Examination of Complicated Grief, Posttraumatic Stress, and Other Psychological Reactions among Student Survivors of the April 16th Shootings at Virginia Tech

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ABSTRACT

The diagnosis of Complicated Grief (CG) is being proposed for inclusion in *DSM–5*. As such, it has been the focus of several studies purporting to build evidence of its validity and its conceptual and statistical distinction from Posttraumatic Stress Disorder (PTSD) and depression (Bonanno et al., 2007; Golden & Dalgleish, 2010; Prigerson et al., 1995b). However, previous research has focused predominantly on bereavement caused by non-violent means (e.g., prolonged terminal illness). This study attempts to explore the nature of CG among a sample of students who survived a mass shooting. Exploratory and confirmatory factor analytic procedures were used to examine and confirm the factor structure of CG as reported in previous studies (e.g., Boelen & van den Bout, 2005; Dillen, Fontaine, & Verhofstadt-Denève, 2008). A refined CG scale was then used as a criterion to demonstrate how different types of traumatic exposure contribute to symptoms of CG and/or posttraumatic stress (PTS). It was hypothesized that exposure items related to bereavement would be more related to CG than to PTS, whereas direct exposure to the shootings would be more closely related to PTS than to CG. Results of exploratory and confirmatory factor analyses supported CG as a unitary construct distinct from PTS and from anxiety/depression. Logistic regression results demonstrated that bereavement status was significantly predicted by CG but not PTS: Participants who scored 1 standard deviation above the mean on the CG scale were 14.64 times more likely to have been bereaved than were those who scored at the mean. SEM analyses were used to provide an additional test of this hypothesis. The final model had acceptable fit as assessed by RMSEA = .046, CI = .043–.049, SRMR = .048, and CFI = .990; however, the Satorra-Bentler Scaled $\chi^2 = 1507.82$, $df = 589$, $p < .001$, did not support the model. Overall, results of SEM suggested that interpersonal loss (i.e., whether a friend was killed, injured, or escaped from the shootings) predicted CG but not PTS, whereas perceived threat predicted both CG and PTS.

Dedication

This work is dedicated to those who were killed, to those who were injured, and to those who will never forget 4/16.

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As I begin the next phase of my life, please know that each of you has, in his or her way, influenced me greatly and for the better. Thank you.

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Chapter 1 - Introduction

On April 16, 2007, Sueng Hui Cho shot 49 Virginia Tech students and faculty members in two separate attacks, killing himself in the end (Virginia Tech Review Panel, 2007). After Cho killed two students in a residential hall, he killed 30 individuals and wounded 17 others in classrooms at Norris Hall. During the two attacks, students, faculty, and staff witnessed police and emergency medical personnel responding to the event without any knowledge of the ongoing violence. Witnesses observed 10 students escape by jumping out of the windows of a second-story classroom. Many individuals on campus saw medical personnel treating those who had been injured, and live televised reports began soon after the second attack started. Thus, many individuals were directly and indirectly exposed to the shootings and had the potential for developing trauma-related psychopathology, such as Posttraumatic Stress Disorder (PTSD) and Complicated Grief (CG), and were also at elevated risk for developing other mental disorders, including Major Depressive Disorder (MDD).

The majority of the population (69–90%) reports experiencing at least one of a variety of traumatic events (Breslau & Kessler, 2001; Breslau et al., 1998; Norris, 1992). Trauma is associated with symptoms of depressed mood, anxiety, irritability, insomnia, avoidance of trauma reminders, and difficulties functioning in daily activities. Generally, research on the psychological effects of trauma focuses on PTSD, which is the disorder commonly associated with trauma. However, other disorders (e.g., MDD) can be caused by traumatic experiences, whether individually or comorbid with PTSD.

In the last several years, increasing attention has been given to CG (also known as Prolonged Grief Disorder) as a proposed addition to *DSM–5* to help describe psychological reactions to bereavement, including bereavement caused by a trauma. The proposed diagnostic criteria for CG focus on the predominant theme of longing or yearning for the deceased loved one, under circumstances of traumatic and non-traumatic bereavement. Proposed symptoms include difficulty accepting the loss, anger regarding the loss, a feeling that life is empty or meaningless, feelings of bitterness, preoccupations of the person who died, and experiencing auditory or visual hallucinations of the deceased (Prigerson et al., 1995a). It has been proposed that the diagnosis not be made until symptoms have been present continuously for at least 6 months, because grief symptoms that last for at least 6 months are more likely to indicate clinical significance (Latham & Prigerson, 2004; Prigerson, Vanderwerker, & Maciejewski, 2008;

Prigerson et al., 1995b). Although much of the research has focused on CG as a result of the death of a loved one, there have been studies that suggest that non-bereavement losses, such as a job loss (Archer & Rhodes, 1993), a diagnosis of terminal illness (Harvey, 2000), having a child severely injured (Zinner, Ball, Stutts, & Mikulka, 1991), and losing property due to natural disaster (Shear et al., 2011a), are also associated with symptoms of CG. Thus, there is reason to expect that mass shootings could also lead to CG, especially because there is suspicion that traumatic deaths and injuries are even more likely to cause CG than are losses caused by natural causes (Prigerson et al., 2007). The proposed study will examine diagnoses of CG among student survivors of the April 16th shootings, especially those who reported traumatic bereavement. It is acknowledged that this approach will therefore entail examining a subtype of CG (i.e., CG subsequent to trauma-related bereavement), because bereavement by natural causes will not be explored. Although this is a limitation in that the results are not generalizable to CG caused by natural bereavement, this study will provide information regarding trauma-related CG, which has not received much attention in the literature to date.

1.1 - Defining CG

Diagnostic criteria for CG. There is not yet a clear consensus regarding the symptoms and criteria for CG. In an early project to develop explicit criteria for CG, Kim and Jacobs (1991) proposed that separation distress—as evidenced by crying, yearning for the deceased, and having preoccupations of the deceased—would be the hallmark of CG. In an effort to provide additional clarification, Horowitz et al. (1997) assessed symptoms of CG using two grief questionnaires: the Texas Revised Inventory of Grief (Zisook, DeVaul, & Click, 1982) and the Reaction to Loss Inventory (Horowitz & Field, n.d.). Because there was no gold standard assessment for CG at the time (indeed, this is still the case), Horowitz et al. used a median split to define caseness (i.e., those in the upper 50% of scores were assumed to be cases of CG, whereas the others were assigned to be non-cases). Criteria were then compared to determine what response patterns would have acceptable psychometric properties based on this estimate of a gold standard. Horowitz et al. (p. 909) proposed criteria for CG, which notably included yearning for the deceased and avoiding reminders of the bereavement (see Table 1 for full criteria proposed by Horowitz et al.).

Prigerson et al. (1999) continued to refine the symptoms and criteria of CG and proposed a full set of criteria, including suggestions for number of symptoms required to meet criteria for a diagnosis (see Table 2 for full proposed criteria). Similar to Horowitz et al. (1997), Prigerson et al. derived the criteria by creating an estimated gold standard of CG. Unlike Horowitz et al., Prigerson et al. used the upper quintile of CG symptom severity as the cutoff for CG caseness. This approach was utilized because there was no available gold standard assessment for CG; the authors suggested that choosing a higher cutoff score for determining CG caseness would derive a purer CG symptom profile. Differing diagnostic criteria were then tested against the estimated gold standard, with the goal of optimizing sensitivity and specificity. The criteria proposed were found to have very good levels of classification accuracy (sensitivity = .93; specificity = .93).

To meet criteria for CG, Prigerson et al. (1999) suggested that an individual would need to endorse a required criterion of yearning by reporting at least 3 of 4 symptoms in that category, in addition to endorsing at least 4 of 8 symptoms related to mood disturbance and maladaptive cognitions. Prigerson et al. did not retain a behavioral symptom related to avoidance, contrary to the proposed criteria of Horowitz et al. (1997). Furthermore, Prigerson et al. suggested that 14 months was too long for a symptom duration requirement, although they acknowledged that their data did not enable them to test an alternative to this.

Boelen and van den Bout (2007) reported that the criteria proposed by Prigerson et al. (1999) were effective in measuring CG in four samples of bereaved individuals: those who experienced a violent loss 6–12 months ago, those who experience a non-violent loss 6–12 months ago, those who experience a violent loss over 1 year ago, and those who had a non-violent loss over 1 year ago. Using the Dutch version of the Inventory of Complicated Grief–Revised (Boelen, van den Bout, de Keijser, & Joijtink, 2003), Boelen and van den Bout found that good levels of specificity (.93–.99) and sensitivity (.73–.82) were achieved across the samples when case/non-case decisions were based on whether individuals reported at least 3 of 4 A-2 criteria at least “frequently,” 4 of 8 B criteria at least “frequently,” and the D criterion (impairment) at least “sometimes.”

Prigerson et al. (2008) outlined revised criteria for the CG diagnosis while asserting that the predominant feature of CG is a state of chronic mourning characterized by yearning and longing for the deceased, as indicated by symptoms such as excessive loneliness and thinking about the deceased so much that it interferes with activities. They suggested that yearning for the

deceased is a mandatory symptom that must occur daily or to a distressing degree over the past month. In addition, the authors proposed that CG be diagnosed if, in addition to yearning, individuals reported at least 4 of the following 8 symptoms: *trouble accepting the death, inability to trust others since the death, bitterness, difficulty moving on with life* (e.g., forming new relationships), *emotional numbness, feeling that life is empty/meaningless, feeling that the future has no meaning or prospect for fulfillment,* and *feeling agitated* (Prigerson et al., 2008, Table 1). The criteria put forth by Prigerson et al. also include requirements that (a) symptoms cause significant impairment or distress and (b) yearning and at least one other symptom must persist for at least 6 months.

More recently, another diagnostic scheme was proposed (Prigerson et al., 2009) with important differences in suggested symptoms and criteria for the minimum number of symptoms needed to qualify for the diagnosis. Prigerson et al. (2009) suggest that individuals must endorse at least 5 of 9 symptoms; although 6 of the symptoms are similar to those suggested by Prigerson et al. (2008), differences include (a) the addition of *confusion about one's role or diminished sense of self, feeling stunned/shocked,* and *avoiding reminders of the loss*; and (b) excluding the symptoms of *feeling that the future has no meaning or prospect for fulfillment* and *feeling agitated*.

Finally, a recent factor analytic study (Simon et al., 2011) included a factor analysis among positive cases of CG, in order to explore possible symptom clusters in the Inventory of Traumatic Grief (ICG; Prigerson et al., 1995a). This factor analysis among CG cases revealed 6 factors: (a) yearning for and preoccupation with the deceased, (b) anger and bitterness, (c) shock and disbelief, (d) estrangement from others, (e) hallucinations of the deceased, and (f) behavioral changes (e.g., avoidance). Simon et al. (2011) suggest that their results can inform the criteria for CG by highlighting which symptoms can be grouped into coherent diagnostic criteria (see Table 3). Based on the results of their study, Simon et al. reported that high levels of sensitivity (.94) and specificity (.99) were obtained when criteria were based on requiring at least one symptom from the yearning cluster in addition to the endorsement of at least two other symptom clusters.

Because there has not yet been a consensus regarding the criteria for CG, this study will involve comparing different criteria proposed by previous studies. This comparison could be particularly useful in clarifying the nature of CG among traumatically bereaved individuals, because most of the work on CG has involved bereavement of natural causes. Although some

studies (e.g., Boelen & van den Bout, 2007) have included mixed samples in which a minority of participants was bereaved under violent circumstances, these studies have not focused on searching for how traumatic bereavement could differ from non-traumatic bereavement, and they have not had sufficient sample sizes to run factor analysis on the subset of participants who experienced traumatic bereavement. In the study by Boelen and van den Bout (2007), 15% (N = 153) were violently bereaved; the authors reported that optimal diagnostic criteria did not differ for these individuals compared to those with non-traumatic bereavement. Although Boelen and van den Bout did explore CG among traumatically bereaved individuals, the proposed study is unique in that it is the first reported project to explore CG among survivors of a mass shooting, with a range of exposure levels to the traumatic event. Thus, this study will provide an important contribution to the literature on CG by exploring diagnostic criteria among traumatically bereaved individuals.

Distinguishing CG from other disorders. In discussing an experimental diagnosis proposed for adoption in *DSM–5*, such as CG, it is important to distinguish CG from "normal grief" or "non-pathological grief" (hereafter referred to as "NG"). There is evidence that CG can be distinguished from NG based on impairment and distress, in that CG is predictive of functional impairment and clinically significant distress, whereas NG is not associated with either (Prigerson et al., 1995a). However, *DSM* (APA, 2000) criteria for a mental disorder also indicate that the symptoms should be distinct from normal behaviors. There is a concern that NG and CG might not be distinct; indeed, Hogan, Worden, and Schmidt (2003) reported that CG was not distinct from NG. However, other studies (Boelen & van den Bout, 2008; Dillen, Fontaine, & Verhofstadt-Denève, 2008) have provided evidence that CG and NG can be distinguished as related, yet distinct, constructs. Although this issue is not fully settled, the discrepant findings reported by Hogan et al. can likely be attributed to differences in their sample and their measures. Therefore, as suggested by Hogan et al., additional research will need to examine the distinction between CG and NG in diverse samples of bereaved individuals. Such studies would be easier to interpret if they were conducted utilizing commonly used measures of CG and NG. At this time, the emerging consensus in the literature is that the impairment caused by CG symptoms and the preliminary support that suggests that CG is distinct from NG justifies the conceptualization of CG as a pathological response.

Because the features of CG include significant sadness brought about by experiencing a loss, this disorder has some commonalities with both MDD and PTSD. Like MDD, CG includes symptoms related to sadness and the loss of interest in activities (Shear, Frank, Houck, & Reynolds, 2005). Similar to some cases of PTSD, CG can be caused by a traumatic bereavement and often includes a presentation of intrusive thoughts (Shear et al., 2005), although not all cases of CG are caused by unexpected or violent bereavement.

Although the symptoms of CG do overlap with PTSD and MDD, the proposed phenomenology of CG indicates that CG is distinct from PTSD and MDD (see Table 4). The primary distinction between CG and PTSD is that the primary feature of CG is longing for a loved one, whereas PTSD is characterized by fear that a particular trauma will recur or dissociative symptoms in response to trauma-related stimuli. Essentially, CG is a bereavement-specific mood disorder, whereas PTSD is has recently been conceptualized as a heterogeneous category of (a) cases of stress-related fear circuitry disorder (Friedman, Resick, & Keane, 2007), in which the predominant clinical presentation involves heightened fear and anxiety, and (b) other cases of a dissociative subtype, which would include symptoms such as derealization and depersonalization (Friedman et al., 2011a). Although either PTSD or CG might occur after a traumatic event involving the death of a loved one, PTSD is associated with experiencing, witnessing, or being "confronted" by a perceived physical threat to the self or to another individual (APA, 2000, p. 467), whereas CG is triggered by the bereavement, regardless of whether the individual was present when the loved one died. Furthermore, it has been proposed that having a loved one die under expected conditions (e.g., protracted terminal illness) be dropped from the PTSD Criterion A in *DSM–5* (Friedman, Resick, Bryant, & Brewin, 2011b). If this change does occur, many individuals who experience bereavement will not meet Criterion A for PTSD in *DSM–5*—even though they might have arguably met PTSD criteria under the *DSM–IV* Criterion A—although they would meet the triggering event criterion for CG. Finally, although PTSD and CG entail intrusive thoughts (as noted above), there is evidence (Boelen & Huntjens, 2008) that the content of intrusive thoughts related to CG is different from intrusive thoughts related to anxiety. Specifically, positive intrusive memories of the deceased were associated with CG, whereas intrusive images of the moments surrounding the death were associated with anxiety (Boelen & Huntjens, 2008, p. 223).

It also seems that the course of CG over time differs from the typical trajectories of PTSD symptoms and bereavement-related MDD. Specifically, symptoms of bereavement-related MDD often wane over time, whereas symptoms of CG appear to persist (Prigerson et al., 1995b). A one-year follow-up study of survivors of a mass shooting (North, Smith, & Spitznagel, 1997) reported that 54% of the 46 participants who reported having PTSD had recovered by the one-year follow-up. Similarly, Hepp et al. (2008) reported that 6% of accident survivors met symptom criteria for PTSD 2 weeks after an accident (although the authors noted that this did not meet the *DSM* duration criterion of more than one month), whereas only 2% of the sample met criteria 1 year later and 4% met criteria after 3 years. These findings suggest that rates of PTSD in a given sample are generally reduced by approximately 50% the first year after the traumatic event. The long-term course and typical trajectories of CG is still unclear; however, Prigerson et al. (1995b) suggest that those who meet criteria for CG at 6 months will frequently demonstrate persistent long-term symptoms.

CG differs from MDD in that the individual must have experienced the loss of a loved one in order to meet criteria for CG. In addition, the symptoms of sadness, preoccupations or ruminations, and guilt associated with CG are related to missing the deceased loved one, whereas the symptoms of MDD are associated with multiple life domains (Shear et al., 2005). For example, MDD could include ruminations about several perceived failures and mistakes as a parent, a friend, and an employee, whereas CG commonly involves being preoccupied with positive memories of the deceased (Shear et al., 2005).

Finally, there is evidence that CG—especially chronic CG—does not respond well to tricyclic antidepressants and seems to respond poorly to therapies designed to treat MDD (Shear et al., 2005), whereas psychotherapy designed specifically for CG appears to be superior to non-specific interpersonal therapy (Shear et al., 2005). This again suggests that CG is distinct from MDD, even when depression is triggered by bereavement.

In addition to NG, PTSD, and MDD, CG has similarities with and distinctions from bereavement as currently described in *DSM–IV*. CG differs from the current *DSM–IV* Bereavement V-code by proposing a specific set of diagnostic criteria, including symptom criteria and necessary duration of symptoms and impairment. It is possible that CG could replace

Bereavement in *DSM–5* entirely, although retaining the V-code might have utility in cases where individuals seek professional help but do not meet strict criteria for CG, MDD, or any other Axis I disorder.

Factor analytic studies have shown that CG is distinct from other disorders. Boelen, van den Bout, and Keijser (2003) demonstrated in a sample of bereaved adults that traumatic grief was distinguishable from depression and anxiety. Principal axis factoring of items from the ICG, SCL–90 anxiety subscale, and SCL–90 depression subscale revealed a three-factor solution (Prigerson & Jacobs, 2001). However, it should be noted that the authors used an orthogonal (specifically, varimax) rotation, which constrains the solution from having correlated factors. Although this approach might simplify the interpretation, it is perhaps inappropriate given the typically high associations between symptoms of anxiety and depression.

Criticisms of CG. Bonanno (2006) has noted that research has yet to demonstrate solid evidence that CG has incremental validity (i.e., that it explains unique variance when PTSD and depression are also assessed). There is preliminary evidence that CG might possess incremental validity (Bonanno et al., 2007), but additional research is needed to replicate these results. Bonanno (2006) has also pointed out that there is evidence that a significant subset of individuals who meet criteria for CG exhibited significant depressive symptoms before experiencing the loss (Bonanno et al., 2002). Therefore, if researchers do not attempt to assess pre-loss symptoms of depressed mood and other CG-related phenomena, estimates of CG post-loss might be inflated. In additional analyses of the data from Bonanno et al. (2002), individuals who were depressed prior to experiencing the death of a spouse evidenced differences from those who were not depressed until after the death of a spouse (Bonanno, Wortman, & Nesse; 2004). This suggests that the etiological importance of experiencing bereavement is not particularly well understood at this time.

