Grief in Children and Adolescents Bereaved by Sudden Parental Death

Nadine M. Melhem, PhD; Giovanna Porta, MS; Wael Shamseddeen, MD; Monica Walker Payne, MA; David A. Brent, MD

Context: Major advances have been made in our understanding of the phenomenology and course of grief in adults. However, little is known about the course of grief in children and adolescents.

Objective: We report on the course of children’s and adolescents’ grief reactions after sudden parental death and the effect of those reactions on subsequent psychiatric and functional status.

Design: Longitudinal study (July 1, 2002, through January 16, 2007) of bereaved children, adolescents, and families, with yearly comprehensive assessments as long as 3 years after parental death.

Setting: Bereaved children and adolescents and their surviving parents recruited through coroners’ records and a newspaper advertisement.

Participants: A total of 182 parentally bereaved children and adolescents aged 7 through 18 years whose parent died due to suicide, unintentional injury, or sudden natural causes.

Main Outcome Measures: Grief, functional impairment, and incident depression.

Results: Three distinct trajectories of grief reactions were observed in the study participants. In 1 group, which consisted of 10.4% of the sample, grief reactions showed no change 33 months after death. Children and adolescents with prolonged grief reactions had higher rates of previous personal history of depression. Prolonged grief made unique contributions to increased levels of functional impairment, even after controlling for the clinical characteristics before and after the death. Conversely, prolonged grief in children, adolescents, and the surviving caregiver predisposed children and adolescents to an increased hazard of incident depression. Another group (30.8%) showed increased grief reactions 9 months after the death, which gradually decreased over time. Despite this finding, grief reactions in this group also were associated with functional impairment and increased risk of incident depression.

Conclusions: Grief reactions abate over time for most children and adolescents bereaved by sudden parental death; however, a subset shows increased or prolonged grief reactions, which in turn increases the risk of functional impairment and depression. Research regarding interventions designed to relieve the burden of grief in bereaved children and adolescents is needed. Such efforts also should assess and address grief reactions in the surviving parent.

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The death of a parent is consistently rated as one of the most stressful life events that a child or adolescent can experience. In Western countries, 4% of children and adolescents experience the death of a parent, and approximately 1 in 20 children and adolescents in the United States experience such a loss before age 18 years. Major advances have been made in our understanding of the phenomenology and course of grief in adults. We define grief as the subjective experience of loss, but bereavement refers to status with respect to loss, regardless of subjective experience. Although less is known about grief reactions in children and adolescents, preliminary evidence from cross-sectional studies suggests that, similar to adults, some bereaved children and adolescents experience a complicated or prolonged grief reaction (PGR). Reflecting the current terms used in the literature, we use the terms complicated and prolonged interchangeably.

Relatively little is known about the course of grief in children and adolescents. In our ongoing, longitudinal study of the impact of sudden parental death on children and adolescents, exposure to parental death was found to result in an increased risk of depression and posttraumatic stress disorder (PTSD) during the first year and persisting into the second year after the death. Herein, we report on the course of parentally bereaved children’s and adolescents’ grief reac-
tions and their effect on subsequent psychiatric and functional status. We hypothesize that, during the first 3 years after parental death, a subset of parentally bereaved children and adolescents will have a PGR consistent with adult complicated grief, which can be predicted by a prior personal or family history of psychiatric disorders. Conversely, those bereaved children and adolescents with a PGR will have functional impairment beyond that attributed to other psychiatric disorders, as well as increased incidence and earlier onset of depression.

**STUDY SAMPLE**

The sample is from the Impact of Parental Death Study on Children and Families, a study from which several reports have been published previously. 35-37,38 Bereaved children and adolescents were interviewed at 3 points in time: at baseline, which occurred a mean (SD) of 8.5 (3.7) months after the death; then approximately 1 year later, which was 21.4 (4.2) months after the death; and then approximately 2 years later, which was 33.2 (5.7) months after the death. The sample consisted of 182 children and adolescents younger than 18 years at baseline. The sample was 54.4% male, with a mean (SD) age of 12.4 (2.8) years. Most children and adolescents (91.8%) were biologically related to the proband (ie, the deceased parent). Of these, 165 (90.7%) were followed up a year later and 141 (77.5%) were followed up 2 years later. We also interviewed the surviving parents, who were mostly women (89.0%) and a biological parent of the offspring (88.4%).

