Journal of Abnormal Psychology
2007, Vol. 116, No. 2, 342–351

0021-843X/07/$12.00 DOI: 10.1037/0021-843X.116.2.342

Is There More to Complicated Grief Than Depression and Posttraumatic Stress Disorder? A Test of Incremental Validity

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There is growing interest in complicated grief reactions as a possible new diagnostic category for inclusion in the *Diagnostic and Statistical Manual of Mental Disorders*. However, no research has yet shown that complicated grief has incremental validity (i.e., predicts unique variance in functioning). The authors addressed this issue in 2 studies by comparing grief, depression, and posttraumatic stress disorder (PTSD) symptoms with different measures of functioning (interviewer ratings, friend ratings, self-report, and autonomic arousal). The 1st study ($N = 73$) used longitudinal data collected at 4 and 18 months postloss, and the 2nd study ($N = 447$) used cross-sectional data collected 2.5–3.5 years postloss. With depression and PTSD controlled, grief emerged as a unique predictor of functioning, both cross-sectionally and prospectively. The findings provide convergent support for the incremental validity of complicated grief as an independent marker of bereavement-related psychopathology.

Keywords: complicated grief, depression, PTSD, loss, bereavement

What is the essential nature of complicated grief? Several decades' worth of research has shown that grief reactions tend to be multidimensional, often encompassing different types of emotions, as well as transient cognitive disorganization, health problems, or impaired role functioning (for reviews, see Bonanno & Kaltman, 1999, 2001). Although the majority of bereaved people experience these reactions to only a mild or moderate degree and return to preloss levels of functioning relatively soon after a loss (Bonanno, 2004; Bonanno, Moskowitz, Papa, & Folkman, 2005; Bonanno et al., 2002), an important minority, usually 10%–15%, go on to suffer more complicated grief reactions (Bonanno & Kaltman, 2001; Lichtenthal, Cruess, & Prigerson, 2004). There has been controversy over how to best define and measure these more complex grief reactions (Stroebe, Stroebe, Schut, & van den Bout, 2000).

In the current edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*; American Psychiatric Association, 2000), enduring grief-related reactions are diagnosed as depression, posttraumatic stress disorder (PTSD), or another anxiety disorder. Complicated grief (CG) reactions have often been evaluated as a form of depression (e.g., Bruce, Kim, Leaf, & Jacobs, 1990). When bereavement follows a violent death, such as homicide or terrorist attack, grief reactions have been conceptualized as a more involved combination of depression and PTSD (Kaltman & Bonanno, 2003; Neria et al., in press; Zisook, Chentsova-Dutton, & Shuchter, 1998).

In sharp contrast to this view, there is a growing movement among some bereavement researchers and theorists to elevate CG to a separate and unique diagnostic entity (Jacobs, Mazure, & Prigerson, 2000; Lichtenthal et al., 2004). According to this perspective, CG is a syndrome with symptoms not captured by depression or PTSD, such as prolonged and intense pining and yearning for the deceased (e.g., Jacobs et al., 2000; Lichtenthal et al., 2004; Prigerson et al., 1999). Advocates of the CG diagnosis also argue that existing treatments for either depression or PTSD are not efficacious for cases of severe lasting grief reactions (Shear, Frank, Houck, & Reynolds, 2005). Factor analytic studies have further supported that grief is separable from depression and anxiety (Boelen, van den Bout, & de Keijser, 2003; Ogrodniczuk et al., 2003; Prigerson et al., 1996).

Despite the evidence adduced thus far in support of a CG diagnostic category, a critical question remains: Does CG predict *psychological functioning* over and above that of depression and PTSD (Bonanno, 2006)? If the psychopathology of grief is entirely a corollary of existing diagnoses for PTSD and depression, then CG's impact on functioning will be collinear with PTSD and depression, which would call into question the necessity for a new diagnostic entity. Conversely, if CG uniquely predicts functioning, evidencing incremental validity, then this would bolster arguments for conceptualizing CG as a unique diagnosis (Bonanno, 2006). Although they did not present their findings in terms of incremen-

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The research described in this article was supported by grants from the National Institutes of Health (R29-MH57274 and R01-MH73595) to George A. Bonanno and from the Spunk Fund Inc. to Yuval Neria.

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tal validity, Prigerson et al. (1997) reported preliminary evidence that CG symptoms predict worsened health outcomes (blood pressure, heart trouble, and smoking) beyond the effects of depression and anxiety. To our knowledge, however, no study has yet tested whether CG makes an independent contribution to global psychological functioning beyond other forms of bereavement-related psychopathology.

The current investigation was designed to address this issue. We conducted two studies that examined CG's unique effects on psychological and psychophysiological functioning, while controlling for depression and PTSD. We were particularly interested in exploring how robust these effects might be. Accordingly, we examined the incremental validity of CG across different points in bereavement, in samples of different types and sizes, across several different measures of functioning, and using different measures of CG, depression, and PTSD.

Study 1

In the first study, we compared symptoms of CG, depression, and PTSD, measured using structured clinical interviews, with two independent sets of clinician ratings of participants' global psychological functioning, with anonymous ratings of participants' functioning provided by their close friends, and with a measure of participants' autonomic responsivity while they described their lost relationship to an interviewer.