1.2 - Dose–Response Relationships in Trauma-Related Psychopathology

An assumption of the PTSD diagnosis is that there is a dose–response relationship between traumatic events and symptoms of psychopathology. This concept (sometimes called the "adversity–stress model") has found support, but several limitations in the model's explanatory power for the etiology of PTSD have been reported (Rosen & Lilienfeld, 2008). For instance, Criterion A events (e.g., being raped, tortured, or in a serious motor vehicle accident) are more likely to lead to PTS symptoms than are other stressors (e.g., getting a divorce or writing a

dissertation; see Rosen & Lilienfeld, 2008). However, a simple dose–response model would predict that increased exposure to trauma, such as experiencing additional traumatic events, multiple episodes of an ongoing trauma, or being in close physical proximity to a disaster, should increase the risk of PTSD and the severity of symptoms, which is not consistently supported by empirical evidence (Bowman, 1999). For instance, the number of episodes of torture was not predictive of PTS, whereas perceived distress ratings were related to the severity of PTS (Başoğlu & Paker, 1995). On the other hand, Galea et al. (2002) reported that participants who lived near the World Trade Center (i.e., south of Canal Street) during the September 11th terrorist attacks had a 20% prevalence rate of probable PTSD, whereas the prevalence rate for those who lived north of Canal Street was only 6.8%, suggesting that individuals who were closer to the direct effects of the trauma (and who therefore likely received a higher dose of traumatic exposure) did indeed demonstrate more PTS. Finally, the dose–response model cannot explain why only a minority of individuals exposed to a trauma develop PTSD (Breslau et al., 1998). These limitations indicate that the dose–response model would be a more helpful theory if modifications are made.

Current research on the utility of the dose–response model suggests that attempting to catalog the objective trauma potential of different events is problematic at best (McNally, 2003). When *DSM–IV* was published, the Criterion A for PTSD was loosened such that many trauma researchers and clinicians have argued that secondhand trauma exposure can qualify an individual for a PTSD diagnosis. However, there appears to be an obvious qualitative distinction among certain traumas—for example, between being raped at gunpoint and hearing someone relate this experience after the fact (McNally, 2003). Therefore, ostensibly objective schemes to measure trauma exposure by asking individuals to report their histories of various traumatic experiences to create a summed trauma exposure scale run the risk of "combining apples and oranges."

Another related difficulty involves the issue of objective trauma exposure and perceived trauma exposure. Although some studies have focused exclusively on objective trauma exposure (e.g., Galea et al., 2002) some researchers (e.g., Burns, 2009; Piotrkowski & Brannen, 2002; Vernberg, La Greca, Silverman, & Prinstein, 1996) have utilized a composite approach that

combines an objective definition of exposure (e.g., proximity to a disaster or incidence of injury) with subjective assessments of perceived trauma exposure (e.g., perceived life threat at the time of the trauma or threat appraisal of future traumatic events).

Still other researchers (e.g., Silver, Holman, McIntosh, Poulin, & Gil-Rivas, 2002) have argued that indirect exposure, such as watching news coverage of a disaster on television or hearing about someone else's traumatic experiences, can count as traumatic exposure. Silver et al. (2002) measured exposure to the September 11 World Trade Center attacks by asking participants to report firsthand exposure as well as the amount of news coverage of the event they watched (both live and in the days following the attacks). In several of their analyses, they dichotomized the exposure variable (i.e., compared a "no exposure group" with an "exposure group"), and those who were only exposed through watching live television broadcasts were in the exposure group. This dichotomous trauma exposure variable was a significant predictor of PTS when adjusting for previous mental and physical health conditions; however, it was no longer a significant predictor after coping behaviors were added to the model (Silver et al., 2002, Table 4). In their conclusions, Silver et al. suggested that the traditional view of the importance of direct exposure to a trauma in the development of PTS is not supported by their results, because most of the participants in the exposure group had only viewed the attacks live on television, suggesting that the significance of the dichotomized exposure variable is primarily due to the effects of indirect trauma exposure.

However, this inclusion of indirect exposure—particularly via television—with traditional forms of exposure that require the individual was physically present, is controversial. Not only does this approach risk combining qualitatively different types of events (as stated above), but it also could lead to widening the PTSD diagnosis to include additional heterogeneity. For example, it seems unlikely that individuals who experienced the September 11 World Trade Center attacks firsthand experience similar PTS as those who watched the attacks on television hundreds of miles away from the actual disaster (McNally, 2009). Furthermore, there is evidence that merely watching traumatic events on television is not likely to cause symptoms of PTS: Weidmann and Papsdorf (2010) reported no significant differences in PTS between employees who work in a television newsroom and those who work in a radio newsroom, although the television employees spent considerable time exposed to footage of violence or trauma. Similarly, Ahern et al. (2002) found that rates of PTSD and depression were

not related to the amount of television coverage of the September 11 terrorist attacks viewed among individuals who did not experience direct exposure. They did report that, among participants who were directly exposed to the attacks, those who frequently watched images of people falling from the World Trade Center towers were more likely to suffer from PTSD or depression compared to those who were directly exposed to the attacks but did not repeatedly view this image. These findings suggest that media exposure to traumatic events is unlikely to serve as a trauma itself, although it might act as a trauma reminder to those who were personally impacted by the event or a similar one, thereby increasing the probability that these individuals will experience elevated levels of PTS. Alternatively, it could be that some individuals who are experiencing significant PTS seek out media coverage of the trauma in an attempt to cope with their emotional reactions.

It is quite likely that some of the difficulties in finding consistent support for the dose–response model arise from competing definitions, operationalizations, and measures of trauma exposure. If the *DSM–5* criteria for PTSD are changed to alter the exposure criterion (see Friedman et al., 2011b), then most cases of proposed indirect or media exposure will not qualify for a PTSD diagnosis (although the proposed revision would include "learning that the [traumatic event] occurred to a close relative or close friend").

Finally, some of the controversy regarding the dose–response model appears to stem from disagreements about the importance of the variance accounted for by trauma exposure (i.e., the predictive power of trauma severity or duration). Although meta-analyses have demonstrated that other factors—such as level of social support (Brewin et al., 2002) and peritraumatic dissociation (Ozer, Best, Lipsey, & Weiss, 2003)—are more predictive of PTS than is trauma severity, this does not necessarily mean that trauma severity is irrelevant. From a conceptual standpoint, it is difficult to imagine a stress-related disorder (as suggested by Friedman et al., 2011a) without a stressor—imagine diagnosing someone with a Specific Phobia without identifying a feared object or situation, but just claiming that the individual is very fearful of something specific yet unidentified. Consistent with this line of thought, at the end of their meta-analysis, Brewin et al. (2002) concluded that their results are

> consistent with a model in which the impact of pretrauma factors on later PTSD is mediated by responses to the trauma or, alternatively, with a model in which pretrauma factors interact with trauma severity or trauma responses to increase the risk of PTSD (p. 756).

It therefore appears reasonable to modify the dose–response model by including other variables as predictors of PTS while still recognizing the role of traumatic events in the etiology of PTSD.

Therefore, it is plausible that the dose–response model does provide a useful framework for studying the effects of trauma exposure on the development of PTS. Recent research using this theory has been more productive by (1) including other important variables, such as social support (Brewin et al., 2002), premorbid psychopathology (Silver et al., 2002), and peritraumatic dissociation (Ozer et al., 2003), in predictive models of PTS, and (2) conceptualizing and operationally defining *trauma exposure* as a multidimensional construct, including objective (e.g., proximity to disaster, trauma-related injury, and number of traumatic episodes) and subjective (e.g., peritraumatic distress and perceived life threat) components (La Greca, Silverman, Vernberg, & Prinstein, 1996). These two adjustments to the dose–response model have different direct effects on the outcomes of the model. Adding alternative predictors to the model helps explain more variance in PTS (i.e., the outcome variable), making the model have higher overall predictive value, whereas adjusting the definition and measurement of exposure can impact the amount of variance it explains, which could increase the apparent importance of this variable in predicting PTS. This probabilistic framework would still operate under a dose–response model conceptualization, but with the recognition that other factors are also important—including factors that are even more predictive of symptom severity (e.g., perceived life threat). Although this would be a departure from the original theory that emphasized the importance of the traumatic event, it would still recognize the empirically-supported idea that event characteristics can have an impact on symptom severity in certain situations. Several researchers (e.g., Agronick, Stueve, Vargo, & O'Donnell, 2007; La Greca, Silverman, & Wasserstein, 1998) have taken this or similar approaches and emphasized that trauma exposure is an important part—but only a part—of the predictive model for PTS symptoms. For example, La Greca et al. (1998) found that predisaster anxiety and traumatic exposure to Hurricane Andrew were both predictive of PTS symptoms, suggesting that premorbid psychopathology can predispose individuals to demonstrate PTS after experiencing trauma exposure. In another study, Gil and Caspi (2006) found that proximity to a bus bombing was not significantly correlated with perceived threat; however, each of these variables significantly predicted PTS 6 months after the trauma, highlighting the importance of both objective exposure and subjective exposure variables.

The research on the dose–response model of PTSD and the proposed changes in the criteria for *DSM–5* PTSD are relevant to the diagnosis of CG. Specifically, traumatic bereavement—which can be a qualifying event for PTSD in both *DSM–IV* and *DSM–5*—is the triggering event for CG; it is currently unknown whether direct (i.e., witnessing in person) and indirect (e.g., hearing about the death after the fact) types of exposure are differentially predictive of PTSD and CG. It is possible that, under the circumstances of traumatic bereavement, direct exposure to the trauma is more closely associated with PTSD, whereas indirect exposure could be more closely related to later development of CG. Furthermore, it is possible that the dose–response model could apply directly to CG. One could postulate whether certain circumstances of bereavement are more potent than others (e.g., homicide vs. dying of cancer after being in treatment for 8 months). It is also possible that those who experience the death of a close friend or family member are more likely to have CG or to report increased severity of bereavement symptoms than are those who experience the death of an acquaintance. The research on dose–response relationships with trauma and PTSD suggests that attempts to compare objective levels of loss or types of bereavement might be misguided; however, a model that includes objective and perceived exposure might provide a more complete account of the role of trauma exposure in the development of PTSD.

For the purposes of this project, a variation of the dose–response model consistent with the proposed *DSM–5* Criterion A is the guiding framework. Specifically, it is proposed that different qualitative types of exposure—objective exposure to the trauma, traumatic bereavement (conceptualized as a subtype of objective exposure), and subjective perceptions of danger—will have differing relationships to CG and PTS. Indirect exposure (e.g., watching media coverage of the trauma), which is also a type of objective exposure, is not included in the proposed *DSM–5* Criterion A and is not expected to have significant relationships with CG or PTSD, except in individuals who also report direct exposure and/or traumatic bereavement.

Hughes et al. (2011) found that trauma exposure was related to symptoms of PTS; however, an examination of which types of exposure predicted psychopathology suggests that PTSD might not be the only psychological disorder of interest among survivors of the Virginia Tech shootings. Hughes et al. reported that the most predictive types of exposure were *inability to confirm the safety of friends, death of a (not close) friend,* and *death of a close friend*. Therefore, two of the three exposures most predictive of PTS were violent bereavement, which

could also be predictive of CG. Interestingly, bereaved survivors of the shootings meet the triggering event criterion for both PTSD and CG. The proposed study will use the dose–response model to examine the possibility that different elements of trauma exposure are differentially related to different diagnoses; in this case, specifically that direct exposure (e.g., being on campus, being in one of the buildings during the shootings, etc.) to the shootings will be related to PTS, whereas the death of a friend will be related to symptoms of CG. Part of this analysis will involve a closer look at the psychopathological outcomes of those who were bereaved by the shootings, to determine what exposure variables distinguish between the development of PTS and CG.

1.3 - Mass Shootings

Previous research (Johnson, North, & Smith, 2002; North, Smith, & Spitznagel, 1994; North et al., 1997; Trappler & Friedman, 1996) has demonstrated the negative psychological consequences of mass shootings in a variety of settings. Although research often focuses on PTSD, Major Depressive Disorder (MDD), and Substance Abuse/Dependence Disorders, findings often suggest that there are psychopathological reactions to trauma that do not always seem to be described adequately by these diagnostic categories. The trauma-related diagnoses currently researched most commonly do not seem to capture the entire range of trauma reactions. For instance, Johnson et al. (2002) reported that 5% of participants met criteria for PTSD 6–8 weeks after a shooting at a courthouse; however, they also noted that 96% of participants endorsed at least one symptoms of PTSD, and 75% indicated that the incident was "very upsetting," suggesting that some individuals experienced psychological distress that did not meet criteria for PTSD but might still warrant intervention.

In other instances, research has focused on current diagnostic categories but has highlighted the sometimes unanticipated course of symptom expression over time. North et al. (1994) reported that 26.5% (N = 36) of participants developed PTSD 6–8 weeks after surviving a shooting in a crowded cafeteria. North, Smith, and Spitznagel (1997) reported that this percentage had dropped to 16.9% (N = 21; note that there was an additional case of PTSD at one-year follow-up that was unrelated to the shooting) one year after the initial assessments. Of the 21 shooting-related cases of current PTSD at one-year follow-up, 6 were new cases that did not exist at the initial assessment. Furthermore, 6 individuals reported developing new cases of

PTSD that had remitted by the follow-up assessment. Of the 46 individuals who reported PTSD at least once and participated in both assessments, 12 (26%) demonstrated delayed-onset PTSD.

A few studies (Brymer, 2007; Hawkins, McIntosh, Silver, & Holman, 2004; Scrimin et al., 2006) have specifically examined the impact of school shootings. However, no previous studies have examined the CG diagnosis among survivors of a school shooting.

1.4 - Hypotheses

In the first step of analyses, it was hypothesized that a stable factor structure for CG would be discovered with appropriate levels of internal consistency. This will involve selecting the items that best capture the construct of CG, as determined by exploratory and confirmatory factor analytic procedures (see Method section). It is hypothesized that items from the ICG will demonstrate a single factor among the entire sample, although additional items from the survey might also be appropriate for inclusion in a CG symptom scale. It was predicted that symptoms pathognomonic of CG would be distinguishable from those of PTS and general depression and anxiety problems when examined via factor analysis, as previously reported in other studies (e.g., Boelen et al., 2003). It was also hypothesized that exploratory and confirmatory factor analyses among participants with high CG scores would reveal symptom clusters, consistent with previous research (Simon et al., 2011).

In the second step, the scale tested in the first step was used in a structural equation model to determine which variables at Wave 1 are significant predictors of CG at Wave 2. It was hypothesized that traumatic bereavement would be more related to CG than to PTS, whereas direct exposure to the shootings—as measured by being on campus, hearing gunshots, proximity to the shootings, seeing someone injured or killed, and being injured—would be more closely related to PTS than to CG. It was also hypothesized that being unable to confirm someone's safety would be a significant predictor of CG among traumatically bereaved students. Finally, among individuals who were traumatically bereaved by the shootings, it is hypothesized that interaction effects of indirect trauma exposure (e.g., watching media coverage of the shootings) and direct exposure will increase the odds of developing symptoms of PTS. This study is the first to test this hypothesis directly among a sample of mass shooting survivors.

Chapter 2 - Method

2.1 Participants

The method of data collection for Wave 1 has been previously detailed elsewhere (Hughes et al., 2011). For the purposes of this study, the relevant highlights of this sample are presented, along with the additional details of Wave 2 (which have not been reported previously).

Participants of the current study were 4639 student survivors of the April 16th shootings at Virginia Tech. Data in Wave 1 were gathered from an online e-mail survey delivered July 10, 2007, by the Virginia Tech Center for Survey Research. Participants provided consent before completing the survey. When the survey was closed, 20% of the target sample of 23,214 currently enrolled students had completed the survey, 0.1% refused to participate, and 79.9% did not respond.

Wave 2 data were gathered from 861 students about 1 year after the shootings occurred. Because of the longitudinal nature of the current study, only data from these 861 students who responded at Wave 2 were used for primary analyses.

2.2 Measures

Trauma exposure. Questions regarding where students were and what happened to them during each phase of the crisis were included to measure different types of exposure to the shootings. Hughes et al. (2011) categorized these variables into 5 groupings: awareness of the events as they occurred (e.g., hearing campus announcements or watching TV reports about the events as they unfolded), physical proximity to the events (e.g., being in a building where a shooting took place or hearing shots), direct exposure (e.g., witnessing students fleeing or personally being injured), indirect exposure (e.g., being outside on campus during lockdown or trying unsuccessfully to confirm the safety of a close friend during lockdown), and traumatic loss (i.e., a significant other's, friend's, or acquaintance's death, injury, or escape from the shootings). Awareness, proximity, and direct exposure were assessed for the first shooting and the second shooting separately. A total of 18 compound stressor variables were created by logically combining individual responses. For example, the compound variable *death of someone close* was created by combining items that inquired whether individuals had lost a boyfriend,

girlfriend, spouse, or other close friend. These compound variables were used to measure the 5 types of trauma exposure described above; 6 of the 18 compound variables were previously found to be significant predictors of posttraumatic stress (Hughes et al., 2011).

Based on dose–response theory, proposals for *DSM–5* (Friedman et al., 2011b), and recent research (e.g., Ahern et al., 2002), exposure variables are classified as direct objective exposure (e.g., hearing shots), subjective exposure (e.g., perceived threat to self), and indirect objective exposure (e.g., watching television reports of the shootings). It should be noted that traumatic bereavement (i.e., what Hughes et al., 2002, termed *traumatic loss*) is classified as direct objective exposure in dose–response theory.

In addition to the items of objective trauma exposure, there are two items that measure participants' subjective perceptions of trauma severity: "How afraid were you that you might be killed at your worst moment on April 16?" and "How afraid were you that someone you cared about would be seriously hurt or killed?" Participants were asked to respond on a 0–10 scale, with higher numbers reflecting higher levels of fear. These items will also be included in the model to ascertain the relationship of subjective trauma exposure severity with PTS and symptoms of CG.

CG symptoms. Wave 2 includes 28 items that cover the proposed criteria of CG. The items were based on the Inventory of Complicated Grief (Prigerson et al., 1995a) as well as other studies (Prigerson et al., n.d.; Shear et al., 2005) that involved assessing CG. Items are on a 5-point Likert-type scale (1 = *never*; 5 = *always*). It should be noted that the questionnaire contained 30 items purported to measure CG, but two items were excluded from analyses in order to comply with prohibitions from the Virginia Tech IRB. Prigerson et al. (1995a, Table 3) reported excellent internal consistency ($\alpha = .94$) for the 19-item Inventory of Complicated Grief; preliminary analysis of the 28 items of CG among the 747 participants with complete data revealed a similar level of internal consistency in this sample ($\alpha = .95$).

PTS symptoms. Symptoms related to PTSD were assessed using a modified version of the Trauma Screening Questionnaire (TSQ; Brewin et al., 2002), which has been previously validated (Brewin et al., 2002; Walters, Bisson, & Shepherd, 2007). In the surveys in this study, the TSQ was modified to refer specifically to the April 16th shootings at Virginia Tech. A

similar version of the TSQ was used in a prior study of the survivors of Hurricane Katrina and was found to have excellent diagnostic concordance with a structured clinical interview (Galea et al., 2007; Kessler, Galea, Jones, & Parker, 2006).

The PTSD screening measure used in this study contained 10 items: three intrusion items, two avoidance items, two altered cognition/mood items, and three hyperarousal items. Primary analyses were conducted using a continuous variable of total PTS, created by summing responses across all subscales. For categorical analyses, respondents were classified as *high PTS* if they endorsed significant symptoms (at least one re-experiencing symptom, at least one avoidance symptom, at least one altered cognition and mood symptom, and at least one hyperarousal symptom) occurring at least 4–5 days/week. Participants who do not meet these criteria were classified as non-cases. Preliminary analyses conducted using 823 complete cases suggest that the total PTS scale has excellent internal consistency ($\alpha = .91$) in this sample.

Anxiety–mood symptoms. The K6 scale is a 6-item screening instrument of nonspecific psychological distress (Kessler et al., 2003) that focuses on symptoms of *DSM–IV* anxiety and mood disorders. Each item asks participants to rate how frequently they experienced a given symptom (e.g., feeling "nervous") on a 5-point Likert-type scale, with 0 indicating "none of the time," and 4 indicating "all of the time." Total scores are obtained by summing the items; scores of 13–24 indicate probable serious mental illness, scores of 8–12 suggest probable mild/moderate mental illness, and scores of 0–7 are considered noncases. Preliminary analyses utilizing 840 cases with complete data indicated that the K6 has good internal consistency ($\alpha = .87$) in this sample.