We compared cohort individuals lost to follow-up with those retained in the study on demographic and clinical characteristics at baseline (no. of variables tested = 24; corrected α = .002). No differences were found in grief reactions, our main outcome, as measured by the Inventory of Complicated Grief—Revised for Children (ICG-RC). 35,39 between cohort individuals retained and those lost to follow-up (mean [SD], 55.5 [19.7] vs 53.7 [18.6]; t = 0.33; df = 152; P = .74). Cohort individuals lost to follow-up had higher scores on the negative coping frequency scale (mean [SD], 5.2 [0.6] vs 4.1 [1.6]; t = -4.49; df = 17.65; P < .001) and were more likely to blame others for the death (66.7% vs 24.4%, Fisher exact test, P = .001) compared with those retained.

**RECRUITMENT**

Deceased probands were aged 30 through 60 years and had children or adolescents aged 7 to 18 years. Their deaths had occurred within 24 hours as a result of suicide (n = 42), unintentional injury (n = 31), or sudden natural causes (n = 51). Bereaved families were recruited via coroners' reports (49.7%) and responses to a newspaper advertisement (50.3%). Details regarding the recruitment and representative nature of the sample were published previously. 35,37,38 In brief, monthly lists of deaths by suicide, unintentional injury, or sudden natural causes, occurring between July 1, 2002, and January 16, 2007, were obtained from the coroner's offices of Allegheny County and neighboring counties after obtaining approval from the Institutional Review Board at the University of Pittsburgh. Suicides are those determined as definite suicidal deaths by the coroner; all undetermined or ambiguous deaths were excluded (such as death by falling and death by firearms that was thought to be unintentional). Only unintentional and natural events that resulted in sudden death are included. Unintentional events in which multiple family members died or were seriously injured also were excluded. The causes of sudden natural death included in this sample were myocardial infarction (n = 21), other heart conditions (n = 18), infections (n = 1), and others (n = 11) (eg, cancer, diabetes mellitus, stroke, aneurysm, or complications from gastric bypass surgery). The unintentional deaths consisted of 11 drug overdoses, 8 motor vehicle crashes, and 12 other causes (such as drowning or exposure to cold). Drug overdoses were carefully reviewed to rule out those with possible suicidal intent. Probands who died of drug overdoses had no previous history of suicide attempts compared with those who completed suicide (0% vs 35.7%, Fisher exact test, df = 1, P = .02). A letter was sent from the coroner's office to the next of kin of 1773 deceased probands, of whom we were able to reach 1638 families (92.4%). The letter included a description of the study and a stamped refusal card. If no refusal card was received within 2 weeks, the interviewers called the next of kin to check his or her eligibility and to attempt to enlist his or her participation. Of those contacted, 16.2% sent back the refusal card, 52.5% were not eligible because the deceased was single and had no offspring or had none in the eligible age range, and 23.7% were determined to be eligible. The eligibility rate was similar across types of death, and 71.0% of eligible individuals participated. We also expanded our efforts to include other means, such as radio and additional newspaper advertisements and distribution of posters and brochures to physicians, hospitals, funeral directors, community mental health facilities, and other service providers. A psychological autopsy of the proband was conducted with the next of kin, often a spouse or parent of the deceased, as the informant. Demographic and clinical characteristics (No. of variables tested = 12; corrected α = .004) of probands whose families were recruited through the coroner's office were compared with those whose families were recruited via advertisements and were found to be similar except for higher rates of alcohol or substance abuse disorders (71.3% vs 30.0%, χ² = 8.09, P = .004) in the probands whose data were obtained through the coroner's office. A higher proportion of probands who had experienced sudden natural death (70.0%) were discovered via response to the advertisement by their families because individuals who die of sudden natural causes frequently do not come to the attention of the coroner. Finally, the demographic characteristics of those who died of suicide and unintentional injuries were similar to those who died of those causes in Allegheny County (ie, metropolitan Pittsburgh, Pennsylvania). 40