We obtained clinician ratings of psychological functioning using the Global Assessment of Functioning (GAF) scale (Jones, Thornicroft, Coffey, & Dunn, 1995). We were particularly attentive to the need to achieve a high level of reliability for this measure. Previous studies using GAF scores have reported mixed success in achieving adequate reliability, with some studies reporting high agreement (e.g., Edson, Lavori, Tracy, & Adler, 1997) and some reporting relatively low agreement (e.g., Michels, Siebel, Freyberger, Schonell, & Dilling, 2001). However, the primary factors that appeared to promote reliability in GAF ratings across studies were that raters received training and were provided with sufficient clinical data to assess functioning (Jones et al., 1995). As we describe in detail below, we met both of these conditions in the current study.

Additionally, we obtained GAF ratings from two different sources. One set of ratings came from the interviewers who conducted the structured clinical interviews. Because the structured interviews included several different modules, interviewers were provided with a relatively broad array of information about participants' level of functioning. These ratings were obtained at both waves of assessment, thus enabling us to examine whether CG at 4 months could uniquely predict functioning at 18 months. A second set of ratings came from an independent team of clinicians who observed participants' behavior from videotapes of a completely different and more open-ended interview.

To provide a further independent measure of functioning, we also obtained anonymous ratings from participants' close friends at both waves of data collection. The friend informants rated the participant in five different domains of functioning. In previous research using this approach, the friend ratings proved to be a reliable and valid marker of functioning when averaged to a composite score (Bonanno, Moskowitz, et al., 2005; Bonanno, Rennicke, & Dekel, 2005). In the current study, we used the composite friend rating score to assess both cross-sectional and prospective prediction by CG symptoms.

Finally, we examined whether CG evidenced incremental validity in relation to participants' autonomic responsivity, measured at 4 months of bereavement during a 6-min interview segment in which they described their prior relationship with the deceased. We included the autonomic measure for two important reasons. First, as noted above, although there is preliminary evidence for the incremental validity of CG symptoms in relation to general health status (Prigerson et al., 1997), no research has yet examined CG's incremental validity in relation to physiological responses that could be directly tied to psychological events related to the loss. Second, previous evidence suggests that autonomic responsivity to the loss may be especially sensitive to different aspects of the grief reaction. There is a consistent body of research linking PTSD reactions to heightened autonomic response to cues that serve as reminders of the traumatic event (for a review, see Orr & Roth, 2000). By contrast, previous research suggests that intense grief reactions are associated with reduced autonomic reactivity to cues of the loss (e.g., Bonanno, Keltner, Holen, & Horowitz, 1995). However, no research has yet attempted to tease apart these divergent reactions in a bereaved sample. Affirmative evidence of this nature would bolster arguments that both CG and PTSD symptoms capture unique aspects of the grief response.

Method

Participants and Procedure

To recruit this sample, we made information about the study available to potential bereaved participants living in the Washington, DC, metropolitan area by sending letters describing the study to recently bereaved individuals listed in public obituaries and to individuals likely to have contact with bereaved individuals (e.g., medical and mental health professionals, clergy). Letters encouraged bereaved individuals who might be interested in joining the study to contact the researchers by phone or mail. Participants were recruited between 1997 and 2000 (for additional details, see Bonanno, Moskowitz, et al., 2005).

At approximately 4 months postloss, Wave 1 (W1), participants completed questionnaires, a structured clinical interview, and a semistructured interview. The questionnaires and structured interview were repeated at approximately 18 months postloss, Wave 2 (W2). Participants were paid $60 at each wave. Data were available at both waves of assessment for 73 participants (53 conjugally bereaved and 20 parentally bereaved), who were on average 51.7 years old ($SD = 7.7$ years), primarily female (65%), and primarily Caucasian (89%).

Structured Clinical Interview

Symptom scores. Participants were asked a series of questions corresponding to symptoms for major depressive disorder (MDD; 9 items, $\alpha = .92$) and symptoms of PTSD that did not overlap with the MDD items (14 items, $\alpha = .82$) as listed in the fourth edition of the *DSM* (*DSM–IV;* American Psychiatric Association, 1994). CG symptoms adapted from Horowitz et al. (1997) were also assessed: strong yearning for the deceased; preoccupation with thoughts about the loss; recurrent regrets or self-blame about own

behavior toward the deceased; recurrent regrets or blame regarding the behavior of others toward the deceased; difficulty accepting the finality of the loss; marked loneliness or sense of emptiness; pervasive sense that life is meaningless; and unusual difficulty developing new relationships (8 items, $\alpha = .77$).

Interviews were conducted by seven doctoral candidates in clinical psychology blind to the goals and hypotheses of the study. Interviews were videotaped, and each interviewer coded a randomly selected set of five additional interviews. Interrater reliability for the symptom items was very high (average $\kappa = .97$). The symptom data were used to create both continuous symptom scores and diagnoses of MDD and PTSD. Although a *DSM* CG diagnosis is not yet available, following Prigerson (2004) we defined a CG diagnosis as five or more CG symptoms.

GAF ratings. To increase reliability, interviewers were trained for GAF ratings. Further, the structured interviews included a collaborative task designed to provide supplementary clinical data for GAF ratings. For this task, participants described their personality characteristics and the roles and activities that define them as a person, and how these facets changed over time. At the completion of the entire interview, a 0–100 GAF score was assigned by the interviewer on the basis of criteria detailed in the *DSM–IV*. For computation of interrater reliability, GAF scores for 25 randomly selected participants were recoded from a videotape of the interview. Interrater reliability was high (intraclass correlation [ICC] = .88).