2.3 Analytic Plan

Missing data. Among the 861 participants at Wave 2, items of CG had a missingness rate of 3.4–5.7% (i.e., 29–49 missing cases), PTS items were missing in 0.7–1.7% of cases (i.e., 6–15 cases), and K6 items were missing in 0.5–0.9% (i.e., 4–8 cases). To determine how missingness on these variables was related to other variables in the data, correlations were analyzed among CG, PTS, K6, and traumatic exposure (the latter being comprised of items from Wave 1).

For the purposes of the exploratory factor analysis (EFA), missing data among the 861 participants of Wave 2 was examined using EQS 6.1 (Bentler, 2010). Expectation maximization (EM) was utilized to impute values for missing data. For confirmatory factor analyses (CFA) and

structural equation modeling (SEM), full information maximum likelihood (FIML) estimation is available in EQS 6.1 (Bentler, 2010). FIML in EQS is advantageous because it is includes robust procedures for estimating standard errors and χ^2 when normal distribution assumptions are violated (Bentler, 2004). However, FIML can only be used in EQS 6.1 when using the maximum likelihood (ML) estimator on continuous variables. Given the nature of the data (explained below in the relevant portions of the Results section), this approach was not deemed the most appropriate; therefore, listwise deletion was used for CFA and SEM analyses. For some of the regression models, missing values were imputed using the Multiple Imputation procedure of SPSS 20. In order to ensure stable coefficient estimates, 100 datasets with imputed values were created in order to provide pooled coefficient estimates. However, this method does not include procedures for creating pooled model estimates.

EFA. The first part of analyses included a psychometric evaluation of the items in the dataset that were designed to measure CG. EFA was conducted in IBM SPSS 20 Statistics using generalized least squares (GLS) to determine the factor structure of the CG items as a scale as well as to examine the relative contributions of each item. GLS estimation was chosen because the data are ordinal at the item level. (Incidentally, ML estimation provided similar results, although these are not presented here). As needed, items were dropped from the scale, and the scale was re-evaluated. The numbers of factor retained in each analysis was determined by (a) using parallel analysis to provide an estimate of the maximum number of factors that should be retained (Zwick & Velicer, 1986) and (b) plotting the eigenvalues in a scree plot (Gorsuch, 1983). Each parallel analysis was conducted by factor analyzing 1000 datasets of random data and calculating the 95^{th} percentile of the eigenvalues, thereby creating a similar threshold as the $\alpha = .05$ level used commonly in significance testing (Hayton, Allen, & Scarpello, 2004). Tabachnick and Fidell (2007) suggest that items with loadings of at least .32 can be considered to be indicators of a factor, although they point out that a loading of .32 indicates that only 10% of the item's variance overlaps with the factor. In the present study, CG items that have loadings on the CG factor below .40 will be considered poor indicators and will be dropped. This cutoff was chosen because it is somewhat more demanding than the .32 cutoff (which might lead to the inclusion of items that are not good measures of CG) but not so strict as to exclude too many items.

Studies (Guadagnoli & Velicer, 1988; Tabachnick & Fidell, 2007) on the necessary sample size for factor analytic procedures suggests the current sample size (N = 861) should be appropriate for most solutions. Because previous research (Prigerson et al., 1995a; Simon et al., 2011) has reported that CG generally has a single-factor structure, it was anticipated that the sample size would be excellent for factor analyzing the CG items in the data. Depending on the number of participants who score highly on CG, there might not be sufficient power to replicate the findings reported by Simon et al. (2011) that CG has 6 factors among those who meet criteria for probable CG. Tabachnik and Fidell (2007) suggest that, under circumstance in which several items load highly (> .80) on their respective factors, sample sizes of approximately 150 might be sufficient for an exploratory factor analysis. To the extent that these conditions are not met in for the various EFA procedures performed in this study, the results will be presented with a cautionary discussion about the stability of the factor solution.

CFA. To confirm the results from the EFA, CFA was conducted in EQS 6.1 (Bentler, 2010). Because the data are best conceptualized as ordinal at the item level, the preferred method for CFA in EQS would be arbitrary distribution generalized least squares (AGLS). However, AGLS estimation often requires very large sample sizes (e.g., N = 2500); therefore, maximum likelihood estimation with robust standard error estimation (ML ROBUST) and testing will be utilized whenever a CFA model does not converge with a proper solution using AGLS estimation (Brown, 2006). Currently, these methods are the best ways to conduct SEM analyses on ordinal data using EQS 6.1 (Bentler, 2006). Model fit for ML ROBUST-estimated models was assessed by examining the comparative fit index (CFI), the standardized root mean square residual (SRMR), and the root mean square error of approximation (RMSEA). When AGLS was used, model fit was assessed by the RMSEA and the adjusted AGLS fit index (AAFI). Finally, although there is still some debate regarding the role of χ^2 in assessing model fit, recent research suggests that it is still an important fit index (Kline, 2011). Due to deviations from multivariate normality among the data, corrected χ^2 indices were examined to assess model fit (the specific corrected χ^2 used is noted where appropriate). Due to the fairly large sample sizes used in the models in this study (N = 721–825, depending on the analysis), it should be noted that model χ^2 is partly dependent on sample size and might be significant even under circumstances of minor

model misfit. Areas of localized strain were assessed by examining the standardized residuals, which in EQS are the difference between the observed correlations and the model-implied correlations.

Competing models were compared using the Akaike Information Criterion (AIC), with lower values indicating the preferred model. Modification indices (e.g., the EQS Lagrange multiplier) were unavailable for this study, because EQS analyzes the polychoric correlation matrix of ordinal data yet requires a covariance matrix to be used to produce modification indices. It is anticipated that this will not be problematic for the CFA in this study, because any additional parameter estimates would run counter to the theoretical proposal that the three psychopathology constructs are distinct. Individual item loadings for final models will be presented; however, polychoric correlation analysis does not entail estimating measurement error of ordinal indicators as free parameters.

All CFA models were estimated using the original ordinal data. CFA was attempted on the dichotomized version of the CG–21; however, the data were so skewed that no estimation method could converge and provide estimates for model or parameter estimates.

All CFA models were based on the entire sample (N = 825). Because of the small number of participants who met criteria for estimated clinical levels of CG (N = 104) or who experienced the loss of a significant other or close friend (N = 103), neither subsample was used for separate CFA models.

SEM. After completing the above analyses to explore the psychometric properties of the revised CG scale, this scale was used as a criterion in a predictive SEM using exposure variables from Wave 1 as predictors. Because premorbid psychopathology and previous traumas are significant predictors of posttraumatic psychopathology (Brewin et al., 2000; Ozer et al., 2003), premorbid adjustment (measured on a 5-point scale) and traumatic experiences were added to the model as control variables. Model fit was assessed following the same procedures as the CFA (outlined above). In addition, hypothesis testing was conducted using the guidelines proposed by MacCallum, Browne, and Sugawara (1996) in order to facilitate overall model power analysis.

Power analyses. Because the data have already been collected, post hoc power analyses were conducted as appropriate for the various types of analyses. For regression analyses, G*Power 3.1 (Faul, Erdfelder, Buchner, & Lang, 2009) was used to determine the achieved power for detecting significant regression coefficients. For SEM analyses, power analysis was

planned to include calculating the achieved power of specific parameter estimates as well as overall model fit (Hancock, 2006; Kline, 2011). As discussed in Hancock (2006), estimating power for specific parameter estimates would have involved the following process for each parameter estimate of interest: (a) determining whether the full maximum likelihood model has acceptable fit, (b) fixing peripheral parameter estimates to the values provided by the full model, (c) fixing the focal parameter estimate to 0, (d) running the model to obtain the χ^2 statistic associated with the badness of fit related to the focal parameter being fixed to 0, and (e) calculating the power estimate based on the χ^2 value (i.e., the noncentrality estimate for the parameter in question) and the degrees of freedom. Unfortunately, as explained below, this approach was not compatible with the ordinal nature of the data. Power for overall model fit will be estimated using the root mean square error of approximation (RMSEA), degrees of freedom in the model, and sample size.

Chapter 3 - Results

3.1 Missing Data Exploration and Analyses

As noted above, only 861 individuals from Wave 1 participated in Wave 2 data collection. To test for differences between those who did and those who did not respond to the Wave 2 survey, correlations were analyzed to help determine on which variables the two groups likely differed. This procedure indicated that Wave 1 psychopathology symptoms (PTS, grief, and K6), perceived threat to self, perceived threat to others, gender, direct exposure to the shootings, exposure to media coverage, and being upset about media coverage were related to CG (Table 5), PTS (Table 6), and/or K6 at Wave 2 (Table 7). Having a significant other, close friend, or not close friend killed, injured, or escaped unharmed were related to CG at Wave 2 (see Table 5). Because of the number of comparisons and the large sample size, it is likely that trivial effects will be found to be significant; therefore, results were considered significant if the effect accounted for at least 1% of the variance.

To determine whether the respondent groups differed on the variables that accounted for at least 1% of the variance in the Wave 2 symptom scales, *t*-tests and χ^2-tests were conducted. As displayed in Table 8, significant between-group differences were revealed for direct exposure, $t = 2.55$, $p < .05$, media distress, $t = 2.84$, $p < .01$, and other threat (i.e., fear that someone they knew would be seriously hurt or killed), $t = 2.87$, $p < .01$. Results of the χ^2-tests suggested that the groups differed significantly on gender, $\chi^2 = 33.28$, $p < .001$, and having a significant other, close friend, or not-close friend killed, $\chi^2 = 12.45$, $p < .001$. The groups did not differ on having a significant other, close friend, or not-close friend injured, $\chi^2 = 0.41$, $p = .525$, or escaped, $\chi^2 = 0.00$, $p = .958$. Therefore, these results suggested that respondents who participated in Wave 1 but not in Wave 2 (a) were less likely to have lost a significant other, close friend, or not-close friend, (b) reported less exposure, (c) experienced less fear that someone they knew would be seriously hurt or killed, (d) reported less distress at media coverage, and (e) were more likely to be female. This suggests that those who did participate in Wave 2 were those who were more impacted by the shootings.

Among the 861 participants at Wave 2, items of CG had a missingness rate of 3.4–5.7% (i.e., 29–49 missing cases), PTS2 items were missing in 0.7–1.7% of cases (i.e., 6–15 cases), and K6_2 items were missing in 0.5–0.9% (i.e., 4–8 cases). Although most of the variables of interest were correlated with the Wave 2 symptom scales (see Tables 5–7), results from *t*-tests and χ^2-

tests demonstrated that participants who missed at least one item of the CG scale did not differ from other participants on the predictor variables from Wave 1 (Table 9). Participants also did not differ based on missingness on PTS2 (Table 10); however, there was a significant difference on K6_1 between missingness groups on K6_2, such that those who did not complete all of K6_2 had higher K6_1 scores than respondents who did complete K6_2 (Table 11). The point-biserial correlation between missingness status on K6_2 and scores on K6_1, $r_{pb} = .08$, $p < .05$, indicates that K6_2 missingness accounts for 0.58% of the variation in K6_1, which is less than the criterion outlined above for considering significant effects. As a final step to explore the missingness mechanism in the data, the variables were tested using Little's Missing Completely at Random (MCAR) test (Little, 1988), which indicated that the variables in the model were MCAR, $\chi^2 = 77.35$, $df = 65$, $p = .140$. (It should be noted that it is possible that a different mechanism caused missingness, or that an additional, undetected mechanism exists among the data.) Therefore, full information maximum likelihood (FIML) and related techniques are appropriate for imputing missing data. Specific techniques for handling missing data were implemented for each analysis as described below.

3.2 - Factor Structure of CG

Data screening. Before conducting EFA, data were analyzed for quality. Any participant who omitted 50% or more of the CG, PTS2, or K6_2 items was considered to be a poor responder and was excluded from further analysis. The final *N* was 825. For EFA, expectation maximization (EM) was conducted with EQS 6.1, because there is currently no method for pooling multiple imputation estimates of factor loadings. (This procedure produced EFA results that were very similar to those obtained when listwise deletion was used.) EFA was conducted with IBM SPSS 20 Statistics on the entire sample using the original data (i.e., items were scored on 0–4 Likert-type scales); the resulting scale (i.e., the finalized CG measure) was then tested on the following three occasions: (a) on the entire sample with dichotomized data (i.e., symptom present/absent), (b) on a clinical subset of the sample using the original data, and (c) on the clinical subset using dichotomized data. Items were dichotomized with 0, 1, and 2 indicating that a symptom was not endorsed, with responses 3 and 4 suggesting significant frequency to qualify as a symptom. Although the anchoring statements are different among the three symptom scales at Wave 2, they are similar enough to suggest that this approach is appropriate across scales.

EFA on the whole sample using original 0–4 scale. Results of the EFA on all 28 CG items showed a probable one-factor solution (see Figure 1). Although the parallel analysis indicated that the first three factors were significant, the author chose to retain only one factor, due to theoretical considerations (i.e., the fact that previous studies have reported one-factor solutions when analyzing entire samples). Most of the items had factor loadings above .40; however, two items ('did you experience pain in the same area of your body as where they were shot?' and 'did you hear their voice speaking to you?') were dropped from the scale due to poor loadings (Table 12).

As a first step to explore the factor structure among CG and PTS2 items, an EFA was conducted on the PTS2 items. Although a three- or four-factor solution was expected, a clear one-factor solution emerged (Table 13). All items had loadings between .632 and .791, indicating that they are very good to excellent indicators of the PTS2 factor (Comrey & Lee, 1992). The remaining 26 CG items and the 10 PTS2 items were analyzed to determine whether any items cross-loaded. Because an EFA solution with two correlated factors was anticipated based on the previously conducted analyses, an oblique rotation (i.e., direct oblimin with Kaiser normalization) was utilized. Parallel analysis revealed that the first three factors were significant and could be retained; however, a two-factor solution was preferred for theoretical reasons. The pattern matrix (Table 14) shows that all of the PTS2 and most of the CG items load onto their respective factors with minimal cross-loadings; however, three items (i.e., 'did you go out of your way to avoid reminders of them?', 'did you feel more distant from people you care about than before the shootings?', and 'did you worry that you might suddenly lose someone else you care about?') had cross-loadings greater than or equal to .40. These three items were dropped from the scale in later analyses. Although one item (i.e., 'did you feel like you lost the ability to care about people?') did have a loading less than .40, it was retained on a provisional basis, because it did not cross-load onto PTS2.

The remaining 23 CG items were factor analyzed with the PTS2 and K6_2. Parallel analysis revealed that the first three factors were significant and could be retained, which was consistent with the a priori prediction that each construct would have a separate but correlated factor. The pattern matrix (Table 15) shows that all of the PTS2 and K6_2 items loaded onto their respective factors. One CG item (i.e., 'did you feel like you lost the ability to care about people?') cross-loaded onto the K6_2 factor. After removing this item from the pool of CG

items, the 22 remaining items were analyzed to explore their factor loadings (Table 16). Sorting the loadings in descending order demonstrates an apparent gap in the loadings, suggesting that the last item (i.e., 'did you have a vision of them standing before you?') is not as effective an indicator of CG as are the other items. This item was dropped from the scale, and the EFA was run with the remaining 21 items. The item loadings did not change by more than .002. These 21 items all had similarly high loadings in this analysis and did not demonstrate significant (i.e., > .32) cross-loadings on PTS2 and K6_2 in the previously analyses; therefore, the finalized CG scale for this study is comprised of these items. To calculate final loadings among the factors, a final EFA was run with the CG–21, the PTS2, and the K6_2 (Table 17). A parallel analysis confirmed that there were three significant factors in the final solution (Figure 2). The CG and PTS2 factors were correlated at r = .63, CG and K6_2 were correlated at r = .46, and PTS2 and K6_2 were correlated at r = .41.

EFA on the whole sample using dichotomized items. The dichotomized items of the CG–21 were factor analyzed among the entire sample, using direct oblimin with Kaiser normalization. Parallel analysis indicated that 3 factors could be retained (Figure 3); however, doing so led to a Heywood case (i.e., estimated extracted communalities exceeded 1.0 for at least one variable). Retaining 2 factors resolved the Heywood case problem; however, some items did not load significantly on either of the factors, suggesting that this solution might not be adequate (Table 18). A one-factor solution was preferred, because its structure appeared more stable: All but two of the items had loadings above .40 (Table 19). Furthermore, previous research (including the EFA conducted above on the original scaling of the data) has provided strong evidence that CG has a unitary factor structure.

EFA on the clinical subsample. A clinical subsample was defined by determining a cutoff on the CG–21 that would be similar to the clinical cutoff used on the ICG. The original ICG has a range of 0–76; the range of the CG–21 is 0–84. Because the current clinical cutoff on the ICG is 30 (39% of the high score), the corresponding score on the CG–21 is approximately 33. Although this procedure cannot be validated by the available data, it provided a subsample of 104 high scorers on the CG–21 that will be adequate for the purposes of this study. Of these 104 students, 35 reported losing a significant other or close friend, 18 reported losing a not-close friend, 7 reported losing someone whom they knew well but did not identify as a friend, 38 reported losing an acquaintance, 60 reported that someone they knew lost someone close to

them, and 1 indicated losing a professor (values sum to greater than 104, because many participants reported losses in more than one relationship category). In sum, 35 participants in the clinical severity subset experienced bereavement that would, based on current conceptualizations of CG criteria, qualify them for a CG diagnosis. Only 10 participants in the clinical subgroup did not report being bereaved in any way; it is unclear how these individuals experienced symptoms of CG when they did not identify being bereaved. (Nine of these 10 participants had CG–21 total scores ranging from 33 to 41, indicating that they were in the lower end of the distribution of scores among participants who scored above the clinical cutoff. The other individual had a total score of 48.) It is possible that they were simply upset about the thought of other students from the Virginia Tech community being killed, but this speculation does not explain why these individuals scored so high on a measure of CG, which assumes that one is bereaved of a close loved one.

Parallel analysis indicated that two factors were significant (Figure 4); however, there is reason to be suspect of these results. First, the CG–21 correlation matrix among the clinical subsample contains several near-zero and negative correlations, suggesting that the matrix might not be suitable for factor analysis. Second, the pattern matrix illustrates that several items do not load significantly on either factor (Table 20). EFA on the dichotomized items produced very similar results: The correlation matrix had near-zero and negative correlations, and several items did not load onto either factor in the pattern matrix. The complications of the EFA among the clinical subsample suggest that the sample size is not large enough to explore the factor structure of CG at clinical severity. Therefore, CFA will not be conducted on this subset.

EFA summary. Results indicate that CG is a unitary construct that is distinct from PTS and general anxiety/mood symptoms, which is consistent with previous research and the hypotheses of this study. The final scale includes 21 of the 28 questionnaire items and has excellent reliability ($\alpha = .95$). Unfortunately, the clinical subsample ($n = 104$) was too small to obtain reliable EFA solutions, making it impossible to explore the possible symptom clusters of CG.

CFA. Two CFA models were hypothesized to fit the data: a one-factor model of the CG–21, and a three-factor model of the CG–21, the PTS2, and the K6_2. Due to the capabilities and limitations of the EQS software, certain procedures were mutually exclusive. For instance, FIML for handling missing data was available only when using ML as the estimator and when

classifying all variables as continuous. In such situations, the appropriate estimator was given priority over estimating or imputing missing data. Therefore, $N = 756$ for the CFA models with the CG–21 items, and $N = 721$ for CFA models on all three symptom scales. Another limitation of EQS is that error variance estimates cannot be constrained to be positive when analyzing ordinal data. Whenever improper solutions were derived via AGLS estimation, this was addressed by switching to ML estimation with robust standard errors, which resolved such issues.