**ASSESSMENT**

Our modified version of the adult Inventory of Complicated Grief (ICG). 16 the ICG-RC, was used to assess grief phenomenology in children and adolescents younger than 18 years. The psychometric properties of this version have been examined previously. 39 The ICG-RC has high internal consistency as measured by a Cronbach α of 0.95 and shows evidence of convergent and discriminant validity in relation to self-reported measures of depression, anxiety, PTSD, and functional impairment. For the surviving parent, the 19-item ICG was used. We use a previously established cutoff of 25 or greater, which identifies adults with complicated grief with significantly worse general, mental, and physical health and social functioning. 39 The Circumstances of Exposure to Death semistructured interview was used to assess the children's and adolescents' experience surrounding and after the death of their parent. 41 Past (ie, prior and at time of death) and current psychiatric disorders in children and adolescents younger than 18 years were assessed using the Schedule for Affective Disorders and Schizophrenia for School-Aged Children, Present (K-SADS-PL). 42 For children who turned 18 years old during the study and for surviving parents, we administered the Structured Clinical Interview for DSM-IV Axis I and II Disor-
Table 1. ICG-RC Scores at Baseline and Follow-up by ICG-RC Classa

<table>
<thead>
<tr>
<th>Variable</th>
<th>Class 1 (n=107)</th>
<th>Class 2 (n=56)</th>
<th>Class 3 (n=19)</th>
<th>Between-Class Test Statistics</th>
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<tr>
<td>Between-class statistics</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9 mo</td>
<td>43.4 (9.7)†</td>
<td>69.2 (16.5)‡</td>
<td>86.8 (10.9)‡</td>
<td>F = 16.0, df = 2, 151, P &lt; .001</td>
</tr>
<tr>
<td>21 mo</td>
<td>38.4 (6.7)†</td>
<td>57.2 (8.1)†</td>
<td>79.9 (12.2)‡</td>
<td></td>
</tr>
<tr>
<td>33 mo</td>
<td>37.8 (8.4)†</td>
<td>53.9 (10.1)‡</td>
<td>79.0 (12.3)‡</td>
<td></td>
</tr>
<tr>
<td>Within-class test statistics</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9 vs 21 mo</td>
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<td>t = 4.1, df = 44</td>
<td>t = 0.8, df = 9</td>
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</tr>
<tr>
<td>P value</td>
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<td>&lt; .001</td>
<td>.47</td>
<td></td>
</tr>
<tr>
<td>21 vs 33 mo</td>
<td>t = 2.0, df = 38</td>
<td>t = 0.4, df = 10</td>
<td>t = 0.0, df = 9</td>
<td></td>
</tr>
<tr>
<td>P value</td>
<td>.08</td>
<td>.05</td>
<td>.70</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: ellipses, not applicable; ICG-RC, Inventory of Complicated Grief–Revised for Children version.

a Small footnote symbols (†, ‡, and †) represent post hoc differences. Use of the same symbol represents no significant post hoc difference at P < .006.

The rest of the analyses were conducted with STATA statistical software. We analyzed 3 outcomes (ICG-RC class, functional impairment, and incident depression). We used a Bonferroni correction to correct for multiple comparisons for univariate analyses with an α of 0.05/3 = .017. Post hoc pairwise comparisons within ICG-RC class were conducted only after a statistically significant test. Three classes resulted from LCGA and, hence, 3 possible pairwise comparisons resulted, for which α was set at 0.017/3 = .006. For all multivariate models, we used P < .05 because variables included in these models already were significant at the corrected level. Because multiple children and adolescents per family were recruited, we included a cluster effect in all regression models. Our power analyses for regression models, using Cohen tables of the Effect Size Index F2, indicate that we have power to detect effect sizes in the order of F2 = 0.23 with 20 variables (ie, main effects and interaction terms) included in the model with α = .01 and power of 0.80. This corresponds to medium to large effect sizes for which this study was designed to detect. We define the most parsimonious model as the simplest model with the highest predictive value as defined by pseudo-R2. We removed variables from the final model if they were not significant at P < .05. Finally, we tested for 2-way interactions between variables in the final model.