Semistructured Interview

Following a short break, physiological sensors were attached to participants, and they were instructed to sit quietly and relax for a 5-min baseline period. After the baseline, participants were administered a semistructured interview designed to elicit more spontaneous verbal and nonverbal material that could be recorded and coded at a later date. The interviewer read a script informing participants that they would be asked to speak for different periods of time about various topics: In the first topic, *relationship with the deceased* (6 min), participants described their relationship with the deceased spouse or child. In the second topic, *current life* (6 min), participants described what their life was like currently, how they were doing, and where they saw their lives going. In the third and fourth topics, participants described a *recent negative event* (2 min) and a *recent positive event* (2 min).

Autonomic activity was measured only during W1 interviews and only from the first 6-min interview topic (relationship with deceased), using an Isolated Bioelectric Amplifier System (Model CUA-07BA; SA Instrumentation, San Diego, CA). Electrocardiograph sensors were placed on the participant's side at the midabdomen, and heart rate was used as the index of autonomic arousal. Previous research (Obrist, 1981) has suggested that increased heart rate is an accurate indication of autonomic activation or active coping behaviors. A mean heart rate was calculated for the 5-min baseline and the 6-min relationship topic intervals. Heart rate change was computed by regressing each participant's average baseline heart rate on his or her average heart rate during the relationship topic; the standardized residuals were used in the analyses (Linden, Earle, Gerin, & Christenfeld, 1997). Owing to data loss and an unanticipated equipment failure, these data were available only for a subset of participants ($n = 34$). Differences between participants with and without physiological data did not approach significance for any of the variables measured in this study.

Videotapes of the entire W1 semistructured interview were available for 47 participants (64% of the sample), and these were coded by an independent team of raters using the GAF as described above. Comparison of participants whose W1 interviews were taped in their entirety with the remainder of the sample did not approach significance for any of the variables included in this study. The raters consisted of four doctoral candidates in clinical psychology and two licensed clinical psychologists. Rater reliability was high (ICC = .81).

Friend Ratings of Participants' Functioning

Each participant distributed rating materials to three close friends who they felt knew them well and with whom they had relatively consistent contact. The friend informants returned the rating materials directly to the researchers by mail. Each friend rating packet included a 10-item questionnaire that asked the informant to rate the participant's functioning in five domains: mental health, physical health, quality of social interactions, ability to accomplish goals, and coping ability. Ratings were made on a 7-point scale. The 10 items were averaged to create a composite functioning score ($\alpha = .80$). The friend informants also indicated the frequency with which they typically interacted with the participant, using a 5-point scale (1 = *less than once a month*; 3 = *about once a week*; 5 = *almost daily*).

To bolster reliability, we used a participant's friend data only if ratings from at least two friends were returned. This criterion was met for the majority of participants at W1 ($n = 59$, 81%) and W2 ($n = 44$, 61%). There were no significant demographic differences between participants with usable friend data and participants who did not have two friend ratings, with the exception that participants with two friend ratings at W2 were more likely to be Caucasian (42/44, 95.5%) than participants with one or no friend ratings at W2 (24/30, 80.0%). Correlations between ratings from different friends for the same participant were all significant and in the moderate range (rs = .25–.40).

Results and Discussion

At W1, 10% of the sample ($n = 7$) met criteria for MDD, 15% ($n = 11$) met criteria for PTSD, and 17% ($n = 13$) met criteria for CG. At W2, 12% of the sample ($n = 9$) met criteria for MDD, 10% ($n = 7$) met criteria for PTSD, and 10% ($n = 7$) met criteria for CG. Of those meeting CG criteria at W2, 5 (71%) had met CG criteria at W1. Consistent with the presumed independence of CG, 5 of 13 participants (39%) who had met CG criteria at W1 did not meet criteria for either MDD or PTSD, and 4 of 7 participants (57%) who had met CG criteria at W2 did not meet criteria for either MDD or PTSD.

Table 1 lists the means and standard deviations for symptoms (depression, PTSD, and CG), interviewer-rated GAF scores, and friend ratings of participants' functioning. Significant reductions from W1 to W2 were observed for each symptom measure (depression, PTSD, and CG). The functioning measures (GAF and interviewer's ratings) did not evidence significant change over time. A repeated measures multivariate analysis of variance to

Table 1
Means (and Standard Deviations) for Symptom and Functioning Measures at 4 and 18 Months Postloss (Study 1)

Measure	W1	W2	*t*(72)
Symptom totals from structured clinical interviews			
Depression symptoms (0–9)	1.70 (1.92)	1.31 (2.02)	2.53*
PTSD symptoms (0–14)	2.98 (2.53)	2.40 (2.64)	2.28*
CG symptoms (0–8)	2.50 (1.89)	1.46 (1.91)	5.57***
GAF from structured interview (0–100)	78.14 (9.70)	78.07 (12.73)	0.52
GAF from semistructured interview (0–100)	76.84 (11.23)		
Friend rating of participant functioning (0–7)	4.74 (0.65)	4.84 (0.75)	0.71[a]

Note. W1 = Wave 1; W2 = Wave 2; PTSD = posttraumatic stress disorder; CG = complicated grief; GAF = Global Assessment of Functioning.
[a] For this comparison, $df = 38$.
* $p < .05$. *** $p < .001$.

examine the possible influence of gender and loss type on GAF failed to reveal significant main effects or interactions involving these variables.