Using the AGLS estimator, the one-factor CG–21 model had acceptable fit as measured by two of the fit indices, RMSEA = .063, 90% CI = .058–.067, and AAFI = .979. However, fit was unacceptable as measured by the SRMR = .242 and $\chi_{agls}^2 = 315.46$, $df = 189$, $p < .001$. There were no outliers in the distribution of standardized residuals, suggesting an absence of localized areas of strain; the largest deviation had an absolute value of .40. Thus, the model did not replicate the observed correlation matrix well. However, because there is currently debate regarding the use and interpretation of the SRMR when estimating model fit with alternative estimators (e.g., AGLS) on ordinal-level data (Beauducel & Herzberg, 2006; Brown, 2006), the model was also estimated with ML ROBUST. This produced mediocre but acceptable fit indices, RMSEA = .083 (CI = .079–.088), CFI = .986, SRMR = .051, although the model produced a significant χ^2 statistic, Satorra-Bentler Scaled $\chi^2 = 1175.47$, $df = 189$, $p < .001$. An exploration of the standardized residuals revealed that the ML ROBUST-estimated model generally reproduced the observed correlation matrix within acceptable limits, suggesting an absence of any localized areas of strain; however, the largest standardized residual had an absolute value of .21. Although the model did not have spectacular fit—especially as estimated by the RMSEA and the Satorra-Bentler Scaled χ^2—a one-factor model was retained. This was decided because, overall, the CFA findings seem to support a one-factor model for the CG–21, especially when interpreted in light of the results of the EFA reported above. Completely standardized factor loadings for each item as estimated by ML ROBUST are provided in Table 21.

After confirming the factor structure of the CG–21, the three symptom scales (i.e., the CG–21, the PTS2, and the K6_2) were examined in a three-factor CFA utilizing the AGLS estimator. However, this procedure resulted in an improper solution; specifically, four of the item error variance estimates were negative. Due to the restrictions of the EQS software mentioned above, the only available solution to this problem was to estimate the model using ML ROBUST

rather than AGLS; variables were still handled as ordinal data. Three of the fit indices produced by ML ROBUST estimation suggested that the model had acceptable fit with the data, RMSEA = .058, CI = .056–.061, CFI = .985, SRMR = .055; however, the χ^2 estimate did not suggest good model fit, Satorra-Bentler Scaled χ^2 = 2162.18, df = 626, p < .001. An exploration of the standardized residuals revealed an absence of any localized areas of strain; the largest standardized residual had an absolute value of .21 but was not an outlier compared to other residuals. CG and PTS2 were correlated at r = .70, CG and K6_2 were correlated at r = .61, and PTS2 and K6_2 were correlated at r = .55.

To test a competing hypothesis that the three symptom scales could all be measuring a single construct, a one-factor model was also tested. The model generally did not demonstrate adequate fit, RMSEA = .106, CI = .103–.108, SRMR = .091, Satorra-Bentler Scaled χ^2 = 5708.26, df = 629, p < .001, although the CFI was acceptable, CFI = .950. Furthermore, the direct comparison of the two models revealed that the three-factor model was superior to the one-factor model, AIC = 910.19 vs. 4450.26, respectively. In addition, a two-factor model, in which CG–21 and PTS2 items were all modeled as indicators of a shared factor and K6_2 items were indicators of a separate, correlated factor, also had poor fit with the data and was inferior to the three-factor model, RMSEA = .090, CI = .087–.092, CFI = .964, SRMR = .080, AIC = 3027.69, Satorra-Bentler Scaled χ^2 = 4283.69, df = 628, p < .001. Therefore, the results provide sufficient evidence that the three-factor model adequately fits data from the three symptom scales; completely standardized factor loadings for each item are provided in Table 22.

3.3 - Predicting Bereavement Status

Continuous CG. The hypothesis that CG symptoms would be more strongly related to bereavement than PTS symptoms would be was tested by predicting bereavement status (0 = *not bereaved*, 1 = *bereaved*) with symptom totals from the CG–21 and PTS2. Symptom totals from Wave 1 (i.e., PTS1, K6_1, and grief1) and previous traumatic experiences were entered as control variables in Step 1; CG–21, PTS2, and K6_2 were entered in Step 2.

Logistic regression was conducted using SPSS 20. Missing values were imputed using the Multiple Imputation procedure of SPSS 20, following guidelines provided by Enders (2010). In order to ensure stable coefficient estimates, 100 datasets with imputed values were created. Estimates of model fit were based on the listwise deletion sample (N = 721), whereas coefficient estimates and odds ratios for predictors were based on the pooled estimates from the original

data and the imputed datasets. Similarly, Wald χ^2 values for individual predictors were from the original data with no imputed values (N = 721), because pooled estimates were not available. Although pooled estimates for model fit were unavailable, model fit indices (i.e., χ^2 and Nagelkerke's R^2) for each imputed dataset were examined and found to be similar to those of the original data. The 95% confidence interval of Nagelkerke's R^2 was calculated on the original data using the formula suggested by Olkin and Finn (1995).

Among continuous predictors, control variables (i.e., previous traumatic experiences and Wave 1 trauma-related psychopathology), and dependent variables, there was significant deviation from MCAR based on Little's MCAR test, $\chi^2(27) = 42.186$, $p = .032$. However, the relationship between K6_1 and K6_2 missingness (reported above) appeared to account for this deviation: Little's MCAR test was no longer significant after removing K6_2, $\chi^2(11) = 6.883$, $p = .808$. Therefore, the model as tested (which included K6_2) had a missing at random mechanism, suggesting that multiple imputation for missing values is more appropriate than listwise deletion.

A test of a model with only the control variables from Wave 1 was statistically significant, $\chi^2(14, 721) = 60.36$, $p < .001$, indicating that the control variables as a set significantly predicted bereavement status. As a set, the Wave 2 symptom total variables significantly improved prediction of bereavement, $\chi^2(3, 721) = 25.34$, $p < .001$. The final model was significant, $\chi^2(17, 721) = 85.70$, $p < .001$. The overall effect size of the model was small, Nagelkerke's $R^2 = .21$, 95% confidence interval = .16–.26. Furthermore, participant classification as predicted by the model was uneven: Although 99% of non-bereaved participants were accurately classified, only 9% of bereaved individuals were correctly classified (overall accuracy = 88%).

Regression coefficients, Wald statistics, odds ratios (ORs), and 95% confidence intervals for odds ratios are presented in Table 23. Of the three predictors of interest, only CG–21 was significant when tested within the complete model, Wald $\chi^2(1, 721) = 21.97$, $p < .001$. Three of the control variables were significant: accidental death of a loved one (lifetime prior to 4/16), Wald $\chi^2(1, 721) = 3.66$, $p < .05$, personal life-threatening illness or injury (during the year before 4/16), Wald $\chi^2(1, 721) = 3.05$, $p < .05$, and Grief1, Wald $\chi^2(1, 721) = 6.43$, $p < .01$.

When controlling for other symptoms and previous traumatic experiences, a one-unit increase on the CG–21 has a small effect on predicting bereavement, OR = 1.06, 95% confidence interval = 1.03–1.08. This means that an individual who scored 1 standard deviation above the mean on the CG–21 (*SD* = 13.81, based on complete data) would be 14.64 times more likely to have been bereaved than would be an individual who scored at the mean.

Replication with case/non-case estimates. The above regression analyses were repeated but with binary (i.e., case/non-case) estimates of *DSM–5* CG and PTSD. Participants were classified as PTSD cases or non-cases using the algorithm described above in the Method section; CG classification was based on the clinical cutoff described above when reporting results of the clinical subsample EFA. Analyses were conducted on a multiple-imputed dataset as described above, except that PTS2 and CG were categorical variables. Little's MCAR test revealed that there was no significant deviation from MCAR among the continuous variables in the model, $\chi^2(3) = 9.289$, $p = .026$. Closer exploration indicated that this was due to the relationship between K6_1 and K6_2, as reported above; this was verified by removing K6_2 and repeating Little's MCAR test, which was no longer significant, $\chi^2(3) = 0.698$, $p = .874$. Therefore, similar to the analysis conducted on the continuous variables (above), the model as tested had a missing at random mechanism.

Similar to as reported above, the control variables from Wave 1 as a set were statistically significant predictors of bereavement, $\chi^2(14, 721) = 58.81$, $p < .001$. As a set, the Wave 2 symptom total variables significantly improved prediction of bereavement, $\chi^2(3, 721) = 11.00$, $p < .05$. The final model was significant, $\chi^2(17, 721) = 69.81$, $p < .001$. The overall effect size of the model was small, Nagelkerke's $R^2 = .17$, 95% confidence interval = .12–.22. Furthermore, participant classification as predicted by the model was uneven: Although 99% of non-bereaved participants were accurately classified, only 7% of bereaved individuals were correctly classified (overall accuracy = 88%). The binary CG estimate was the only predictor of interest that was significant when tested within the complete model, Wald $\chi^2(1, 721) = 10.52$, $p < .01$; Grief was the only significant control variable, Wald $\chi^2(1, 721) = 13.58$, $p < .001$. When controlling for other symptoms and previous traumatic experiences, CG case/non-case status was a strong predictor of bereavement status, OR = 2.86, 95% confidence interval = 1.51–5.39.

3.4 - Predicting Trauma Exposure

The hypothesis that exposure to the shootings would be more related to PTS than to CG was tested by conducting a series of regression models. As described above, multiple imputation was utilized to handle missing data. Estimates of model performance were based on the listwise deletion sample (N = 721), whereas coefficient estimates for predictors were based on the pooled estimates from the original data combined with the imputed datasets. One of 3 exposure variables (i.e., exposure, awareness, and locked-in status) was the criterion in each model. Consistent with the approach employed above, each model controlled for previous trauma and Wave 1 psychopathology (i.e., PTS1, Grief, and K6_1); predictors of interest were PTS2, CG, and K6_2.

The model for predicting exposure was significant, R^2 = .06, F(17, 703) = 2.71, p < .001; however, the predictors of interest were not significant. Similarly, none of the predictors of interest were significant when awareness was the criterion, although the model as a whole was significant, R^2 = .06, F(17, 703) = 2.46, p < .01. For the exposure and the awareness models, standardized coefficients, t values, p values, and squared semipartial correlations are presented in Tables 24 and 25, respectively.

For predicting locked-in status, the overall model was not significant, χ^2(17, 721) = 18.48, p = .359. Consistent with this, the overall effect size of the model was negligible, Nagelkerke's R^2 = .03, 95% confidence interval = .01–.06. Furthermore, participant classification as predicted by the model was mediocre (overall classification accuracy = 58.3%). Regression coefficients, Wald statistics, odds ratios (ORs), and 95% confidence intervals for odds ratios are presented in Table 26. Of the three predictors of interest, only CG–21 was significant when tested within the complete model, Wald χ^2(1, 721) = 4.90, p < .05.

3.5 - Unable to Confirm a Friend's Safety

Continuous CG and PTS. Regression analyses were performed in order to test the hypothesis that being unable to confirm a close friend's safety would be a significant predictor of CG symptoms among traumatically bereaved students (N = 103). Because 10 cases were missing at least one item on the CG–21, regression was conducted on the imputed datasets (described above). Based on the original, complete data, the overall model was not significant, R^2 = .01, F(1, 91) = 1.06, p = .307. Similarly, the pooled coefficient for being unable to confirm a close friend's safety was not significant, B = 5.85, t(101) = 1.25, p = .212. These results were contrary

to the hypothesis. Furthermore, being unable to confirm a close friend's safety did not significantly predict PTS2, $R^2 = .04$, $F(1, 99) = 3.80$, $p = .054$, $B = 6.04$, $t(101) = 1.96$, $p = .050$. An examination of the frequencies of the predictor revealed that 91 of the 103 bereaved participants indicated that they had been unable to confirm a close friend's safety, suggesting that this variable demonstrated significant skewness.

In order to further explore the relationship between being unable to confirm a close friend's safety and CG, the definition of *bereavement* was expanded to include not-close friends. Among this subset of 284 students, 234 indicated that they had been unable to confirm a close friend's safety, suggesting that the predictor is still considerably skewed (although not as badly as among the 103 students who lost a significant other or close friend). Results of these analyses indicated that being unable to confirm a close friend's safety significantly predicted scores on the CG–21, $R^2 = .04$, $F(1, 259) = 11.30$, $p < .001$, $B = 7.42$, $t(282) = 3.35$, $p < .001$, as well as scores on the PTS2, $R^2 = .06$, $F(1, 274) = 16.75$, $p < .001$, $B = 5.97$, $t(282) = 4.08$, $p < .001$. These findings provide limited support for this hypothesis.

Replication with case/non-case estimates. The above regression analyses were repeated in separate logistic regressions using the binary estimates of CG and PTSD case/non-case status as the criterion variables. Based on the 93 cases with no missing data for CG caseness (all bereaved participants had data for PTSD caseness), the overall model was not significant, $\chi^2(1, 93) = 1.61$, $p = .204$. Similarly, the coefficient estimate for being unable to confirm a close friend's safety was not significant, Wald $\chi^2(1, 93) = 1.40$, $p = .237$. In addition, being unable to confirm a close friend's safety did not significantly predict PTS2, $\chi^2(1, 103) = 1.82$, $p = .177$, Wald $\chi^2(1, 103) = 1.54$, $p = .215$. Logistic regression analyses were also performed on the participants who lost close friends and/or not-close friends. In the first analysis, being unable to confirm a close friend's safety significantly predicted CG caseness, $\chi^2(1, 93) = 11.54$, $p < .001$, Wald $\chi^2(1, 93) = 7.63$, $p < .01$, as well as scores on the PTS2, $\chi^2(1, 103) = 15.07$, $p < .001$, Wald $\chi^2(1, 103) = 9.42$, $p < .01$. Being unable to confirm a close friend's safety was highly predictive of CG caseness, OR = 5.12, 95% confidence interval = 1.53–17.12, and of PTSD caseness, OR = 6.55, 95% confidence interval = 1.97–21.76.

3.6 - Interaction between Media Exposure and Direct Exposure

Continuous CG and PTS. Finally, among individuals who were traumatically bereaved by the shootings, it was hypothesized that there would be an interaction between watching media

coverage of the shootings and direct exposure, such that the relationship between direct exposure and PTS2 would be stronger among individuals who watched more media coverage than those who watched less. Multiple hierarchical regression was conducted in two steps: The predictor (i.e., exposure) and the moderator (i.e., exposure to media) were entered in the first step, and the interaction term was entered in the second step. Listwise deletion was employed, which dropped 2 cases from analyses (N = 101). This was chosen rather than using the multiple imputation datasets created as described above, because there are complications in centering the variables when utilizing multiple imputation (Enders, 2010). Because only 2 cases had missing values, listwise deletion seemed appropriate, especially because the variables in this regression model appear to be MCAR among bereaved participants based on Little's MCAR test, $\chi^2(3) = 1.104$, $p = .776$. The predictor and moderator were centered at the mean in order to eliminate nonessential multicollinearity; the interaction term was created using the centered variables (Cohen, Cohen, West, & Aiken, 2003).

The model was significant at step one, $R^2 = .06$, $F(2, 98) = 3.26$, $p < .05$. Exposure was a significant predictor of PTS2, $B = 1.44$, $t(99) = 2.53$, $p < .05$; however, media exposure was not significant, $B = -0.18$, $t(99) = 0.22$, $p = .83$. However, the overall model was not significant after adding the interaction term, $R^2 = .07$, $F(3, 97) = 2.35$, $p = .077$. Exposure continued to be a significant predictor, $B = 1.38$, $t(99) = 2.38$, $p < .05$; however, the interaction term was not significant, $B = 0.41$, $t(99) = 0.74$, $p = .461$, nor was media exposure, $B = -0.11$, $t(99) = 0.13$, $p = .895$. These results did not support the hypothesis.

Post hoc power analysis for the above model, using $\alpha = .05$, $N = 101$, and an estimated effect size of .02 (a common effect size for interaction terms in psychological research; Cohen et al., 2003), indicates that the achieved power was .29, which is unacceptable. Even if the assumptions are altered so that the interaction has an effect size of .05, the achieved power is still only .60. Indeed, Cohen et al. (2003) suggest that, if predictors contain no measurement error (an assumption that is not being made in this study), a sample size of 392 would be required to detect small effect size interactions.

In order to increase power, this hypothesis was also tested when adding students who lost a not-close friend, increasing the potential N to 284. Multiple hierarchical regression was conducted as outlined above. Again, listwise deletion was utilized with minimal loss of cases (N = 276, a loss of only 8 participants), because the variables in this regression model seem to be

MCAR based on Little's MCAR test, $\chi^2(3) = 3.89$, $p = .274$. The model was not significant at step one, $R^2 = .02$, $F(2, 273) = 2.11$, $p = .123$. Exposure and media exposure were not significant, $B = 0.64$, $t(274) = 1.90$, $p = .058$ and $B = 0.44$, $t(274) = 0.87$, $p = .383$, respectively. The overall model was not significant after adding the interaction term, $R^2 = .02$, $F(3, 272) = 1.53$, $p = .208$, nor was the interaction term significant, $B = 0.19$, $t(274) = 0.61$, $p = .543$. These results did not support the hypothesis. Post hoc power analysis with $\alpha = .05$, $N = 276$, and an assumed effect size of .02 indicates that the achieved power was .65. Although this is not ideal, it is a significant improvement over .29. Furthermore, if the effect size of the interaction term is assumed to be .05, the achieved power is .96.

Replication of interaction using case/non-case estimates. The test for media exposure as a moderator of direct exposure in the prediction of PTS was repeated using a case/non-case estimate of PTSD. The steps outlined above were repeated using logistic regression (because the criterion was binary); there were no missing observations in these analyses.

Among students who lost a close friend or significant other, the model was not significant at step one, $\chi^2(2, 103) = 0.16$, $p = .925$. Exposure was not a significant predictor of estimated PTSD case/non-case status, Wald $\chi^2(1, 103) = 0.09$, $p = .767$; media exposure was also not significant, Wald $\chi^2(1, 103) = 0.06$, $p = .805$. The model was not significant after adding the interaction term, $\chi^2(3, 103) = 0.52$, $p = .915$. Coefficients were not significant for exposure, Wald $\chi^2(1, 103) = 0.04$, $p = .836$, media exposure, Wald $\chi^2(1, 103) = 0.04$, $p = .848$, and the interaction term, Wald $\chi^2(1, 103) = 0.36$, $p = .549$. These results are similar to those of the ordinary least squares regression analyses reported above.

Among students who lost a close friend and/or a not-close friend, the model was not significant at step one, $\chi^2(2, 284) = 2.38$, $p = .304$. Exposure and media exposure were not significant, Wald $\chi^2(1, 284) = 1.50$, $p = .221$, and Wald $\chi^2(1, 284) = 1.03$, $p = .309$, respectively. The overall model was not significant after adding the interaction term, $\chi^2(3, 284) = 3.46$, $p = .326$. Coefficients were not significant for exposure, Wald $\chi^2(1, 284) = 1.04$, $p = .307$, media exposure, Wald $\chi^2(1, 284) = 0.98$, $p = .322$, and the interaction term, Wald $\chi^2(1, 284) = 1.07$, $p = .302$. These results are similar to those of the ordinary least squares regression analyses reported above.

3.7 - SEM Analyses

It was hypothesized that exposure and perceived threat would predict PTS, whereas bereavement and related threats (e.g., having a friend escape from a classroom during the shootings) were hypothesized to predict CG. This hypothesis was tested by comparing two competing models. In the predicted model, exposure and perceived threat factors had pathways to PTS but not to CG; similarly, the factor of bereavement (and related events) had a pathway to CG but not to PTS. In the alternative model, pathways were added to see whether CG and PTS were significantly predicted by the other type of exposure event.

These models were estimated using ML, with the outcome variables (i.e., items on the CG and the PTS2) classified as ordinal, just as in the CFA above. Predictor variables were classified as continuous regardless of their actual nature, because failing to classify categorical predictors as categorical generally is not problematic in EQS 6.1 (Bentler, 2006). Furthermore, attempts to classify the predictors as categorical led to convergence problems. Because the models were estimated with ML with ordinal-level data, missing values could not be handled using the MISSING = ML option to provide FIML estimates for parameters. Therefore, listwise deletion was used. This was not anticipated to cause problems, because the data in these analyses did not violate the MCAR assumption, based on means and variance homogeneity $\chi 2$ tests conducted within the SEM analyses. Because of extreme deviations from multivariate normality, ML ROBUST standard errors were used to interpret model fit indices and parameter significance tests.

