data for all study variables (n = 495) were not substantially different. The total variance explained was 15%. Predictors in the final regression model were unchanged across samples.

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