Regression Analyses

To examine the incremental validity of CG symptoms in relation to the various measures of functioning, we conducted a series of hierarchical regression analyses. We report the squared semipartial correlation (sr^2) for each variable, an indicator of the unique variance accounted for by the variable. In each analysis, W1 depression and PTSD symptoms were always entered on a first step. For prospective analyses predicting functioning at W2, the first step also included W1 scores for the functioning measure. CG symptoms at W1 were always entered on a second step. Finally, on a third step we considered the possible influence of loss type and its interaction with CG symptoms. However, as in each of the analyses considered in this article, the third step failed to increase the variance explained. Therefore, this step is not reported below.

The three types of psychopathological symptoms were highly intercorrelated: depression with PTSD symptoms (at W1, $r = .67$; at W2, $r = .74$); depression with CG symptoms (at W1, $r = .63$; at W2, $r = .59$); and PTSD symptoms with CG symptoms (at W1, $r = .68$; at W2, $r = .49$). For this reason, in each of the regression analyses reported below, we examined collinearity diagnostics. Following conventions suggested in the literature, we assumed problematic levels of collinearity when the condition index (CI) exceeded 20 (Belsley, Kuh, & Welsch, 1980) or when the variance inflation factor (VIF) exceeded 10 (Hair, Anderson, Tatham, & Black, 1995). In none of the analyses did the CI or VIF approach these levels. Therefore, statistically, we assumed that multicollinearity was not a serious concern.

Predicting Global Functioning (GAF)

An initial set of regressions used independent sets of clinician-rated GAF scores as the dependent variable. These analyses (summarized in Table 2) consistently supported the incremental validity of CG symptoms as predictors of both concurrent and prospective psychological functioning.

In the first analysis, the dependent variable was W1 GAF ratings from the structured clinical interviews. The first step of this analysis included only W1 depression and W1 PTSD symptoms and proved significant, $F(2, 71) = 26.04$, $p < .001$. However, forced entry of W1 CG symptoms on the second step significantly increased the GAF variance explained, $F_{change}(1, 70) = 5.93$, $p < .05$. CG symptoms uniquely explained 5% of the W1 GAF vari-

Table 2
Hierarchical Regressions Predicting GAF Scores (Study 1)

Model and variable	*B*	*SE*	β	sr^2
Predicting W1 GAF rated by structured clinical interview[a]				
Model 1				
W1 depression symptoms	−1.79**	0.63	−.35	.07
W1 PTSD symptoms	−1.40**	0.48	−.36	.07
Model 2				
W1 depression symptoms	−1.30*	0.64	−.25	.03
W1 PTSD symptoms	−0.83	0.52	−.21	.02
W1 CG symptoms	−1.59*	0.65	−.31	.05
Predicting W1 GAF rated from videotapes of semistructured interview[b]				
Model 1				
W1 depression symptoms	−2.16**	0.83	−.38	.08
W1 PTSD symptoms	−1.45**	0.62	−.34	.07
Model 2				
W1 depression symptoms	−1.53†	0.83	−.27	.04
W1 PTSD symptoms	−0.63	0.67	−.15	.01
W1 CG symptoms	−2.11*	0.85	−.38	.07
Predicting W2 GAF rated by structured clinical interview[c]				
Model 1				
W1 depression symptoms	−3.73***	0.77	−.57	.16
W1 PTSD symptoms	−0.76	0.59	−.15	.01
W1 GAF scores	0.12	0.14	.09	.01
Model 2				
W1 depression symptoms	−3.26***	0.76	−.50	.12
W1 PTSD symptoms	−0.11	0.61	−.02	.00
W1 GAF scores	0.01	0.14	.01	.01
W1 CG symptoms	−2.19**	0.79	−.33	.05

Note. GAF = Global Assessment of Functioning; W1 = Wave 1; W2 = Wave 2; PTSD = posttraumatic stress disorder; CG = complicated grief.
[a] Model 1: $F(2, 71) = 26.04$, $p < .001$; $R^2 = .42$. Model 2: $F(3, 70) = 20.54$, $p < .001$; $R^2 = .47$. [b] Model 1: $F(2, 46) = 17.33$, $p < .001$; $R^2 = .43$. Model 2: $F(3, 45) = 14.94$, $p < .001$; $R^2 = .50$. [c] Model 1: $F(3, 68) = 26.95$, $p < .001$; $R^2 = .54$. Model 2: $F(4, 67) = 24.09$, $p < .001$; $R^2 = .59$.
† $p < .10$. * $p < .05$. ** $p < .01$. *** $p < .001$.

ance, compared with 2% for PTSD symptoms and 3% for depression symptoms.

We repeated these same regression procedures using W1 GAF ratings coded independently from videotapes of the semistructured interview ($n = 48$). This analysis yielded a similar pattern of results. The initial equation that included only W1 depression and PTSD symptoms again proved significant, $F(2, 46) = 17.33$, $p < .001$. Forced entry of W1 CG symptoms on the second step again significantly increased the GAF variance explained, $F_{change}(1, 45) = 6.22$, $p < .05$. As in the previous analysis, CG symptoms uniquely explained more GAF variance (7%) than either depression (4%) or PTSD (1%) symptoms.

Of note, the same pattern of results emerged when the symptom measures were examined as predictors of GAF scores from the structured clinical interview at W2. This first step for this analysis, which included W1 depression and PTSD symptoms as well as W1 GAF scores, proved significant, $F(3, 68) = 26.95$, $p < .001$. On the second step, with W1 depression and PTSD symptoms and W1 GAF already in the equation, W1 CG symptoms significantly increased the W2 GAF variance explained, $F_{change}(1, 67) = 7.63$, $p < .01$. In this case, depression symptoms uniquely explained the most variance (12%). However, CG symptoms also uniquely accounted for a meaningful portion (5%) of W2 GAF variance.