Given the hypothesized relationships between (a) exposure and PTS and (b) bereavement and CG, the predicted model was tested and compared with the alternative model. A depiction of the relationships among the latent variables is presented in Figure 5.

The predicted model had acceptable fit as measured by three fit indices, RMSEA = .043, 90% CI = .041–.046, SRMR = .055, CFI = .989; however, poor fit was indicated by Satorra-Bentler Scaled $\chi 2 = 1660.04$, df = 697, $p < .001$. The alternative model demonstrated very similar fit: RMSEA = .043, 90% CI = .040–.046, SRMR = .054, CFI = .989, and Satorra-Bentler Scaled $\chi 2 = 1641.07$, df = 694, $p < .001$. For both models, the hypothesis of exact fit is rejected, because the 90% confidence intervals for RMSEA do not include 0. Hypothesis of close fit is not rejected, because the lower bound of the confidence interval is below.050. Finally, the

hypothesis of not-close fit is rejected, because the upper bound of the confidence interval is below .050. Comparing the models directly revealed that the predicted model had worse fit than the alternative model had, AIC = 266.04 vs. 253.07, respectively.

Closer inspection of the results revealed that the Exposure latent factor did not have significant pathways to either PTS2 or CG. This issue was further examined by exploring correlations between Exposure indicators (i.e., exposure, helping an injured individual, and being in lockdown) and PTS2 total scores. The correlation between the exposure scale and PTS2 was .08, $p < .05$; the correlations between helping an injured individual and PTS2 and between being in lockdown and PTS2 were not significant ($r = -.01$ and $-.03$, $p = .786$ and .374, respectively). Furthermore, only 11 participants reported having helped an injured individual, suggesting that this particular item is very skewed and therefore has little predictive power in the sample. Therefore, Exposure was considered to be a nonsignificant predictor of PTS2, and both models were retested with the omission of this construct and its indicators.

After determining that Threat and Interpersonal Loss were significant predictors of PTS and CG, a latent construct, Previous Trauma, was added to the model as a control. Indicators of Previous Trauma were items that assessed whether participants had ever before 4/16 experienced an assault, life-threatening accident or injury, murder/suicide of a loved one, any other risk of death, or had witnessed someone being seriously injured or killed. It was hypothesized that Previous Trauma would be related to PTS2 and CG; therefore, paths leading to each construct of psychopathology were included in the model. As above, two versions of the model were tested: one with the predicted pathways from Threat to PTS2 and from Interpersonal Loss to CG, and an alternative with additional pathways from Threat to CG and from Interpersonal Loss to PTS2.

The predicted model had acceptable fit as measured by three of the fit indices, RMSEA = .037, 90% CI = .035–.040, SRMR = .046, CFI = .990; however, poor fit was suggested by the Satorra-Bentler Scaled $\chi2 = 1640.05$, $df = 811$, $p < .001$. The alternative model also had acceptable fit as measured by two fit indices, RMSEA = .037, 90% CI = .034–.040, SRMR = .051, although the CFI and $\chi2$ were again below the cutoff, CFI = .991, Satorra-Bentler Scaled $\chi2 = 1618.33$, $df = 809$, $p < .001$. For both models, the hypothesis of exact fit is rejected, because the 90% confidence intervals for RMSEA do not include 0. Hypothesis of close fit is not rejected, because the lower bound of the confidence interval is below.050. Finally, the

hypothesis of not-close fit is rejected, because the upper bound of the confidence interval is below .050. Comparing the models directly revealed that the predicted model had worse fit than the alternative model had, AIC = 18.05 vs. 0.33.

Although both of the above models did generally have evidence of acceptable fit, the pathways from the previous trauma latent construct to both psychopathology constructs were not significant in the alternative model, suggesting that the indicators of previous trauma were not significant predictors of CG or PTS. Therefore, this model is misspecified, and Previous Trauma must be removed in order to compare the pathways of interest (i.e., even though these pathways were significant in the predicted model, the misspecification in the alternative model makes it impossible to compare the models for the pathways of interest).

The revised predicted model (i.e., excluding Exposure and Previous Trauma) had acceptable fit as measured by RMSEA = .047, CI = .044–.049, SRMR = .049, and CFI = .990; however, poor fit was suggested by Satorra-Bentler Scaled $\chi 2 = 1534.03$, $df = 592$, $p < .001$. The alternative model also had acceptable fit as measured by two fit indices, RMSEA = .046, CI = .043–.049, SRMR = .048, and CFI = .990; again, the Satorra-Bentler Scaled $\chi 2 = 1506.58$, $df = 588$, $p < .001$, did not suggest good fit. For both models, the hypotheses of exact fit, close fit, and not-close fit were identical to those reported for the models above. Comparing the models directly revealed that the predicted model had worse fit than the alternative model had, AIC = 350.03 vs. 330.58, respectively; however, there was a nonsignificant path from Interpersonal Loss to PTS, suggesting that the alternative model was misspecified.

The final model was therefore estimated based on the predicted model but with a pathway added from Perceived Threat to CG. This model had acceptable fit as measured by RMSEA = .046, CI = .043–.049, SRMR = .048, and CFI = .990; however, poor fit was suggested by Satorra-Bentler Scaled $\chi 2 = 1507.82$, $df = 589$, $p < .001$. Hypotheses of exact fit, close fit, and not-close fit were the same as above. The AIC showed an advantage for this model over the predicted model, AIC = 329.82 vs. 350.03, respectively. Furthermore, the final model included all significant pathways from the exogenous latent constructs to the endogenous constructs, suggesting that the predicted model—by not include a pathway from Perceived Threat to CG—is misspecified. Therefore, the final model (Figure 6) was retained as the best model of perceived threat, interpersonal loss, CG, and PTS for this sample.

Chapter 4 - Discussion

The first hypothesis was that a stable factor structure for CG would be discovered with appropriate levels of internal consistency; it was anticipated that a one-factor solution would best fit the entire sample. Results from the EFA provided strong evidence consistent with this hypothesis. Of the 28 survey items intended to measure CG, 21 of them were retained for the final scale. The retained items did not cross-load onto other factors of psychopathology, as demonstrated in the EFA that included PTS2 and K6_2. Furthermore, CFA results corroborated the EFA findings that the CG–21 has a unitary factor structure and that the three symptom scales combined can be adequately modeled with 3 factors. Thus, the CG–21 is an internally consistent measure of CG, and CG is a distinct construct from PTS and general anxiety/depression among a sample of traumatically bereaved young adults.

After exploring and confirming the factor structure of CG among the entire sample, EFA was utilized on a clinical subsample in an attempt to replicate previous research (Simon et al., 2011) that suggested the presence of 6 symptom clusters among CG. Unfortunately, the relatively small size of the clinical subsample (n = 104) was insufficient to provide stable estimates of the factor structure in EFA; due to this problem, CFA was not attempted. Therefore, the symptom clusters of CG could not be explored.

After exploring and confirming the factor structure of CG and creating the CG–21, the CG–21 was used in logistic regression to test the hypothesis that CG would be more related to bereavement status than would be PTS. Scores on the CG–21 predicted bereavement status, but scores on the PTS_2 did not. In fact, the CG–21 was a powerful predictor: When controlling for previous traumas and symptoms of psychopathology at Wave 1, participants who scored 1 standard deviation above the mean on the CG scale were 14.64 times more likely to have lost a close friend than were those who scored at the mean. When CG–21 scores were dichotomized into a case/non-case estimate, it remained a significant predictor of bereavement status. Therefore, the hypothesis that CG would be a better predictor of bereavement status than PTS would be was supported.

The corollary hypothesis to the above was that PTS would be more related to direct exposure than CG would be. This hypothesis was not supported: When controlling for previous trauma and Wave 1 psychopathology, neither CG nor PTS were related to direct exposure to or

awareness of the shootings. Furthermore, CG was related to locked-in status, whereas PTS was not; however, the interpretation of this finding is unclear, because higher scores on the CG–21 were predictive of lower probabilities of being locked in a room on campus during the shootings.

Given the conceptualization of CG—and the apparent distinctiveness of this disorder from PTSD and PTS (noted above)—it is unclear why being locked in during the shootings would be related to CG symptoms. Although objective exposure was conceptualized as a distinct influence from bereavement and related interpersonal losses, these factors are by no means mutually exclusive; although correlations among exposure and bereavement-related events ranged from non-significant to significant but of small effect size (see Tables 5, 6, and 7), it is possible that other variables not included in this project mediate or moderate the relationships between measures of objective exposure and of interpersonal loss. Future research could explore these possibilities, thereby explaining the relationship between locked-in status and bereavement. For instance, it might be that, among bereaved individuals, those who were locked in were in more peritraumatic distress, predisposing them to develop symptoms of CG. Furthermore, it is possible, given the number of comparisons and the rather small effect size of this finding, that this is a spurious outcome. In light of the dose-response model, it is also surprising that none of the regression results found that exposure variables were related to PTS at Wave 2. The lack of supportive findings could be due to problems with defining and measuring exposure; indeed, these variables were not included in the final SEM model because of their lack of predictive power. Although no *a priori* hypotheses were made regarding Wave 1 psychopathology, it is interesting to note that PTS at Wave 1 (and no other measure of psychopathology at either time point) was a significant predictor of exposure. However, the exploratory nature of this finding precludes any true hypothesis testing.

Taken together, the regression analyses conducted to test hypotheses about distinct pathways for CG and PTS provide mixed results. CG generally seems to be related uniquely to bereavement, although whether someone was locked in during the shootings was related to later development of CG but not PTSD. Direct exposure was related strongly to symptoms of either disorder. SEM analyses (discussed below) were conducted in order to clarify the nature of these relationships.

The hypothesis that being unable to confirm a close friend's safety would be a significant predictor of CG among traumatically bereaved students was partially supported, but only with important limitations. Being unable to confirm a close friend's safety during the shootings was not related CG among bereaved individuals when using the strict (and arguably most appropriate) definition of *bereavement* (i.e., the death of a significant other or close friend). Both CG and PTS2 were significant predictors when the definition of *bereavement* was loosened to include the death of not-close friends as well as close friends and significant others. However, the current conceptualization of CG is that it is a pathological, prolonged grief reaction to a loved one's death; therefore, it is unclear how relevant CG-like reactions are in response to a not-close friend's death. Furthermore, PTS2 was not expected to be a significant predictor of being unable to confirm a close friend's safety. Bereavement as defined in either scenario was heavily skewed, although somewhat less so when the loose definition was applied, and the skewness likely attenuated estimates of association. These results suggest that it is possible that being unable to confirm a close friend's safety was a peritraumatic phenomenon that could be related to psychopathology in an indiscriminate manner; there is no evidence from this study that this variable is related specifically to CG (or to PTS).

Given the above results, it is perhaps not surprising that the hypothesis of an interaction between indirect and direct trauma exposure in predicting PTS among traumatically bereaved was not supported. It is interesting to note that, despite the results discussed above regarding the weak association between exposure and PTS2, exposure was a significant predictor of PTS2 among those who lost a significant other or close friend. This is consistent with the dose-response model but is not consistent with other findings in this study (especially the SEM model, reported above and discussed below). This discrepancy among findings is likely due to the lack of control variables (e.g., PTS1, Grief, and previous death of a loved one) utilized in this model compared to the ones discussed above (indeed, exploratory analyses not presented here seem to support this possibility). Although the failure to reject the null hypotheses for interaction terms in the two models analyzed could be due to problems frequently encountered in the dose-response literature (i.e., difficulties in defining and measuring exposure), perhaps the most convincing explanation in this project is that the power was insufficient; indeed, a subsample of 101 bereaved participants is clearly insufficient for testing moderation. Although the second model (i.e., the one with *bereavement* defined as those who lost a not-close friend as well as those who

lost a significant other or close friend) did ameliorate this problem modestly, power was still inadequate, based on the most realistic assumptions regarding the expected size of an interaction term. Furthermore, the approach of addressing a statistical problem by introducing a conceptual problem (in this case, increasing power by changing the operationalization of one of the constructs) is admittedly questionable except under exploratory circumstances. Therefore, given the lack of power in the regression models for testing interaction terms, it seems premature to claim that the hypothesis regarding an interaction between direct and indirect exposure among those who have been traumatically bereaved has been refuted. Future research designed specifically to address this question seems warranted, especially given the current debate on the influence of media coverage of trauma and the development or exacerbation of psychopathology among viewers.

Structural equation modeling was utilized to determine which variables at Wave 1 would predict CG at Wave 2. It was hypothesized that traumatic bereavement and related events (e.g., having a friend injured in the shootings) would be related to CG but would not be significantly predictive of PTS, whereas direct exposure to the shootings—as measured by being on campus, hearing gunshots, proximity to the shootings, seeing someone injured or killed, and being injured—would be more closely related to PTS than to CG. The results of the SEM analyses did not fully support these hypotheses: The accepted model demonstrated that interpersonal loss predicted only CG and not PTS, whereas perceived threat predicted both PTS and CG. Therefore, interpersonal loss was uniquely associated with CG, but perceived threat was not uniquely associated with PTS. Furthermore, neither exposure to 4/16 nor previous traumatic experiences predicted psychopathology at Wave 2. These analyses suggest that perceived threat might play a role in PTS and in CG, whereas bereavement and related events might be more specific to CG.

However, the models estimated to test this hypothesis did have shortcomings. Specifically, the Satorra-Bentler Scaled χ^2 was significant in every model estimated. This might be at least partly attributable to the fairly large sample size. Although the other fit indices provided evidence of good fit, it cannot be ruled out that the Satorra-Bentler Scaled χ^2 is demonstrating some degree of misfit. This might also be due to violations of assumptions of

multivariate normality or the ordinal nature of the data; although the Satorra-Bentler Scaled χ^2 does adjust for deviations from multivariate normality, it is not clear how effective such adjustments are (especially with ordinal data).

It should also be noted that the model with the best fit as measured by fit indices is not necessarily the model that best describes the relationships among the variables in the population. For example, it is possible that adding pathways improves fit significantly, even if the pathways should not exist (e.g., correlating error terms without cause), because of the relationship between sample size and chi square values. Therefore, even though the alternative model was superior to all other models tested, it is possible that the model that truly explains associations and pathways among the population was not even tested. Furthermore, due to the unique nature of the population from which this sample was taken, it is possible that the final SEM model—even if it is accurate for the population in question—is not applicable to other populations (e.g., individuals who have been traumatically bereaved due to single-victim acts of violence).

It was also hypothesized that being unable to confirm a close friend's safety would be a significant predictor of CG among traumatically bereaved students. This hypothesis was not supported, nor was there evidence that, among bereaved students, PTS2 was predicted by being unable to confirm a close friend's safety. This finding was unexpected, given the theorized relationship between being concerned for someone's safety and the later discovery that a close friend (although possibly not the same person) has been killed. Among individuals who lost a significant other, close friend, or a not-close friend, CG was significantly predicted by being unable to verify a close friend's safety; unfortunately, it is unclear how this should be interpreted. If someone is uncertain whether a close friend is safe, how does that influence their reaction to a not-close friend's death? The results of the regression suggest that it predicts higher severity of CG, but the mechanism behind this relationship is unclear. It should be noted that among the 104 bereaved students, 91 (88%) were unable to confirm a close friend's safety; therefore, it is possible that prediction was attenuated by the skewness of this variable. However, in the sample of bereaved students (including those who lost a not-close friend), 234 (82%) of 284 endorsed being unable to verify a close friend's safety, suggesting that skewness may not entirely explain the failure to predict CG among the bereaved subset. It is possible that uncertainty of a loved one's safety during a crisis would be a better predictor of CG among bereaved individuals if future research utilized continuous variables instead of a single

dichotomous item. For example, one could measure the amount of time elapsed in uncertainty of the close friend's safety, or one could measure the subjective distress experienced during the period of uncertainty.

Overall, the results provide support for the validity of the CG construct. CG could be distinguished from PTS and general anxiety/depression symptoms. Furthermore, CG was predictive of bereavement status even after controlling for other traumatic experiences and other psychopathology: Participants who scored 1 standard deviation above the mean on the CG scale were 14.64 times more likely to have lost a close friend than were those who scored at the mean. Scores on PTS did not predict bereavement status.

Furthermore, SEM results provided corroborative evidence that interpersonal loss (i.e., the death of a close/not-close friend, having a close/not-close friend get injured, or having a close/not-close friend escape) uniquely predicts CG. However, perceived threat predicted both PTS and CG, which was contrary to the hypothesis that it would uniquely predict PTS. This might be due to several factors. First, it has been noted in previous research (Prigerson et al., 2007) that bereavement due to violent causes might be more likely to lead to CG than is natural bereavement; the SEM results of this study might be indicating that higher levels of perceived threat could be involved in this phenomenon. Second, it is possible that individuals who were bereaved retroactively report higher levels of perceived threat due to the fact that they did lose (or almost lost) a friend. Finally, it should be noted that one of the two items in the perceived threat construct was, "How afraid were you that someone you cared about would be seriously hurt or killed?" Therefore, although this item is part of perceived threat as currently conceptualized, it is probable that this item is more related to CG than is the item regarding fear for personal safety.

4.1 - Naming CG

The term *Complicated Grief* was only recently adopted by mental health professionals, although clinicians and researchers have been studying this disorder for several years under different names. Although there appears to be some consensus on using this name, the term is somewhat problematic: It is unclear what is meant by *complicated,* and there is no specificity about the type of grief that qualifies one for the diagnosis. Furthermore, current literature (Holland, Neimeyer, Boelen, & Prigerson, 2009) suggests that the distinction between normal and pathological bereavement-related grief is based on severity and duration rather than a

specific set of symptoms. Given that the precipitating event for developing CG is, by definition, the loss of a loved one, it would be logical to include *bereavement* in the name of the disorder. Furthermore, because the disorder tends to be chronic in nature, bringing back the term *prolonged* might be appropriate. Therefore, I would suggest calling this disorder *Prolonged Bereavement Disorder*.

4.2 - Clinical Implications and Future Directions

Although PTSD is most frequently the disorder associated with traumatic events, other disorders can be triggered by trauma. Traumatic bereavement can be a Criterion A event in leading to PTSD; however, the findings of this study demonstrate that this type of trauma also has the potential to lead to symptoms of CG—in fact, traumatic events might, in some cases, be more likely to lead to CG than to PTS. The results of this study—especially from the logistic regression—suggest that CG might be more prevalent than PTS in situations where survivors are not directly exposed to violence but are traumatically bereaved. Alternatively, it is possible that a survivor's level of exposure is moderated by bereavement such that exposure has less effect in leading to PTS in the context of bereavement. In other words, it is possible that traumatically bereaved individuals who are exposed to trauma are less distressed by their personal trauma exposure than they are by the bereavement. Future research focusing on this issue seems prudent.

The student survivors of the 4/16 shootings experienced symptoms of CG—a disorder that is not yet formally recognized—even 1 year after the tragedy occurred, suggesting that even in the absence of clinically significant PTS symptoms, many individuals might benefit from clinical attention. This suggests that clinicians working with survivors of traumatic bereavement should assess for and be ready to treat disorders other than PTSD—including CG. Indeed, there is some evidence that the clinical field is already paying attention to this area. For instance, Cognitive Processing Therapy (CPT) for PTSD has an optional module for helping patients with PTSD who experienced bereavement due to their traumatic event, ostensibly because clinicians and researchers have recognized that bereavement issues often require clinical attention (Resick, Monson, & Chard, 2010). This module and the work of Shear et al. (2005) could be the foundation for future psychotherapy interventions that target CG, whether as a comorbid disorder or in a pure clinical presentation.