Paired t tests were used to examine changes in ICG-RC scores between 9 and 21 months and between 21 and 33 months within each class or trajectory identified, and analysis of variance was used to compare scores among classes at 9, 21, and 33 months. We compared children and adolescents in the 3 classes identified on the following domains assessed at baseline: demographic; clinical characteristics before the death, such as personal and family history of psychiatric disorders; circumstances of exposure to the death; and clinical characteristics at 9 months after the death. We used univariate statistics for these comparisons (derived from the χ2 test, the Fisher exact test, and analysis of variance) with post hoc pairwise comparisons, as described herein. Because we identified 3 classes, multinomial logistic regression was used to examine the most parsimonious set of predictors. Regression, using the hierarchical method, was applied including the variables (n = 17) that were significantly different among ICG-RC classes (Table 1). We controlled for the effect of time of the baseline interview in relation to time of death. We included 4 blocks in the hierarchical model in the order in which they are listed: characteristics before the death, circumstances of exposure to death (eg, type of death), clinical diagnoses after the death in offspring and the surviving parent, and finally, self-reported symptoms and psy-
variables were included in a multivariate Cox proportional
haz- 
cions. Fifteen variables, including ICG-RC class, were signifi-
tics. We used Wil-
monious predictors testing for main effects and 2-way inter-
variables, including ICG-RC class, were significantly associated with

tial characteristics assessed are correlated with diagnostic vari-
t and effects are of interest only if they were
nostics assessed are correlated with diagnostic variables in block 3 and their effects are of interest only if they were statistically significant in the presence of variables from preceding blocks (ie, previous psychiatric history and clinical di-
We then examined whether the course of functional impair-
differed by ICG-RC class beyond other clinical characteristics. We used mixed-effects regression models with GAS scores as the outcome and with main effects of class or correlate, time, and class or correlate × time interaction. Of these analyses, 0 variables, including ICG-RC class, were significantly associated with GAS scores as a main effect or in interaction with time. Next, we examined a multivariate mixed model, which included ICG-RC class, time, the other correlates that were significantly associated with GAS scores, and the results of testing for 2-way interactions with time. A total of 13 variables (ie, main effects and interactions) were included in the multivariate model. We compared models with time as random vs fixed using a likelihood ratio test and used time as random or fixed accordingly. The ICG-RC class was represented with a 2-df parameter. The time of baseline and fol-
was separated from block 3, because the self-reported symptoms and psychosocial characteristics assessed are correlated with diagnostic variables in block 3 and their effects are of interest only if they were statistically significant in the presence of variables from preceding blocks (ie, previous psychiatric history and clinical di-
compared with nonbereaved control individuals.
We report the incidence rate ratio (IRR) of depression by ICG-RC class. Also, we examine whether ICG-RC scores at 9 months predict incident depression during follow-up beyond other correlates significantly associated with incident depression (n = 4 variables). Logistic regression is used to identify the most parsi-
We conducted all analyses for the original and an im-
We applied the Multiple Imputation by Chained Equations technique in STATA (ie) with an inclusive stra-
We then examined whether the course of functional impair-
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RESULTS