Predicting Friend Ratings of Participants' Functioning

The next set of analyses examined whether CG evidenced incremental validity in relation to averaged ratings of functioning obtained anonymously from two of participants' close friends. These analyses used the same analytic strategy as for the GAF scores, but with the composite friend-rated functioning score as the dependent variable and friend-rated frequency of contact with the participant as an addition control variable. The results (summarized in Table 3) again supported the incremental validity of CG symptoms but only in the prospective analysis.

Using W1 friend ratings as the dependent variable, the first step, consisting of W1 depression and PTSD symptoms and friend-rated contact, proved significant, $F(3, 54) = 9.68$, $p < .001$. However, forced entry of W1 CG symptoms on the second step failed to significantly increase the W1 friend-rated variance explained, $F_{change}(1, 53) = 0.33$, $p = .56$.

Affirmative evidence for the incremental validity of CG did emerge, however, when we examined prospective prediction of W2 friend ratings. The first step of this analysis, which included W1 depression and PTSD symptoms and W1 friend ratings, proved significant, $F(4, 33) = 7.55$, $p < .001$. When W1 CG symptoms were forced into the model on the second step, this time the change proved significant, $F(1, 32) = 6.86$, $p < .001$. CG symptoms accounted for 9% of the variance in W2 friend ratings, compared with less than 1% for depression and PTSD symptoms.

Predicting Heart Rate Change

A final set of hierarchical regressions examined whether CG uniquely explained autonomic responsivity obtained at W1 while participants discussed their lost relationship. For participants with physiological data ($n = 34$), symptom scores were first regressed on the W1 baseline heart rate data and then, in a second regression, on W1 residualized heart rate change during the semistructured interview. These analyses are summarized in Table 4.

Table 3
Hierarchical Regressions Predicting Friend Ratings of Participant Functioning (Study 1)

Model and variable	*B*	*SE*	β	sr^2
Predicting W1 friend-rated functioning[a]				
Model 1				
W1 depression symptoms	−0.11*	0.05	−.35	.07
W1 PTSD symptoms	−0.06†	0.04	−.26	.04
W1 friend-rated frequency of contact	0.09	0.07	.14	.02
Model 2				
W1 depression symptoms	−0.12*	0.05	−.39	.07
W1 PTSD symptoms	−0.07†	0.04	−.31	.04
W1 friend-rated frequency of contact	0.09	0.08	.13	.02
W1 CG symptoms	0.04	0.06	.10	.01
Predicting W2 friend-rated functioning[b]				
Model 1				
W1 depression symptoms	−0.02	0.06	−.06	.01
W1 PTSD symptoms	−0.12*	0.05	−.39	.09
W1 friend-rated functioning	0.36*	0.17	.31	.07
W1 friend-rated frequency of contact	0.14	0.10	.18	.03
Model 2				
W1 depression symptoms	0.04	0.06	.11	.01
W1 PTSD symptoms	−0.05	0.05	−.18	.01
W1 friend-rated functioning	0.38*	0.16	.34	.08
W1 friend-rated frequency of contact	0.18†	0.09	.24	.05
W1 CG symptoms	−0.18*	0.07	−.45	.09

Note. W1 = Wave 1; W2 = Wave 2; PTSD = posttraumatic stress disorder; CG = complicated grief.
[a] Model 1: $F(3, 54) = 9.68$, $p < .001$; $R^2 = .35$. Model 2: $F(4, 53) = 7.25$, $p < .001$; $R^2 = .35$. [b] Model 1: $F(4, 33) = 7.55$, $p < .001$; $R^2 = .48$. Model 2: $F(5, 32) = 8.49$, $p < .001$; $R^2 = .57$.
† $p < .10$. * $p < .05$.

The first regression used baseline heart rate as the dependent measure. Inclusion of W1 depression and PTSD symptoms on the first step proved significant, $F(2, 34) = 3.45$, $p < .05$. On this step, baseline heart rate was uniquely predicted by PTSD symptoms ($\beta = .56$, $p < .05$). Inclusion of W1 CG symptoms on the second step failed to increase the R^2, $F_{change}(1, 33) = 2.49$, $p = .13$. It should be noted, however, that CG symptoms did exert a small effect, predicting 6% the baseline heart rate variance. With a larger sample, it seems plausible that the influence of CG on baseline heart rate would reach significance.

The regression for residual heart rate change when participants discussed the loss did reveal incremental prediction for CG. Inclusion of W1 depression and PTSD symptoms on the first step did not explain a significant portion of the heart rate change variance, $F(2, 30) = 0.74$. However, with depression and PTSD symptoms controlled on the second step, W1 CG symptoms entered significantly as an inverse predictor, $F_{change}(1, 29) = 4.41$, $p < .05$. With all three symptom types in the equation, heart rate change was predicted independently by PTSD ($\beta = .53$, $p = .05$) and CG symptoms ($\beta = –.55$, $p < .05$), which uniquely explained approximately the same portion of heart rate change variance (12% and 13%, respectively). A compelling aspect of these results, which we discuss in greater detail below, is that CG and PTSD symptoms exhibited opposite relations to heart rate response.