It is noteworthy that this study examined PTS and CG in the context of proposed changes for *DSM–5*. The results of this study suggest that CG and PTSD remain distinct disorders even when estimating PTSD diagnoses using proposed criteria for *DSM–5* (Friedman et al., 2011b). Furthermore, these findings have implications for clinical and research efforts on traumatically bereaved individuals. Although the proposed *DSM–5* guidelines indicate that traumatic bereavement could lead to either PTSD or CG, traumatically bereaved participants in this study were more likely to endorse symptoms of CG than of PTS. Although this does not indicate that PTSD Criterion A should not include traumatic bereavement, it does suggest that clinicians should be aware of CG as a relatively common psychopathological reaction to traumatic bereavement. Additional research on the prevalence rates of PTSD and CG among a representative sample of traumatically bereaved individuals seems warranted.

4.3 - Limitations

The results of this study must be interpreted in the context of several limitations. The attrition between Wave 1 and Wave 2 was significant: Only 861 (19%) of the 4639 Wave 1 participants responded to the Wave 2 survey. Furthermore, the response rate to the Wave 1 survey was similarly low; thus, it is unlikely that the participants in this study are representative of the population of 4/16 student survivors. On the other hand, an exploration of missingness among the variables suggested that much of the missingness might have been MCAR, although missingness on K6_2 was related to K6_1 scores (see Table 11).

Because the questionnaire was designed to be administered via Internet to a large sample, clinical measures of mental disorders were not feasible. Therefore, all constructs of psychopathology are measured by using questionnaire items that estimate PTS and general anxiety/depression. Even the disorder of prime interest—CG—was measured only via questionnaire. Although the questionnaire approach made it possible to obtain a much larger sample and conduct complex analyses, the trade-off is that mental disorders could not be verified via other means (e.g., structured clinical interviews).

PTS and PTSD were particularly difficult to estimate in this study. The measure used as a foundation (TSQ) for the questionnaire items has been used in several other epidemiological studies (Galea et al., 2007; Kessler et al., 2006; Walters et al., 2007); however, the version included in the present study does not match *DSM–IV* or proposed *DSM–5* criteria for PTSD very well. Furthermore, the questionnaire items were significantly altered from their original

form, which makes it difficult to determine to what extent previous validation studies apply to this form of the items. It is noteworthy that the PTS scale used in this study did not demonstrate three factors in the EFA, even though *DSM–IV* would suggest this level of structure. Finally, the instructions for responding to the PTS2 scale at Wave 2 asked participants to endorse symptom severity for their worst month in the past year, making it impossible to designate the actual timing of the variable overall. In other words, one could determine for which month each individual was responding to the PTS2 items, but this leads to inconsistencies when performing analyses. For instance, for some participants, PTS2 is measuring the same time period as PTS1. This made it impossible to make strong causal claims when predicting PTS2.

Although the vast majority of students reported knowing someone who died, only 103 (12%) of the final sample of 825 reported losing a significant other or a close friend. This subsample was too small for reliable exploration of (a) factor structure via EFA, (b) factor structure via CFA, and (c) interaction effects among bereaved students. The sample size issue was only somewhat ameliorated by including students who had lost a not-close friend to the bereaved subsample (N = 284) and came at the cost of increasing the heterogeneity of relationship to the deceased. On the other hand, the type of bereavement (i.e., traumatic bereavement caused by violence in a school) was constant among all participants, precluding this from adding to heterogeneity.

As is often the case, the uniqueness of this sample was a strength and a limitation. The 4/16 shootings at Virginia Tech provided an opportunity to answer specific questions about students exposed to mass violence; however, this also constrains the generalizability of the findings. For example, it is unclear to what extent CG symptoms would be related to bereavement status among non-students of similar age. Future researchers could provide important clarifications by predicting and describing CG among individuals bereaved by single-victim violence and by studying CG among young adults in the general population.

Perhaps one of the more notable statistical limitations of this study was the difficulty in estimating SEM or path models with appropriate fit. This study highlight the tradeoffs inherent in choosing an estimation method: When working with data that includes dichotomous, 5-level ordinal, and continuous variables, estimation methods other than standard ML should be used. However, such alternative methods are often mutually exclusive with other statistical techniques, depending on the software used for the analyses. For instance, EQS 6.1 can analyze ordinal data

using polychoric correlations (or relationships between ordinal and continuous variables using polyserial correlations), but utilizing this technique precluded using FIML for handling missing data. Furthermore, alternative estimation methods can provide adjusted fit indices (e.g., Satorra-Bentler Scaled χ^2); however, it is not yet clear how effective such adjustments are. Finally, the deviations from multivariate normality among the data made it impossible to run power analyses for SEM models, lessening confidence in statements regarding any given pathway's lack of significance.

Finally, although this study contributes to previous literature on CG as a pathological reaction to bereavement (Holland et al., 2009; Prigerson et al., 1995b), the distinction between pathological and normal reactions to bereavement was not explored or tested in this project. Previous research (Holland et al., 2009) has indicated that the distinction between CG and normal grief following bereavement seems to be severity of symptoms, rather than the presence or absence of particular symptoms. Consistent with this, the majority of participants in the current study did not endorse significant levels of CG on the CG–21, as indicated by an overall mean score of approximately 16.80 (see Tables 10 and 11). This mean is in the lower end of the possible range of the CG–21 (0 to 84) and indicates that most participants did not experience high levels of bereavement-related grief; however, 103 participants reported scores in the estimated clinical range. Therefore, the conceptualization of CG and the use of the ICG (revised to the CG–21 for this study) are consistent with previous research; however, to test the hypothesis that CG is distinct from normal bereavement-related grief among traumatically bereaved students, additional research is needed. This could include testing the hypothesis that higher scores on the CG–21 are predictive of higher functional impairment and/or psychological distress.

4.4 - Conclusion

The results corroborate the growing research on CG (or perhaps *Prolonged Bereavement Disorder*) as a psychological reaction to bereavement, including bereavement under traumatic circumstances. The pattern of findings suggests that bereavement is a distinct stressor from trauma exposure as defined in dose-response model research; furthermore, these results corroborate the current literature, demonstrating that direct exposure (a) is difficult to define and measure and (b) is inconsistently predictive of PTS, depending on other variables that are included in the models. The potential for indirect exposure (e.g., live media coverage of

violence) to cause or exacerbate PTS among traumatically bereaved individuals remains unclear. Despite the limitations noted above, the results of this study indicate that CG can be a problem among traumatically bereaved individuals, even in the absence of significant PTS symptoms. Given these results and conclusions, and in the context of the current state of the literature on CG, it is proposed that including CG in *DSM–5* in the section of "Criteria Sets and Axes Provided for Further Study" will encourage further study and enhance clinical awareness while avoiding putting forth diagnostic criteria without sufficient research.

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Table 1 - *Criteria for CG Proposed by Horowitz et al. (1997)*

Criterion A
The individual experiences the death of a loved one at least 14 months ago.
Criterion B
The individual endorses at least 3 of the following 7 symptoms with an impairing level of severity: 1. Unbidden memories or intrusive thoughts 2. Strong spells or pangs of severe emotion 3. Distressingly strong yearnings or wishes that the deceased were there 4. Feelings of being far too much alone or personally empty 5. Excessively staying away from people, places, or activities that remind the individual of the deceased 6. Unusual levels of sleep interference 7. Loss of interest in work, social, caretaking, or recreational activities to a maladaptive degree

Table 2 - *Criteria for CG Proposed by Prigerson et al. (1999)*

Criterion A
The individual experiences the death of a loved one.
Criterion B
The individual endorses at least 3 of the following 4 symptoms as occurring at least *sometimes* (scored as a 2 on a scale of 0–4): 1. Intrusive thoughts 2. Yearning for the deceased 3. Searching for the deceased 4. Loneliness
Criterion C
The individual endorses at least 4 of the following 8 symptoms as occurring at least *often* (scored as a 3 on a scale of 0–4): 1. Purposelessness of feelings of futility about the future 2. Feeling numbness, detachment, or an absence of emotional responsiveness 3. Difficulty acknowledging the death (e.g., disbelief) 4. Feeling that life is empty or meaningless 5. Feeling that a part of oneself has died 6. Having a shattered world view 7. Assuming the symptoms or harmful behaviors of, or related to, the deceased 8. Excessive irritability, bitterness, or anger related to the death
Criterion D
Symptoms persist for at least 2 months.
Criterion E
The individual experiences significant impairment caused by symptoms.

Table 3 - *Symptom Clusters of the Inventory of Complicated Grief (Simon et al., 2011)*

Symptom Cluster 1: Yearning and preoccupation with the deceased
1. I think about this person so much that it's hard for me to do the things I normally do
4. I feel myself longing for the person who died
13. I feel that life is empty without the person who died
16. I feel that it is unfair that I should live when this person died
19. I feel lonely a great deal of the time ever since he/she died
Symptom Cluster 2: Anger and bitterness
6. I can't help feeling angry about his/her death
17. I feel bitter over this person's death
Symptom Cluster 3: Shock and disbelief
3. I feel I cannot accept the death of the person who died
7. I feel disbelief over what happened
8. I feel stunned or dazed over what happened
Symptom Cluster 4: Estrangement from others
9. Ever since he/she died it is hard for me to trust people
10. Ever since he/she died I feel like I have lost the ability to care about other people or I feel distant from people I care about
18. I feel envious of others who have not lost someone close
Symptom Cluster 5: Hallucinations of the deceased
11. I have pain in the same area of my body or have some of the same symptoms as the person who died
14. I hear the voice of the person who died speak to me
15. I see the person who died stand before me
Symptom Cluster 6: Behavior change, including avoidance or proximity seeking
2. Memories of the person who died upset me
5. I feel drawn to places and things associated with the person who died
12. I go out of my way to avoid reminders of the person who died

Note. Table adapted from Table 4 in Simon et al. (2011). Symptom numbers represent the item number as presented in the Inventory of Complicated Grief (ICG).

Table 4 - *Comparison of Symptoms of Trauma-related Diagnoses*

PTSD	MDD	CG
Intrusion	**1. Depressed mood**	1. Yearning
1. Intrusive recollections	2. Recurrent thoughts of death	**2. Intrusive thoughts**
2. Recurrent nightmares	3. Weight change	**3. Emotional numbness**
3. Dissociative reactions (e.g., flashbacks)	**4. Sleep disturbance** OR hypersomnia	**4. Feeling agitated**
4. Emotional distress to cues	5. Psychomotor agitation/retardation	5. Disbelief/difficulty accepting the death
5. Physiological reactivity to cues	6. Feelings of worthlessness/**guilt**	6. Life feels empty
Persistent Avoidance	7. Fatigue/energy loss	7. Future holds no purpose
1. Avoiding trauma-related thoughts & conversations	8. **Concentration problems**/indecisiveness	8. **Inability to trust others**
2. **Avoiding trauma-related activities & people**	9. **Lack of interest in activities**	9. Having similar symptoms as deceased
Altered Cognitions and Mood		10. Bitterness
1. Inability to recall important aspect of trauma		11. Difficulty moving on (e.g., new relationships)
2. Negative expectations about self, others, and/or the world		12. Diminished sense of self (e.g., part of self has died)
3. ***Distorted blame of self*** *or others*		13. **Avoiding reminders** of the death
4. ***Pervasive, negative emotional state*** *(e.g.,* ***guilt)***		14. Shattered worldview
5. Lack of interest in activities		
6. Feelings of estrangement		
7. Inability to experience positive emotions		
Hyperarousal and Reactivity		
1. **Irritability**/*aggression*		
2. Reckless/self-destructive behavior		
3. Hypervigilance		
4. Exaggerated startle response		
5. Concentration problems		
6. Sleep disturbance		

Note. **Bold** font indicates that the symptom is shared with one of the other disorders in the table; *italics* font denotes new symptoms (or adjustments made to old symptoms) of PTSD proposed for *DSM–5*. Clusters for PTSD symptoms are based on proposals for *DSM–5*. Information for *DSM–5* PTSD was derived from Friedman et al. (2011b). Information for CG was collected from Boelen & van den Bout (2007), Prigerson et al. (2009), and Prigerson & Maciejewski (2006).

Table 5 - *Correlations between Wave 1 Predictors and CG–21 (*n *= 760)*

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13
1. CG–21	1	.30**	.39**	.59**	.61**	.46**	.30**	.13**	.12**	.26**	.32**	.12**	.13**
2. Threat to Self		1	.34**	.31**	.30**	.20**	.23**	.32**	.09	.19**	.09	.05	.06
3. Threat to Other			1	.35**	.43**	.19**	.27**	.08	.14**	.25**	.19**	.16**	.16**
4. PTS1				1	.76**	.69**	.28**	.16**	.13**	.30**	.13**	.06	.10**
5. Grief1					1	.51**	.29**	.10**	.17**	.26**	.26**	.12**	.14**
6. K6_1						1	.14**	.08	.05	.17**	.08	.03	.06
7. Gender							1	.03	.11**	.18**	.10**	.02	.02
8. Direct Exposure								1	-.02	.11**	.05	.09	.10**
9. Media Exposure									1	-.09	-.08	-.06	-.01
10. Media Distress										1	.08	.08	.10**
11. Other Killed											1	.13**	.15**
12. Other Injured												1	.21**
13. Other Escaped													1

Note. CG–21 = Complicated Grief–21; PTS1 = Posttraumatic Stress at Wave 1; Grief1 = Grief at Wave 1; K6_1 = K6 at Wave 1.

**$p < .01$.

Table 6 - *Correlations between Wave 1 Predictors and PTS2 (*n = *824)*

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13
1. PTS2	1	.26**	.37**	.65**	.56**	.48**	.35**	.08	.06	.34**	.20**	.07	.08
2. Threat to Self		1	.35**	.32**	.32**	.21**	.22**	.30**	.10**	.18**	.19**	.06	.06
3. Threat to Other			1	.35**	.43**	.18**	.26**	.08	.13**	.26**	.21**	.161**	.17**
4. PTS1				1	.76**	.69**	.27**	.14**	.13**	.29**	.14**	.068	.10**
5. Grief1					1	.50**	.27**	.11**	.17**	.25**	.27**	.127**	.15**
6. K6_1						1	.14**	.07	.03	.18**	.09	.035	.07
7. Gender							1	.00	.08	.19**	.09	.011	.01
8. Direct Exposure								1	-.02	.10**	.08	.10**	.09**
9. Media Exposure									1	-.07	-.08	-.04	-.01
10. Media Distress										1	.07	.08	.08
11. Other Killed											1	.12**	.15**
12. Other Injured												1	.19**
13. Other Escaped													1

Note. PTS2 = Posttraumatic Stress at Wave 2; PTS1 = Posttraumatic Stress at Wave 1; Grief1 = Grief at Wave 1; K6_1 = K6 at Wave 1.

**$p < .01$.

Table 7 - *Correlations between Wave 1 Predictors and K6_2 (*n = *840)*

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13
1. K6_2	1	.14**	.17**	.41**	.31**	.51**	.14**	.05	.00	.08	.11**	.02	.07
2. Threat to Self		1	.35**	.31**	.31**	.20**	.23**	.32**	.09**	.19**	.10**	.06	.06
3. Threat to Other			1	.35**	.44**	.19**	.26**	.09	.14**	.26**	.20**	.16**	.17**
4. PTS1				1	.77**	.68**	.27**	.14**	.13**	.29**	.14**	.08	.11**
5. Grief1					1	.51**	.28**	.11**	.17**	.25**	.27**	.13**	.16**
6. K6_1						1	.14**	.07	.03	.18**	.08	.02	.08
7. Gender							1	.02	.10**	.18**	.09**	.03	.02
8. Direct Exposure								1	-.01	.10**	.07	.09**	.09**
9. Media Exposure									1	-.07	-.09**	-.06	-.01
10. Media Distress										1	.08	.08	.08
11. Other Killed											1	.13**	.16**
12. Other Injured												1	.20**
13. Other Escaped													1

Note. K6_2 = K6 at Wave 2; PTS1 = Posttraumatic Stress at Wave 1; Grief1 = Grief at Wave 1; K6_1 = K6 at Wave 1.

**$p < .01$.

Table 8 - *Descriptive Statistics and t-Tests Comparing Wave 1 Only and Wave 2 Respondents*

Variables	Wave 1 Only (n = 3778) M (SD)	Wave 2 (n = 861) M (SD)	t-values	p-values
Self-threat	2.91 (2.96)	3.03 (2.90)	1.03	.305
[a]Other threat	7.48 (2.58)	7.74 (2.40)	2.87	.004
PTS1	8.37 (8.78)	8.98 (8.63)	1.84	.066
Grief1	11.79 (8.78)	12.14 (8.69)	1.08	.279
K6_1	4.27 (4.16)	4.39 (4.24)	0.76	.447
Direct Exposure	1.29 (1.62)	1.44 (1.62)	2.55	.011
Media Exposure	3.80 (1.06)	3.74 (1.07)	-1.37	.170
Media Distress	3.68 (1.06)	3.80 (1.03)	2.84	.004

[a]Because the groups had unequal variances on Other Threat, the adjusted t- and p-values are presented for this variable.

Table 9 - *Descriptive Statistics and t-Tests Exploring Missingness on CG–21*

Variables	Missing (n = 101) M (SD)	Complete (n = 760) M (SD)	t-values	p-values
Self-threat	2.85 (2.90)	3.05 (2.91)	0.65	.516
Other threat	7.70 (2.31)	7.75 (2.42)	0.18	.858
PTS1	8.14 (8.36)	9.09 (8.66)	1.05	.296
Grief1	10.99 (8.15)	12.30 (8.75)	1.42	.155
K6_1	4.62 (4.32)	4.36 (4.23)	-0.59	.558
Direct Exposure	1.39 (1.65)	1.45 (1.61)	0.39	.698
Media Exposure	3.79 (1.01)	3.73 (1.07)	-0.51	.609
Media Distress	3.86 (1.06)	3.79 (1.03)	-0.68	.494
PTS2	15.10 (9.35)	15.46 (10.00)	0.33	.739
K6_2	4.88 (4.36)	5.04 (4.45)	0.33	.739

Note. Because of missing observations on PTS2 and K6_2, the total n's for these variables are 824 and 840, respectively.

Table 10 - *Descriptive Statistics and t-Tests Exploring Missingness on PTS2*

Variables	Missing (n = 37) M (SD)	Complete (n = 824) M (SD)	t-values	p-values
Self-threat	2.82 (2.57)	3.04 (2.92)	0.46	.645
Other threat	7.89 (2.64)	7.74 (2.41)	-0.40	.691
PTS1	7.63 (7.82)	9.04 (8.66)	0.99	.324
Grief1	11.18 (8.46)	12.19 (8.70)	0.70	.486
K6_1	3.74 (3.62)	4.42 (4.26)	0.97	.330
Direct Exposure	1.26 (1.43)	1.45 (1.62)	0.71	.479
Media Exposure	3.97 (1.00)	3.73 (1.07)	-1.38	.169
Media Distress	3.84 (1.15)	3.79 (1.02)	-0.29	.776
CG–21	17.00 (11.91)	16.80 (13.89)	-0.08	.940
K6_2	5.09 (4.69)	5.02 (4.43)	-0.08	.934

Note. Because of missing observations on CG–21 and K6_2, the total *n*'s for these variables are 760 and 840, respectively.

Table 11 - *Descriptive Statistics and t-Tests Exploring Missingness on K6_2*

Variables	Missing (n = 21) M (SD)	Complete (n = 840) M (SD)	t-values	p-values
Self-threat	2.95 (3.23)	3.03 (2.90)	0.12	.904
Other threat	7.86 (2.48)	7.74 (2.40)	-0.22	.826
PTS1	11.10 (8.33)	8.93 (8.63)	-1.14	.256
Grief1	12.57 (8.61)	12.13 (8.69)	-0.23	.820
K6_1	6.43 (4.71)	4.34 (4.22)	-2.24	.026
Direct Exposure	0.95 (1.24)	1.46 (1.62)	1.41	.158
Media Exposure	4.10 (0.89)	3.73 (1.07)	-1.54	.123
Media Distress	3.62 (1.12)	3.80 (1.03)	0.80	.426
CG–21	18.69 (14.33)	16.78 (13.81)	-0.50	.620
PTS2	16.00 (8.98)	15.41 (9.94)	-0.24	.808

Note. Because of missing observations on CG and PTS2, the total *n*'s for these variables are 760 and 824, respectively.