GRIEF TRAJECTORIES

The best-fitting model of ICG-RC trajectories using LCGA consisted of 3 classes (Figure 1), which correspond to class 1, consisting of 107 cohort individuals (58.8%) who scored in the lower 50th percentile on the ICG-RC at 9 months after the death (interquartile range, 68-40), who experienced a significant decrease in grief scores between 9 and 21 months, and whose scores remained low afterward (Table 1); class 2, consisting of 56 individuals (30.8%) with grief scores in the 75th percentile at 9 months and who showed a steady decline of their manifestations of grief from 9 through 33 months; and class 3, consisting of 19 individuals (10.4%) with grief scores in the 75th percentile at 9 months, with virtually no change at 21 months and 33 months later. Significant differences were found in ICG-RC scores among the 3 trajectories (P < .001) at the 9-, 21-, and 33-month assessments since the death, with pairwise comparisons resulting in significantly higher scores for class 3, followed by class 2 and class 1. Of the 19 individuals in class 3, 6 (31.6%) met the criteria for depression, anxiety, or PTSD throughout the follow-up period. Seven individuals (36.8%) had 1 of these diagnoses at the time of death that was prolonged or had occurred, and 6 (31.6%) did not meet the criteria for any of these disorders.

BASELINE PREDICTORS OF GRIEF TRAJECTORIES

We compared ICG-RC classes with respect to demographic,
clinical characteristics before the death, circumstances of exposure to the death, and clinical characteristics 9 months after the death (Table 2). Using multinomial logistic regression (Table 3), the most parsimonious model (χ² = 61.9, P < .001, pseudo-R² = 0.29) that differentiated class 2 and 3 from class 1 included parental loss due to unintentional death and higher self-reported depression in the child or adolescent at 9 months. In contrast, child or adolescent functional impairment at 9 months, previous history of depression, and incident PTSD significantly differentiated class 3 from class 1 and class 2. A previous history of bipolar disorder in the proband significantly differentiated class 2 from class 1 only.

COURSE OF FUNCTIONAL IMPAIRMENT BY GRIEF TRAJECTORIES

No significant differences were found in the course of functional impairment, as measured by the CGAS, by

Figure 1. Latent class growth curve modeling on the Inventory of Complicated Grief–Revised for Children version (ICG-RC). Red, green, and blue lines represent classes 1, 2, and 3, respectively. Circles indicate the sample mean; triangles indicate the estimated mean. To avoid nonconvergence and local solutions, we used 100 random sets of starting values with 10 final optimizations and we verified that the final highest likelihood value was replicated at least twice.

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ICG-RC class, as evident by the nonsignificant class \times time interaction (Table 4). However, a main effect for class 3 showed significantly lower scores on the GAS, which reflects worse functioning ($\beta=-14.1$, $z=-2.7$, $P=.006$).

Using multivariate mixed-effects regression ($\chi^2=87.6$, $P<.001$, pseudo-$R^2=0.26$), overall worsening was observed in functional impairment of bereaved children and adolescents over time ($\beta=-1.6$, $z=-2.7$, $P=.006$). This
decline in functional impairment was more prominent in class 2 ($\beta=-3.7, z=-2.1, P=.03$) relative to class 1 and most markedly in class 3 relative to class 1 ($\beta=-9.3, z=-3.3, P=.001$). These findings persisted, even after controlling for other significant correlates, namely, current anxiety symptoms ($\beta=-0.7, z=-2.9, P=.003$) and PTSD symptoms ($\beta=-0.52, z=-5.5, P<.001$).

GRIEF TRAJECTORIES AND INCIDENCE OF DEPRESSION

A total of 59 incident cases (32.6%) of depression occurred within 3 years after the death. Class 2 (IRR, 2.9; 95% confidence interval [CI], 1.60-5.30; $P<.001$) and class 3 (2.5; 0.98-5.80; $P=.001$) had an almost 3-fold increased incidence of depression compared with class 1, but no difference was observed between classes 2 and 3 ($0.87; 0.34-1.97; P=.38$).