Table 4
Hierarchical Regression Predicting W1 Baseline HR and HR Change Score

Model and variable	*B*	*SE*	β	sr^2
W1 baseline HR[a]				
Model 1				
W1 depression symptoms	−1.37	0.94	−.31	.05
W1 PTSD symptoms	1.89*	0.73	.56	.17
Model 2				
W1 depression symptoms	−0.75	0.99	−1.07	.01
W1 PTSD symptoms	2.43**	0.79	.72	.22
W1 CG symptoms	−1.66	1.05	−.37	.06
W1 residualized HR change score[b]				
Model 1				
W1 depression symptoms	−0.10	0.20	−.11	.01
W1 PTSD symptoms	0.08	0.07	.28	.04
Model 2				
W1 depression symptoms	0.04	0.09	.09	.01
W1 PTSD symptoms	0.14*	0.07	.53	.12
W1 CG symptoms	−0.21*	0.10	−.55	.13

Note. W1 = Wave 1; HR = heart rate; PTSD = posttraumatic stress disorder; CG = complicated grief.
[a] Model 1: $F(2, 34) = 3.45$, $p < .05$; $R^2 = .17$. Model 2: $F(3, 34) = 3.23$, $p < .05$; $R^2 = .23$. [b] Model 1: $F(2, 30) = 0.73$, $p = .48$; $R^2 = .05$. Model 2: $F(3, 29) = 2.01$, $p = .13$; $R^2 = .17$.
* $p < .05$. ** $p < .01$.

Study 2

The use of multiple, independent markers of functioning was a clear strength of Study 1. However, a potentially important limitation of the study was its relatively small sample size. Small samples are susceptible to possible sample biases. An additional concern of particular importance for the study of CG is that a small sample means that relatively few participants will evidence the type of extreme psychopathology suggested by the construct.

In Study 2, we sought to replicate and extend the findings of Study 1 using a larger and potentially more severely distressed sample. Specifically, in Study 2 we sought further evidence for the incremental validity of CG in a sample of individuals who had lost loved ones between 2.5 and 3.5 years earlier, in the September 11, 2001, terrorist attacks (described in Neria et al., in press). This sample provided a particularly demanding context in which to test the incremental validity of CG. As noted earlier, losses due to violent death often evoke the most severe grief reactions and typically include both chronic depressive symptoms and elevated PTSD symptoms (Bonanno & Kaltman, 1999; Kaltman & Bonanno, 2003; Zisook et al., 1998). Not surprisingly, people who lost loved ones in the September 11 attacks also tend to show elevated depression and PTSD (Bonanno, Galea, Bucciarelli, & Vlahov, 2006; Neria et al., 2006). Such high levels of depression and PTSD, however, suggest that there may be relatively less unexplained variance in functioning that might be accounted for by CG. Additionally, because recruitment for Study 2 began several years after September 11, using Web sites devoted to those who lost loved ones in the attack, we anticipated that this pool of potential participants would tend toward more severely distressed individuals. Finally, to further explore the generalizability of findings from Study 1, in Study 2 we used different measures of depression, PTSD, and CG, as well as a different and more elaborate measure of functioning.

Method

Participants and Procedure

The study was conducted using a Web-based, secured and encrypted survey (for similar methods, see Silver, Holman, McIntosh, Poulin, & Gil-Rivas, 2002). Recruitment of individuals who had lost family members in the September 11 terrorist attack was initiated on March 14, 2004, and completed on February 5, 2005, using an online invitation placed on Web sites of relevant family organizations (e.g., Families of September 11; Voices of September 11th) or sent directly to the members of such organizations. More detail on the recruitment is provided elsewhere (Neria et al., in press).

Complete data on depression, PTSD, CG, and functioning were available for 447 participants. Of these participants, 346 (77.4%) were female. The mean age was 45 ($SD = 11.5$) years. A majority of participants reported an annual family income above $40,000 (83%), were Caucasian (94%), were married (55%), and had completed at least 4 years of college (68%).

Measures

Major depression was measured using the Patient Health Questionnaire (Spitzer et al., 1994), which assesses current symptoms of *DSM–IV* major depression by self-report ($\alpha = .88$). Respondents indicate the extent to which they experience each symptom on a 0–4 scale. Responses of 2 (*more than half the days*) or 3 (*nearly every day*) count toward an MDD diagnosis. The Patient Health Questionnaire has shown good agreement with independent ratings of depression provided by mental health professionals (Spitzer, Kroenke, & Williams, 1999).

PTSD related to September 11 was measured using the PTSD Checklist—Civilian Version (Weathers, Litz, Herman, Huska, & Keane, 1993). This measure is a well-validated screening instrument for PTSD (Blanchard, Jones-Alexander, Buckley, & Forneris, 1996) and consists of 17 items corresponding to *DSM–IV* PTSD symptoms ($\alpha = .92$). Possible scores for each item range from 1 (*not at all*) to 5 (*extremely*). Following recent studies (Hoge et al., 2004; Schlenger et al., 2002), we adopted a conservative cut-point of 50 for PTSD.

CG reactions were assessed using a nine-item screening measure (Prigerson, 2004; Prigerson & Jacobs, 2001) for the following symptoms: yearning; preoccupation with the deceased; trouble accepting the loss; detachment; bitterness; loneliness; feeling that life is empty; feeling that part of one's self has died; and loss of security/safety. Respondents indicate the frequency of these experiences in the past month on scale ranging from 1 (*almost never*) to 5 (*always*). Internal consistency in this study was adequate ($\alpha = .86$). Following Prigerson (2004), we assigned a CG diagnosis when participants reported at least five symptoms at a level of *often* or *always*.