Table 12 - *Factor Matrix for All 28 CG Items*

Item	Loading on CG Factor
UPTHT	.711
LONG	.747
MISS	.806
NODO	.791
NOACCP	.680
AVOID	.546
DISTRES	.746
EMPTY	.772
PTDIED	.728
DRAWN	.672
ANGRY	.630
DISBEL	.707
DAZED	.662
NUMB	.741
TRUST	.599
DISTAN	.579
NOCARE	.478
NOMOVE	.751
*PAIN	.309
*HVOICE	.373
VISION	.433
UNFAIR	.649
UNPLM	.637
BITTER	.679
ENVY	.572
LONELY	.742
UPSTHT	.715
WORLOS	.625

*Variables with loadings lower than .40 will be dropped from later analyses.

Table 13 - *Eigenvalues for PTS2*

Factor	Eigenvalue
1	5.573
2	0.849
3	0.704
4	0.613
5	0.518
6	0.435
7	0.375
8	0.342
9	0.311
10	0.280

Table 14 - *Pattern Matrix for 26 CG Items and PTS2 Scale*

Item	CG Factor	PTS2 Factor
UPTHT	**.599**	.165
LONG	**.899**	-.199
MISS	**.948**	-.186
NODO	**.795**	.002
NOACCP	**.671**	.013
*AVOID	**.330**	.320
DISTRESS	**.683**	.095
EMPTY	**.862**	-.125
PTDIED	**.776**	-.064
DRAWN	**.712**	-.051
ANGRY	**.534**	.135
DISBEL	**.667**	.060
DAZED	**.546**	.164
NUMB	**.641**	.140
TRUST	**.426**	.246
*DISTAN	**.340**	.344
NOCARE	**.366**	.150
NOMOVE	**.628**	.172
VISION	**.444**	-.040
UNFAIR	**.582**	.089
UNPLM	**.494**	.202
BITTER	**.538**	.195
ENVY	**.477**	.137
LONELY	**.811**	-.095
UPSTHT	**.580**	.191
*WORLOS	**.334**	.427
PTS_fear	.062	**.711**
PTS_dream	.089	**.603**
PTS_distant	.076	**.592**
PTS_avoidtalk	-.025	**.669**
PTS_sleep	.070	**.708**
PTS_avoid	-.013	**.640**
PTS_concen	-.034	**.766**
PTS_thoughts	.078	**.712**
PTS_worry	-.055	**.791**
PTS_irritable	.017	**.740**

Note. **Bold** font is used to demonstrate which factor the item is intended to measure.
*Indicates variables that will be omitted from further analyses.

Table 15 - *Pattern Matrix for 23 CG Items, PTS2, and K6_2*

Item	CG Factor	PTS2 Factor	K6_2 Factor
UPTHT	**.619**	.193	-.066
LONG	**.916**	-.109	-.141
MISS	**.947**	-.119	-.082
NODO	**.772**	.021	.019
NOACCP	**.649**	.006	.049
DISTRESS	**.652**	.086	.077
EMPTY	**.816**	-.134	.098
PTDIED	**.746**	-.063	.059
DRAWN	**.721**	-.003	-.073
ANGRY	**.536**	.131	.011
DISBEL	**.689**	.092	-.083
DAZED	**.559**	.156	-.009
NUMB	**.568**	.063	.235
TRUST	**.392**	.156	.177
*NOCARE	**.260**	.026	.344
NOMOVE	**.571**	.129	.173
VISION	**.415**	-.030	.053
UNFAIR	**.555**	.063	.097
UNPLM	**.506**	.229	-.052
BITTER	**.524**	.178	.060
ENVY	**.432**	.081	.165
LONELY	**.767**	-.101	.100
UPSTHT	**.607**	.215	-.070
PTS_fear	.115	**.739**	-.099
PTS_dream	.099	**.613**	.004
PTS_distant	.013	**.518**	.233
PTS_avoidtalk	.004	**.675**	-.031
PTS_sleep	.075	**.730**	.007
PTS_avoid	-.003	**.619**	.032
PTS_concen	-.045	**.765**	.070
PTS_thoughts	.131	**.758**	-.121
PTS_worry	.011	**.692**	.038
PTS_irritable	-.016	**.671**	.187
NERVOUS	.028	.239	**.469**
HOPELESS	.067	-.010	**.787**
RESTLESS	.039	.241	**.525**
EFFORT	.004	.126	**.709**
DEPRESS	.072	-.043	**.697**
WORTHLESS	.001	-.114	**.840**

Note. **Bold** font is used to demonstrate which factor the item is intended to measure.
*Indicates variables that will be omitted from further analyses.

Table 16 - *Factor Matrix for 22 CG Items, Sorted in Descending Order*

Item	Loading on CG Factor
MISS	.820
NODO	.794
EMPTY	.772
LONG	.763
LONELY	.745
DISTRESS	.744
NOMOVE	.742
PTDIED	.730
NUMB	.727
UPSTHT	.716
DISBEL	.712
UPTHT	.711
NOACCP	.682
DRAWN	.677
BITTER	.677
DAZED	.661
UNFAIR	.646
ANGRY	.633
UNPLM	.631
TRUST	.577
ENVY	.567
VISION	.415

Table 17 - *Pattern Matrix for CG–21, PTS2, and K6_2*

Item	CG Factor	PTS2 Factor	K6_2 Factor
UPTHT	**.620**	.188	-.063
LONG	**.916**	-.119	-.130
MISS	**.949**	-.130	-.071
NODO	**.776**	.013	.022
NOACCP	**.654**	-.002	.054
DISTRESS	**.656**	.078	.082
EMPTY	**.818**	-.138	.097
PTDIED	**.749**	-.068	.059
DRAWN	**.718**	-.006	-.068
ANGRY	**.538**	.126	.017
DISBEL	**.692**	.083	-.073
DAZED	**.561**	.149	-.002
NUMB	**.576**	.060	.224
TRUST	**.396**	.156	.165
NOMOVE	**.576**	.127	.165
UNFAIR	**.556**	.055	.105
UNPLM	**.504**	.228	-.050
BITTER	**.526**	.174	.062
ENVY	**.438**	.077	.160
LONELY	**.769**	-.105	.100
UPSTHT	**.606**	.211	-.063
PTS_fear	.114	**.738**	-.096
PTS_dream	.094	**.616**	.005
PTS_distant	.022	**.517**	.210
PTS_avoidtalk	.005	**.674**	-.032
PTS_sleep	.071	**.733**	.006
PTS_avoid	-.004	**.622**	.026
PTS_concen	-.042	**.762**	.069
PTS_thoughts	.127	**.760**	-.120
PTS_worry	.010	**.688**	.046
PTS_irritable	-.013	**.674**	.176
NERVOUS	.034	.231	**.478**
HOPELESS	.078	-.018	**.789**
RESTLESS	.043	.235	**.530**
EFFORT	.013	.119	**.712**
DEPRESS	.082	-.050	**.700**
WORTHLESS	.011	-.120	**.838**

Note. **Bold** font is used to demonstrate which factor the item is intended to measure.

Table 18 - *Pattern Matrix for Dichotomized CG–21 Items in Two-Factor Solution*

Item	Loading on Factor 1	Loading on Factor 2
UPTHT	**.676**	-.063
LONG	.282	**.474**
MISS	.326	**.479**
NODO	.397	.253
NOACCP	**.403**	.130
DISTRESS	**.484**	.133
EMPTY	-.132	**.821**
PTDIED	.062	**.499**
DRAWN	.237	.273
ANGRY	**.683**	-.081
DISBEL	**.605**	-.020
DAZED	**.631**	-.137
NUMB	.299	.062
TRUST	.301	.121
NOMOVE	.383	.185
UNFAIR	**.549**	.064
UNPLM	**.510**	-.068
BITTER	**.650**	-.021
ENVY	.323	.111
LONELY	-.040	**.789**
UPSTHT	**.719**	-.114

Note. **Bold** font is used to demonstrate salient loadings (> .40).

Table 19 - *Factor Matrix for Dichotomized CG–21 Items in One-Factor Solution*

Item	Loading on CG Factor
UPTHT	.570
LONG	.632
MISS	.677
NODO	.563
NOACCP	.470
DISTRESS	.547
EMPTY	.514
PTDIED	.447
DRAWN	.431
ANGRY	.559
DISBEL	.537
DAZED	.470
NUMB	*.321*
TRUST	*.370*
NOMOVE	.495
UNFAIR	.552
UNPLM	.412
BITTER	.577
ENVY	.384
LONELY	.577
UPSTHT	.566

Note. *Italics* font is used to demonstrate weak loadings below the .40 cutoff.

Table 20 - *Pattern Matrix for CG–21 Items among Clinical Subsample (*n = *104)*

Item	Loading on Factor 1	Loading on Factor 2
UPTHT	-.125	.374
LONG	**.815**	-.082
MISS	**.857**	.019
NODO	**.409**	.336
NOACCP	.042	**.493**
DISTRESS	.205	.244
EMPTY	**.679**	-.009
PTDIED	**.589**	.049
DRAWN	.304	.010
ANGRY	-.090	**.613**
DISBEL	-.166	**.624**
DAZED	-.009	**.586**
NUMB	.040	.384
TRUST	.067	.272
NOMOVE	.165	.263
UNFAIR	.366	.339
UNPLM	-.258	.215
BITTER	.052	**.520**
ENVY	.063	.198
LONELY	**.616**	.030
UPSTHT	-.042	**.595**

Note. **Bold** font is used to demonstrate salient loadings (> .40).

Table 21 - *Completely Standardized Loadings for CG–21 One-Factor CFA Model*

Item	Loadings
UPTHT	.754
LONG	.784
MISS	.856
NODO	.862
NOACCP	.758
DISTRESS	.813
EMPTY	.872
PTDIED	.842
DRAWN	.751
ANGRY	.666
DISBEL	.754
DAZED	.718
NUMB	.821
TRUST	.684
NOMOVE	.832
UNFAIR	.723
UNPLM	.684
BITTER	.721
ENVY	.681
LONELY	.837
UPSTHT	.751

Table 22 - *Completely Standardized Loadings for Three-Factor CFA Model of the CG–21, PTS2, and K6_2*

Item	CG Factor	PTS2 Factor	K6_2 Factor
UPTHT	.757		
LONG	.782		
MISS	.849		
NODO	.859		
NOACCP	.760		
DISTRESS	.817		
EMPTY	.872		
PTDIED	.841		
DRAWN	.752		
ANGRY	.668		
DISBEL	.748		
DAZED	.717		
NUMB	.818		
TRUST	.689		
NOMOVE	.838		
UNFAIR	.729		
UNPLM	.685		
BITTER	.723		
ENVY	.680		
LONELY	.839		
UPSTHT	.752		
PTS_fear		.800	
PTS_dream		.726	
PTS_distant		.680	
PTS_avoidtalk		.700	
PTS_sleep		.820	
PTS_avoid		.682	
PTS_concen		.815	
PTS_thoughts		.820	
PTS_worry		.723	
PTS_irritable		.763	
NERVOUS			.673
HOPELESS			.871
RESTLESS			.716
EFFORT			.835
DEPRESS			.792
WORTHLESS			.830

Table 23 - *Final Logistic Regression Model of Bereavement Status as Predicted by Wave 2 Symptom Totals, Controlling for Previous Traumas and Wave 1 Symptoms*

Variable	*B*	Wald χ^{2a}	Odds Ratio	95% Confidence Interval for Odds Ratio	
				Lower	Upper
Predictor of Interest					
CG–21	0.06***	21.79	1.06	1.03	1.08
PTS2	0.00	0.02	1.00	0.97	1.04
K6_2	-0.06	3.23	0.95	0.88	1.01
Trauma Control Variable (Lifetime)					
Assault	-0.11	0.17	0.89	0.39	2.03
Life-threatening accident or injury	-0.07	0.05	0.93	0.41	2.12
Murder/suicide of loved one	-0.11	0.00	0.90	0.39	2.05
Accidental death of loved one	-0.59*	3.66	0.56	0.33	0.95
Witnessing someone being seriously injured or killed	0.46	0.98	1.59	0.61	4.13
Any other risk of death	0.67	1.61	1.95	0.85	4.51
Trauma Control Variable (Past Year)					
Life-threatening accident, illness, or injury	0.95*	3.05	2.58	1.00	6.63
Assault	-0.64	0.83	0.53	0.14	2.00
Life-threatening illness or injury of loved one	-0.01	0.07	0.99	0.57	1.70
Unexpected death of loved one	-0.28	1.38	0.75	0.40	1.41
Witnessing a serious accident, injury, or death	0.52	0.37	1.68	0.66	4.31
Wave 1 Symptom Scale Control					
Grief1	0.06**	6.43	1.06	1.02	1.10
PTS1	-0.03	0.31	0.97	0.93	1.02
K6_1	0.01	0.20	1.01	0.94	1.09

[a]Wald χ^2 values are from the original data with no imputed values (N = 721), because pooled estimates were not available.

*p < .05. **p < .01. ***p < .001.

Table 24 - *Regression Model of Exposure Predicted by Wave 2 Symptom Totals, Controlling for Previous Traumas and Wave 1 Symptoms*

Variable	β[a]	*t*	*p*
Predictor of Interest			
CG–21	0.10	1.85	.065
PTS2	-0.05	-1.09	.277
K6_2	-0.02	-0.30	.768
Trauma Control Variable (Lifetime)			
Assault	0.00	-0.64	.520
Life-threatening accident or injury	-0.04	-1.40	.162
Murder/suicide of loved one	0.07	1.60	.109
Accidental death of loved one	-0.03	-0.64	.524
Witnessing someone being seriously injured or killed	-0.11	-1.88	.060
Any other risk of death	0.01	0.29	.770
Trauma Control Variable (Past Year)			
Life-threatening accident, illness, or injury	0.07	1.41	.160
Assault	-0.01	0.27	.791
Life-threatening illness or injury of loved one	-0.03	-0.75	.453
Unexpected death of loved one	-0.01	-0.10	.921
Witnessing a serious accident, injury, or death	0.03	0.67	.504
Wave 1 Symptom Scale Control			
Grief1	-0.09	-0.65	.517
PTS1	0.25**	2.62	.009
K6_1	-0.06	-1.16	.246

[a]β values are from the original data with no imputed values (N = 721), because pooled estimates were not available.

**$p < .01$

Table 25 - *Regression Model of Awareness Predicted by Wave 2 Symptom Totals, Controlling for Previous Traumas and Wave 1 Symptoms*

Variable	β[a]	*t*	*p*
Predictor of Interest			
CG–21	0.06	1.20	0.231
PTS2	0.03	0.16	0.873
K6_2	-0.06	-1.02	0.306
Trauma Control Variable (Lifetime)			
Assault	0.02	0.25	0.806
Life-threatening accident or injury	-0.02	-0.10	0.919
Murder/suicide of loved one	-0.02	-0.67	0.502
Accidental death of loved one	-0.04	-0.79	0.428
Witnessing someone being seriously injured or killed	0.09*	2.21	0.027
Any other risk of death	-0.08	-1.93	0.054
Trauma Control Variable (Past Year)			
Life-threatening accident, illness, or injury	0.02	1.41	0.158
Assault	0.05	-0.01	0.993
Life-threatening illness or injury of loved one	0.17***	3.97	0.000
Unexpected death of loved one	-0.07	-1.57	0.116
Witnessing a serious accident, injury, or death	-0.04	-0.51	0.609
Wave 1 Symptom Scale Control			
Grief1	0.01	0.54	0.589
PTS1	0.08	1.20	0.231
K6_1	-0.08	-1.84	0.065

[a]β values are from the original data with no imputed values (N = 721), because pooled estimates were not available.

*p < .05 ***p < .001

Table 26 - *Logistic Regression Model of Locked-in Status Predicted by Wave 2 Symptom Totals, Controlling for Previous Traumas and Wave 1 Symptoms*

Variable	*B*	Wald χ^{2a}	Odds Ratio	95% Confidence Interval for Odds Ratio	
				Lower	Upper
Predictor of Interest					
CG–21	-0.02*	4.90	0.98	0.97	0.99
PTS2	0.01	1.60	1.01	0.99	1.03
K6_2	-0.02	0.45	0.98	0.94	1.02
Trauma Control Variable (Lifetime)					
Assault	-0.05	0.02	0.86	0.48	1.54
Life-threatening accident or injury	-0.07	0.06	1.06	0.61	1.82
Murder/suicide of loved one	0.32	1.15	1.33	0.77	2.32
Accidental death of loved one	0.06	0.08	1.07	0.74	1.55
Witnessing someone being seriously injured or killed	0.06	0.04	1.10	0.63	1.91
Any other risk of death	0.27	1.13	1.43	0.89	2.29
Trauma Control Variable (Past Year)					
Life-threatening accident, illness, or injury	-0.30	0.82	0.74	0.35	1.55
Assault	-0.11	0.26	0.89	0.36	2.22
Life-threatening illness or injury of loved one	-0.13	0.31	0.88	0.62	1.25
Unexpected death of loved one	0.29	1.91	1.33	0.88	2.01
Witnessing a serious accident, injury, or death	0.07	0.07	1.07	0.55	2.08
Wave 1 Symptom Scale Control					
Grief1	-0.02	1.51	0.98	0.96	1.01
PTS1	0.01	0.09	1.01	0.98	1.04
K6_1	0.03	2.36	1.03	0.98	1.08

[a]Wald χ^2 values are from the original data with no imputed values (N = 721), because pooled estimates were not available.

*$p < .05$.

Figure 1. Factor eigenvalues from the EFA on all 28 CG items plotted against the 95th percentile of eigenvalues from 1000 random datasets. The parallel analysis suggests that up to 3 factors could be extracted, although theoretical considerations suggest that a one-factor solution is preferred.

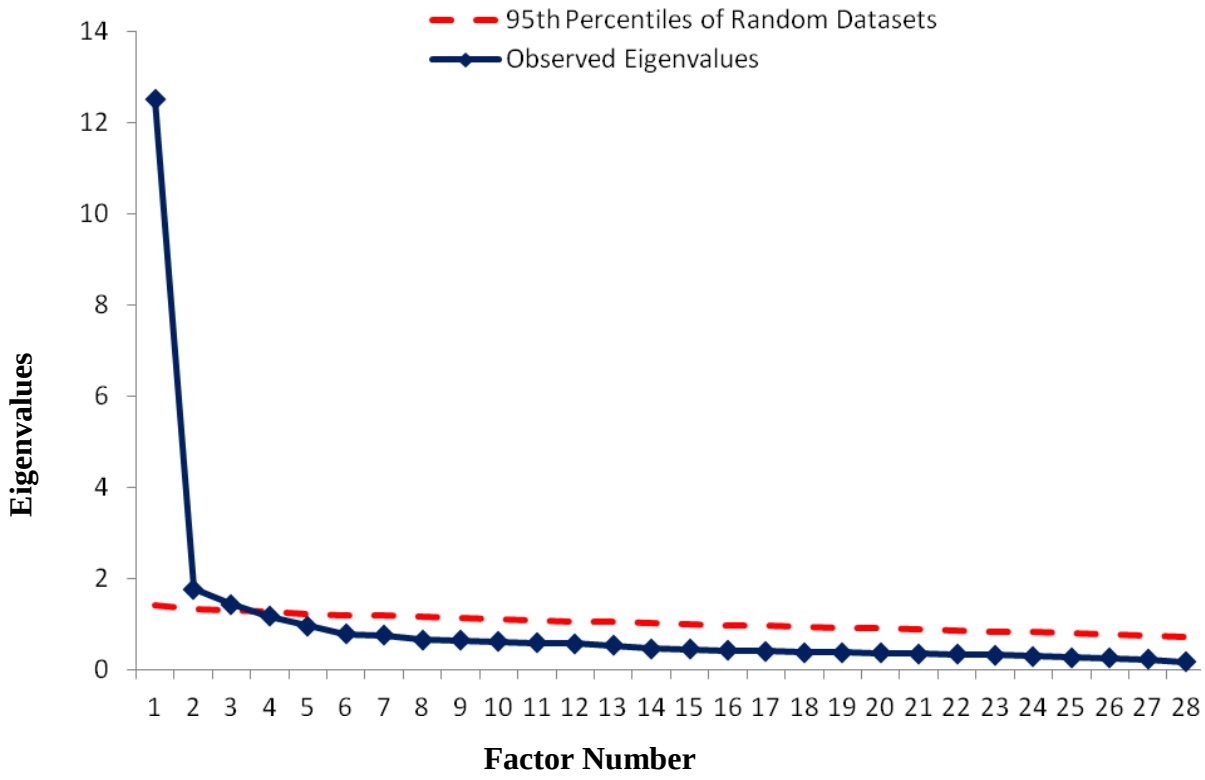


Figure 2. Factor eigenvalues from the EFA on the CG–21, PTS2, and K6_2, plotted against the 95th percentile of eigenvalues from 1000 random datasets. The parallel analysis suggests that up to 3 factors could be extracted, which is consistent with hypotheses.