To assess whether ICG-RC scores at 9 months predict incident depression by 21 or 33 months, we excluded children and adolescents who met criteria for depression at the time of parental death ($n=8$) and those who already had incident depression by the 9-month assessment ($n=41$). We compared individuals with incident depression by 21 or 33 months ($n=18$) with those who did not experience depression on baseline characteristics assessments, including the ICG-RC. The ICG-RC scores at 9 months, child or adolescent rating of aggression, feeling others were accountable for the death, life events since the death, and complicated grief in the surviving parent were significantly associated with incident depression. Using logistic regression, the most parsimonious model ($\chi^2=19.6, P=.003$, pseudo-$R^2=0.33$) included a significant interaction between ICG-RC scores in children and adolescents, and their surviving parent’s complicated grief (odds ratio [OR], 1.2; 95% CI, 1.00-1.36; $z=2.0; P=.049$), meaning that children and adolescents with higher ICG-RC scores and whose surviving parent had complicated grief were at increased risk for incident depression. Also, feeling that others were accountable for the death (OR, 7.4; 95% CI, 1.96-27.94; $z=3.0; P=.003$) and life events since the death (1.2, 1.02-1.42; $z=2.2; P=.03$) were associated with increased risk of incident depression (Hosmer-Lemeshow goodness of fit, $\chi^2=7.4; P=.49$).

GRIEF TRAJECTORIES AND TIME TO ONSET OF DEPRESSION

Parallel analyses conducted to examine differences in time to onset of incident depression also found significant difference among the 3 classes (Wilcoxon test, $\chi^2=13.5; P=.001$; log-rank, $\chi^2=15.2; P<.001$) (Figure 2). Classes 2 and 3 had significantly earlier onset of depression compared with class 1, but no differences were found between classes 2 and 3. Cox proportional hazards regression ($\chi^2=63.7, P<.001$) showed that class 2 (hazard ratio [HR], 3.3; 95% CI, 1.57-7.04; $z=3.1; P=.002$) and class 3 (2.5; 1.06-5.68; $z=2.1; P=.04$) had increased HRs compared with class 1, even after controlling for other characteristics associated with earlier onset of depression, namely, indirect aggression (1.3, 1.16-1.45; $z=4.5; P<.001$), attention-deficit/hyperactivity disorder in the offspring (2.6, 1.06-5.99; $z=2.2; P=.04$), and having had a last confiding conversation with the deceased parent (2.8, 1.15-6.62; $z=2.3; P=.02$).

Table 4. Mixed-Effects Regression Models for Functional Impairment

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Variable</th>
<th>Time</th>
<th>Variable × Time</th>
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<tr>
<td></td>
<td>$\beta$</td>
<td>$P$</td>
<td>$\beta$</td>
</tr>
<tr>
<td>Before the death</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Offspring relationship to the deceased</td>
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<td>.002</td>
<td>6.5</td>
</tr>
<tr>
<td>After the death</td>
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<td></td>
</tr>
<tr>
<td>ICG-RC class $^b$</td>
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<td>-0.05</td>
</tr>
<tr>
<td>2</td>
<td>-14.1</td>
<td>.006</td>
<td>-0.05</td>
</tr>
<tr>
<td>3</td>
<td>-37.9</td>
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<td>-0.7</td>
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<tr>
<td>Offspring sexual abuse</td>
<td>-9.5</td>
<td>.007</td>
<td>-1.5</td>
</tr>
<tr>
<td>Surviving parent complicated grief (ICG score $\geq 25$)</td>
<td>-3.4</td>
<td>.01</td>
<td>-0.6</td>
</tr>
<tr>
<td>Self-reported symptoms</td>
<td>0.05</td>
<td>.49</td>
<td>1.3</td>
</tr>
</tbody>
</table>

Abbreviations: ICG, Inventory of Complicated Grief; ICG-RC, Inventory of Complicated Grief–Revised for Children version; PTSD, posttraumatic stress disorder.

$^a$Time defined as ln (months since the death).

$^b$Introduced as a dummy variable (or 2-df parameter), with class 1 as the reference group.