Global functioning was measured using a 12-item self-report scale, the Medical Outcome Study Short Form Health Survey (SF-12; Ware, Kosinski, & Keller, 1996). The SF-12 measures

eight domains of health status: physical functioning, role limitations due to health problems, bodily pain, general health, vitality, social functioning, role limitations due to emotional problems, and mental health. These items produce a weighted average composite score ($\alpha = .72$). The instrument has shown adequate test–retest reliability (.76; Ware et al., 1996) and construct validity (e.g., Jenkinson, Chandola, Coulter, & Bruster, 2001).

Results and Discussion

As anticipated given the nature of the sample, there was considerable psychopathology: In all, 15.4% of the sample ($n = 69$) met criteria for MDD, 17.2% ($n = 77$) met criteria for PTSD, and 39.8% ($n = 178$) met criteria for CG. Similar to Study 1 and consistent with the hypothesized independence of CG, over half of the participants meeting CG criteria ($n = 98$, 55%) did not meet criteria for either MDD or PTSD.

Means and standard deviations for symptom levels (depression, PTSD, and CG) and the SF-12 score representing global functioning are listed in Table 5. As in Study 1, the three types of psychopathological symptoms were highly intercorrelated: depression with PTSD ($r = .76$) and CG ($r = .49$) and PTSD with CG ($r = .66$). However, neither the CI nor VIF statistic in the analyses below approached problematic levels (see Study 1). Therefore, we assumed multicollinearity was not a serious concern.

To examine the relationships between CG symptoms and functioning, we used the same analytic strategy as in the previous study, with the SF-12 summary score as the dependent variable. This analysis is summarized in Table 6. The initial equation that included only depression and PTSD symptoms proved significant, $F(2, 444) = 80.35$, $p < .001$. Depression was a significant inverse predictor of functioning, but PTSD symptoms did not enter significantly into the model. Forced entry of CG symptoms on the second step (Model 2) significantly increased the SF-12 variance explained, $F_{\text{change}}(1, 443) = 5.84$, $p < .05$. In the final model, both depression and CG symptoms independently predicted reduced functioning. However, depression symptoms uniquely accounted for more variance in self-reported functioning (5%) than did CG symptoms (2%).

General Discussion

The validity of a possible new diagnostic category for CG has been the subject of considerable research and debate (Jacobs et al., 2000; Lichtenthal et al., 2004). In support of the growing interest in CG as a possible new diagnostic entity, previous research has demonstrated that CG symptoms are statistically independent from anxiety and depression (e.g., Boelen et al., 2003). However, apart from one study showing that CG symptoms uniquely predicted worsened health outcomes (Prigerson et al., 1997), no research has yet demonstrated the incremental validity of CG symptoms.

The current investigation provided consistent evidence that CG symptoms reliably predicted functioning over and above depression and PTSD. An important strength of these findings is their consistency across two studies using multiple measures of psychopathology and functioning as well as samples of different type, size, and temporal location in the course of bereavement. The sample for Study 1 was relatively small but exhibited levels of psychopathology typically observed in community samples (Bonanno & Kaltman, 2001). These data were collected longitudinally, however, and showed that 4-month CG symptoms were incrementally and prospectively predictive of both clinician ratings and friend ratings of participants' functioning obtained more than 1 year later. In Study 2 we sought a sample that was considerably larger and had more extreme psychopathology. Although this sample in turn was less representative of the normal grief course and the study was limited by its exclusive reliance on self-report instruments, again CG symptoms evidenced incremental prediction of functioning.

A particularly compelling finding was that CG symptoms showed incremental prediction of cardiovascular responsivity while participants discussed their lost relationship. Although researchers have examined the effects of bereavement on immune functioning, stress response, and brain activity (e.g., Gerra et al., 2003; Gundel et al., 2003; Irwin, Daniels, & Weiner, 1987), there has been minimal exploration of peripheral physiological responses, despite evidence that suggests their relevance to outcome (e.g., Bonanno et al., 1995). Our findings lend further support for the value of a multimethod measurement approach that includes psychophysiological measures (Tomarken, 1995), which may capture aspects of bereavement outcome not accessible by other means.

One aspect of these findings that merits additional comment concerns the opposite effects exerted by CG and PTSD symptoms on heart rate response. Consistent with previous studies, PTSD symptoms were associated with basal heart rate (for a review, see Buckley & Kaloupek, 2001). Also consistent with previous re-

Table 5
Means (and Standard Deviations) for Symptom and Functioning Measures at 2.5–3.5 Years Postloss (Study 2)

Measure	*M* (*SD*)
Depression symptoms (0–36)	17.0 (6.1)
PTSD symptoms (0–85)	37.3 (12.9)
CG symptoms (0–55)	28.3 (7.4)
SF-12 Global Functioning (0–69)	52.3 (9.1)

Note. PTSD = posttraumatic stress disorder; CG = complicated grief; SF-12 = Medical Outcome Study Short Form Health Survey.