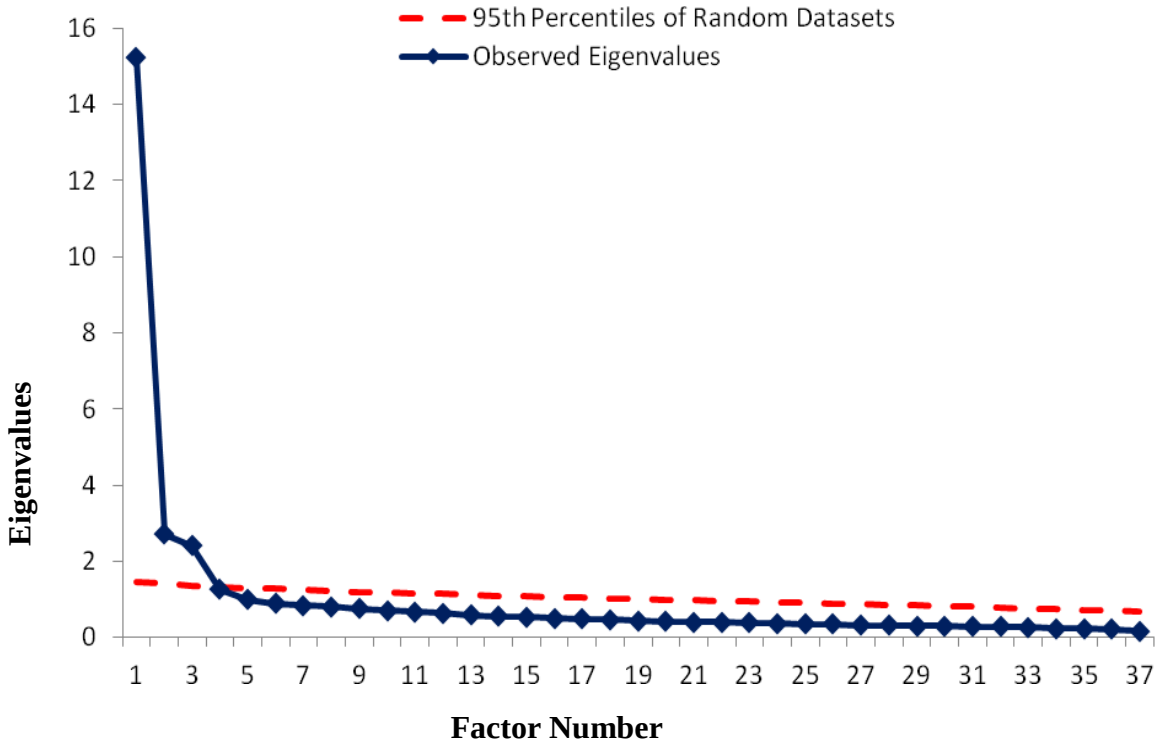


Figure 3. Factor eigenvalues from the EFA on the dichotomized CG–21 plotted against the 95th percentile of eigenvalues from 1000 random datasets. The parallel analysis suggests that up to 3 factors could be extracted; however, a one-factor solution appeared to be more stable and interpretable than three- and two-factor solutions.

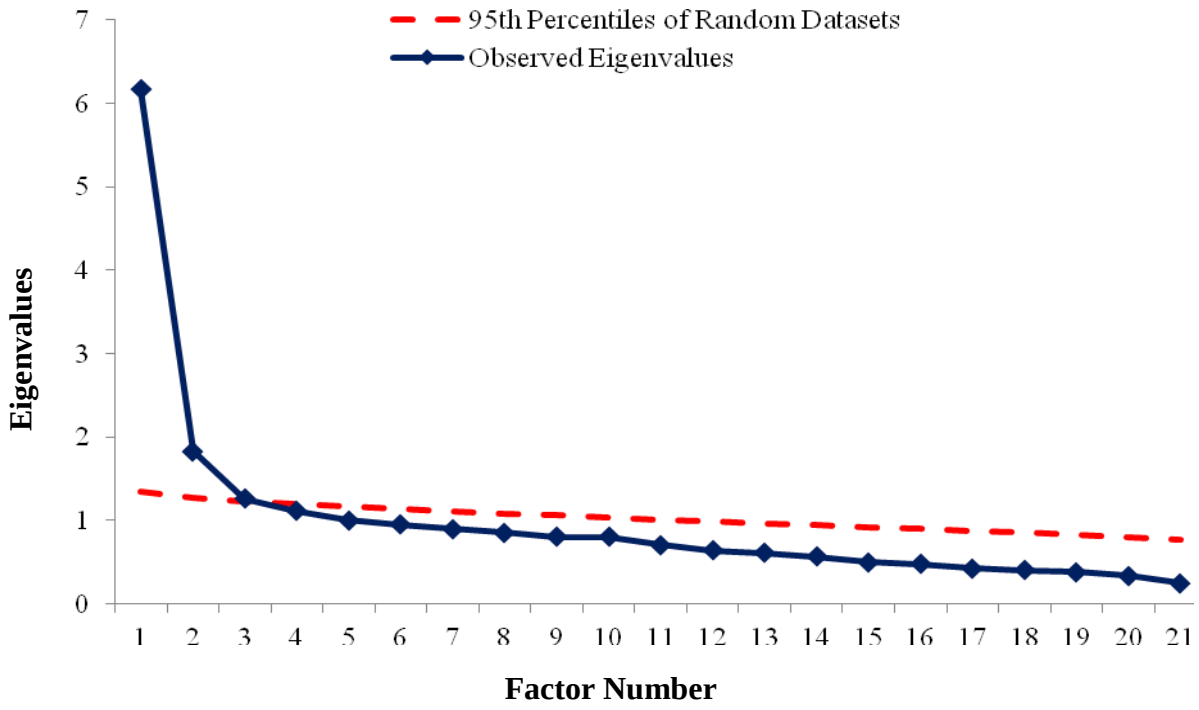


Figure 4. Factor eigenvalues of the CG–21 among the 104 high scorers plotted against the 95th percentile of eigenvalues from 1000 random datasets. The parallel analysis suggests that 2 factors are significant.

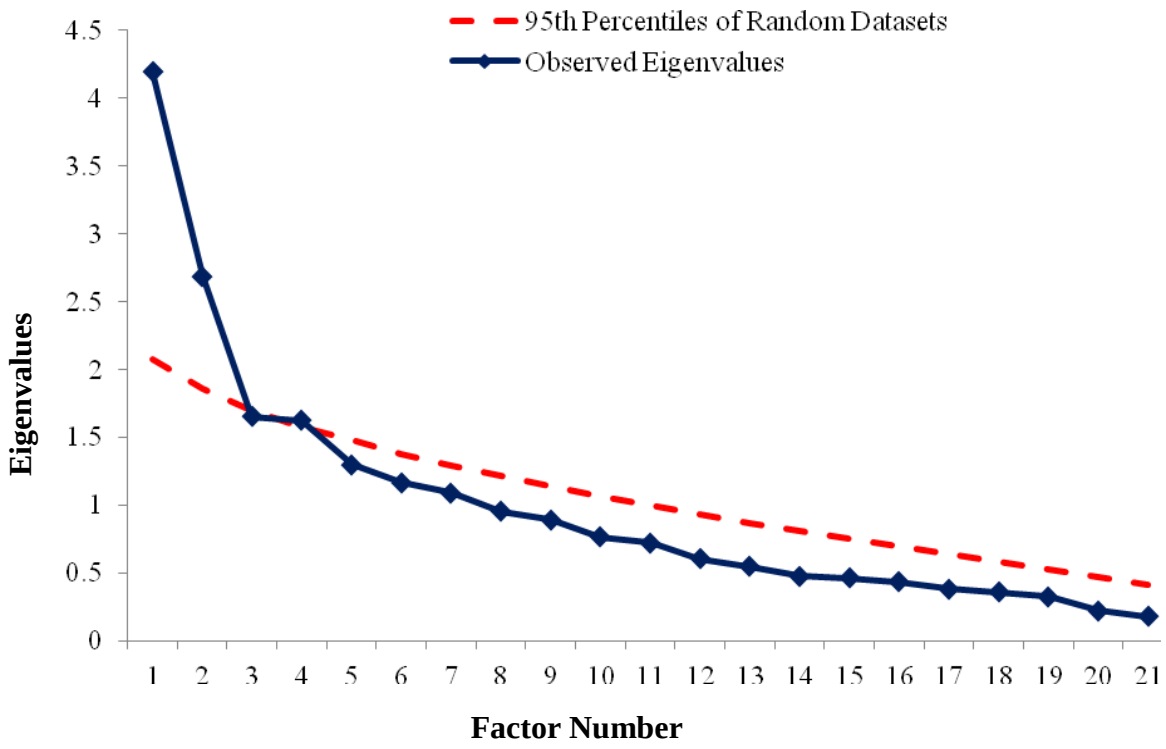


Figure 5. Hypothesized conceptual model of the relationships among exposure, perceived threat, bereavement, injury to other, escape of other from a classroom, PTS, and CG. The competing model is one in which additional pathways (represented by dashed lines) are added.

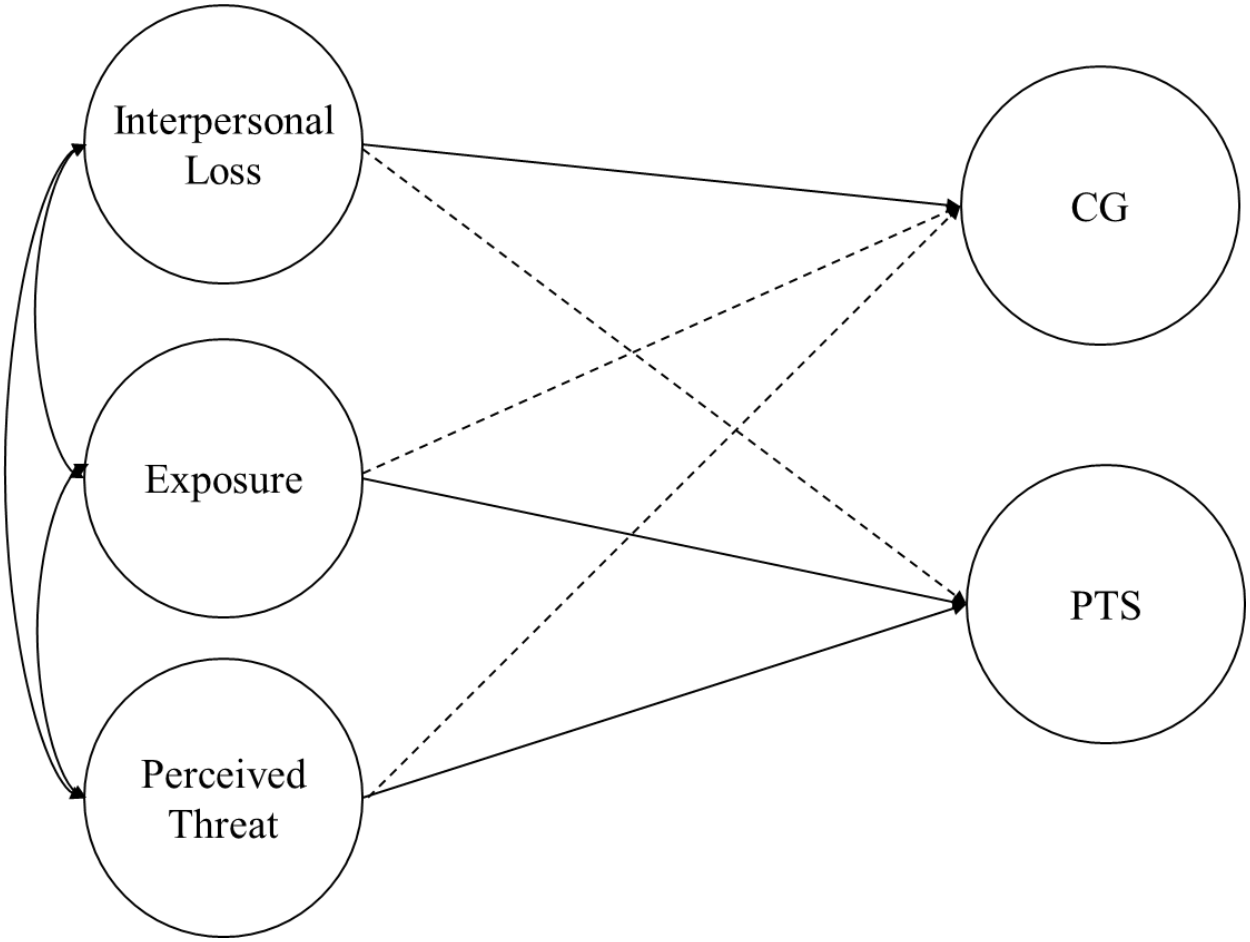
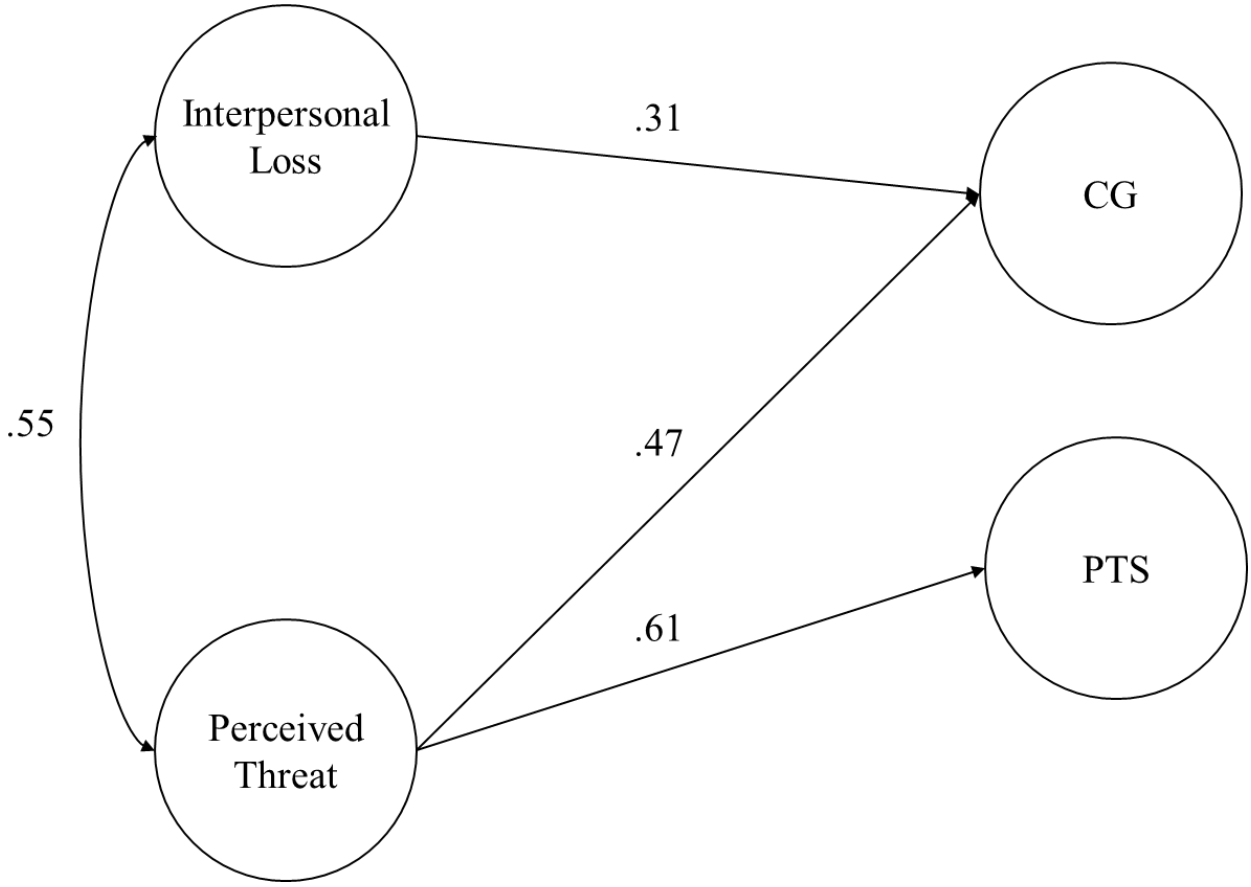


Figure 6. Accepted SEM model of the relationships among perceived threat, interpersonal loss, PTS, and CG.



Appendix A

CG Items in Wave 2 Survey

How often in the past month did . . .

1. upsetting thoughts about how they died get in the way of having good memories of them?
2. you feel yourself longing for them?
3. you miss them so much that it hurt?
4. you think about them so much it was hard to do the things you normally do?
5. you feel that you could not accept their death?
6. *you go out of your way to avoid reminders of them?
7. you have spells of feeling overwhelmed, devastated, or severely distressed by the loss?
8. you feel your life was empty without them?
9. you feel like a part of you died with them?
10. you feel drawn to places and things associated with them?
11. you feel angry about their death?
12. you have a sense of disbelief about their death?
13. you feel stunned or dazed over what happened?
14. you feel so emotionally numb that you could not take pleasure in the things you used to enjoy?
15. you find it harder than before their death to trust people?
16. *you feel more distant from people you care about than before the shootings?
17. *you feel like you lost the ability to care about people?
18. you find it very difficult to move on with life?
19. *you experience pain in the same area of your body as where they were shot?
20. *you hear their voice speaking to you?
21. *you have a vision of them standing before you?
22. you feel it was unfair that you should live when they died?
23. hearing someone talk about them bring up unpleasant memories of the way they died?
24. you feel bitter over their death?
25. you feel envious of people who never lost someone close?
26. you feel lonely without them?
27. you have upsetting thoughts about the way they died?
28. *you worry that you might suddenly lose someone else you care about?

Note. Items were scored on a 0–4 scale (0 = *never*, 1 = *rarely*, 2 = *sometimes*, 3 = *often*, and 4 = *always*).
*Item is not included in the CG–21.

Appendix B

PTS2 Items in Wave 2 Survey

For the following questions, please think about the one month since the shootings when you had the MOST SEVERE emotional reactions of this kind. This might have been the first month after the shootings or some later month.

1. During that worst month, when something reminded you of the shootings, how often did you get very upset or afraid?
2. How often during that worst month did you have dreams about April 16th, 2007 or other bad dreams?
3. How often did you feel more emotionally distant or not close to other people than usual?
4. How often during that worst month did you try not to talk about, think about, or have feelings about what happened?
5. How often did you have more trouble than usual going to sleep or waking up often during the night?
6. How often during that worst month did you try to stay away from people, places, or things that made you remember what happened?
7. How often did you have more trouble than usual concentrating or paying attention?
8. How often did upsetting thoughts, pictures, or sounds of what happened come into your mind when you did not want them?
9. How often did you worry more than usual about bad things that might happen to you or your loved ones in the future?
10. How often during that worst month did you feel more irritable or easily angered than usual?

Note. Items were scored on a 0–4 scale (0 = *never*, 1 = *1 day/week*, 2 = *2–3 days/week*, 3 = *4–5 days/week*, and 4 = *just about every day*).