Table 6
Hierarchical Regression Models for SF-12 Scores for Continuous Symptom Variables (Study 2)

Model and variable	*B*	*SE*	β	sr^2
Model 1				
Major depression symptoms	−0.51***	0.10	−.34	.05
PTSD symptoms	0.02	0.05	.02	.00
Model 2				
Major depression symptoms	−0.51***	0.10	−.34	.05
PTSD symptoms	−0.02	0.06	−.03	.00
CG symptoms	0.10*	0.07	.08	.02

Note. SF-12 = Medical Outcome Study Short Form Health Survey; PTSD = posttraumatic stress disorder; CG = complicated grief. Model 1: $F(2, 444) = 80.35$, $p < .001$; $R^2 = .26$. Model 2: $F(3, 443) = 56.10$, $p < .001$; $R^2 = .28$.
* $p < .05$. *** $p < .001$.

search (e.g., Orr & Roth, 2000), PTSD symptoms predicted increased heart rate when participants were interviewed about the lost relationship. It should be noted that this finding was weaker than is generally observed in trauma research (i.e., PTSD was a significant predictor of heart rate change only when CG symptoms were included in the model) and may reflect a broader phenomenological difference between bereavement, which is sometimes drawn out over many months, and the more focal nature of acute traumatic events. In contrast to the PTSD effects, however, CG symptoms were only mildly and nonsignificantly associated with baseline heart rate and significantly associated with decreased heart rate when participants talked about the loss.

The most parsimonious explanation for this lessened cardiovascular reactivity is the commonly observed pattern of "environmental rejection" following loss (Dawson, Schell, & Catania, 1977). Animal studies have shown, for example, that infants separated from their mother exhibit an initial spike in arousal followed by a similar prolonged pattern of cardiovascular inhibition (Hofer, 1973; Reite & Snyder, 1982). The same type of autonomic pattern appears to characterize human separation responses (Field & Reite, 1984; Hollenbeck et al., 1980). The pairing of this response with CG symptoms, which indicate acute separation distress and preoccupation with the loss, suggests that the absence of cardiovascular responsivity may be best understood as a consequence of emotional withdrawal (Bonanno et al., 1995; Fowles, 1980).

Another possible explanation for this pattern is that more acute grief at 4 months of bereavement may be associated with a stronger need for self-disclosure and a corresponding reduction in autonomic activity and distress at the opportunity to do so (Lepore, Ragan, & Jones, 2000). By contrast, PTSD symptoms covary with heart rate arguably because heart rate change reflects negative affect and defensive behavior triggered by the requirement to talk about the loss (Elsesser, Sartory, & Tackenberg, 2004). Although this issue cannot be resolved in this study, at a minimum the divergent effects of CG and PTSD provide further support for the independence of the constructs.

Despite the strengths detailed above, several limitations must also be acknowledged. One consideration is that although the current investigation is relevant to the ongoing debate about the potential usefulness of CG as a new diagnostic category, we did not directly address diagnostic issues. The data from this investigation affirm that CG symptoms incrementally predict functioning and that many participants met criteria for a categorical CG variable independent of MDD or PTSD. However, the question of how CG symptoms might be organized diagnostically is still very much open to debate. It will be useful for future research to examine the validity of any specifically proposed CG diagnosis using an approach similar to that developed in the current investigation.

A related issue pertains to the high level of correlation between depression, PTSD, and CG symptoms, which raises concern about the clinical significance of the findings. This concern is mitigated by factor analytic studies attesting to the independence of CG symptoms (e.g., Boelen et al., 2003) and in the current investigation by findings that many participants met criteria for the CG diagnosis but not for MDD or PTSD. Nonetheless, it must be acknowledged that the clinical significance of CG symptoms has yet to be established. Although in the current studies the total variance explained in clinician ratings of functioning was always near 50% and CG symptoms typically accounted for more variance in functioning than other types of symptoms, the high correlation between symptom measures suggests that in actual clinical settings it may be difficult to distinguish CG from depression or PTSD symptoms. This may be particularly true in cases where the loss was a result of graphic or violent death. Previous research has indicated that such losses increase the likelihood of both chronic depression and elevated PTSD (Bonanno & Kaltman, 1999) and perhaps decrease the relevance of CG symptoms. And in Study 2 of the current investigation, where the sample had all lost loved ones in the September 11 terrorist attack, the proportion of variance explained by CG was smaller. Further empirical study will be needed to explore this issue.

There is also the potential problem of conceptual overlap in comparing measures of psychological symptoms with global measures of functioning (i.e., healthy functioning is defined in part by the absence of psychopathology). It is important to note, however, that other factors, in addition to the absence of symptoms, contribute to the determination of functioning. In Study 1, we strove to highlight this distinction even further by including additional collaborative tasks as part of the structured interviews and by including GAF ratings coded from a separate, less structured interview and global ratings of participants' functioning provided by their close friends. Despite these caveats, it will be important for further research to continue to examine the incremental validity of CG in relation to other measures of functioning, such as longitudinal performance measures or ratings of functioning provided by different informants (e.g., employers).

In sum, the present findings lend support to CG's dimensionality as a construct separate from depression and PTSD and thus bear relevance to the debate over the appropriateness of creating a separate and unique diagnosis for CG. Although the present findings are in accord with the claims of theorists and researchers who support the creation of a CG diagnosis, it should be emphasized that a full treatment of the merits of a CG diagnosis is beyond the scope of this article. Nevertheless, the present findings demonstrate CG's relevance to adjustment and suggest that specific components of distress related to CG symptoms are appropriate targets for clinical intervention (Mancini & Bonanno, 2006). Efforts are under way to refine a targeted intervention for CG, and in contrast to the overall lack of efficacy usually observed for grief therapies (Jordan & Neimeyer, 2003; Neimeyer, 2000), initial results are promising (Shear et al., 2005).

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Received July 25, 2005
Revision received December 21, 2006
Accepted January 2, 2007 ■