Figure 2. Kaplan-Meier curve of time to onset of depression by Inventory of Complicated Grief–Revised for Children version class. Red, green, and blue lines represent classes 1, 2, and 3, respectively.
More than half of children and adolescents bereaved by sudden parental death experienced relatively rapid resolution of their manifestations of grief within 1 year of the loss of their parent. However, sometimes the course of grief was more problematic, with 30.8% showing a more gradual diminution in grief symptoms and 10.4% showing high and sustained prolonged grief manifestations nearly 3 years after parental death. The experience of a PGR was predicted by a prior personal history of depression and was associated with greater functional impairment beyond that accounted for by other psychopathologic conditions. Children and adolescents with PGRs also show an increased incidence and earlier onset of depression predicted by their grief reactions and those of their surviving parent 9 months after the death, even after controlling for characteristics before and after the death.

Before we discuss the implications of these findings, we need to put them in the context of the strengths and limitations of this study. Ours is the first population-based, longitudinal study, to our knowledge, of the effect of sudden parental death on children and adolescents. It has the largest sample of suicide-bereaved offspring and includes a comprehensive assessment of offspring and surviving parents, in addition to a psychological autopsy assessment of the deceased proband. However, it is difficult to determine whether this sample is representative because coroners' records do not routinely list surviving offspring and because natural deaths are not routinely the province of the coroner. However, our sample of suicides and unintentional deaths was demographically similar to the population that died of those causes in Allegheny County overall, and our response rate was 71.0%, which is relatively high for these types of studies. It is possible that referral biases existed: families experiencing greater discomfort who were more worried about their children and adolescents potentially were more likely to participate. However, it is of interest that those bereaved families recruited by advertisement showed lower rates of proband alcohol or substance abuse disorders, which would argue against such a referral bias. Our profiles of the deceased parents are similar to those obtained in previous psychological autopsy studies, which makes it more likely that our results did not occur simply due to sampling bias. A second limitation is the use of an informant rather than self-report in the case of the deceased probands. However, the psychological autopsy procedure is a specific and fairly sensitive method for determining psychiatric disorders and, if anything, is likely to underestimate the rate of disorder, thus biasing the results more toward the null hypothesis. Although differences were observed between study participants retained and those lost to follow-up regarding negative coping and blaming others for the death, study participants retained were not significantly different from those lost to follow-up regarding grief scores. Finally, the study sample is mostly white and does not include youth bereaved by homicide or anticipated parental death, which limits the generalizability of these findings to those bereaved by other types of death.

We find the profile of children and adolescents with a problematic course of grief (ie, those with gradual diminution of grief symptoms and those with sustained prolonged grief) to include a previous history of depression and a family history of bipolar disorder. Higher rates of psychiatric disorders are expected in those who complete suicide and hence in their families; however, some of us previously reported that early parental death due to a wide range of causes, namely, suicide, unintentional injuries, and sudden natural causes, was associated with a history of bipolar disorder, alcohol and substance abuse, and personality disorders in the deceased parent. Increased rates of psychiatric disorders in the deceased parents suggest a preexisting vulnerability in their offspring that predates parental death and thus puts them at increased risk for adverse outcomes after bereavement.

Children and adolescents with PGRs will show greater functional impairment within the first year after the death and during the 3-year course of follow-up. Moreover, PGRs make a unique contribution to the functional impairment of bereaved children and adolescents because the association between prolonged grief and impairment persists even after controlling for personal and family history of psychiatric disorders before and after the death. Melhem and colleagues previously reported, in a cross-sectional analysis of this sample, that grief scores are associated with functional impairment beyond other psychopathologic conditions. Herein, we show that the association between prolonged grief and impairment persists over time. These results are convergent with findings in bereaved adults.

We found a bidirectional relationship between prolonged grief and psychiatric vulnerability. Prior psychiatric disorder in the offspring and parent increased the likelihood of the occurrence of prolonged grief in the offspring. However, even controlling for these factors, prolonged grief was associated with an increased hazard of incident depression. Thus, prolonged grief makes a unique contribution to the functional impairment and psychiatric morbidity of bereaved children and adolescents, which justifies a clinical focus not only on psychiatric sequelae but also on prolonged grief. In adults, interventions that target complicated grief have been shown to be effective in restoration of function and relief from grief compared with interpersonal therapy. Although preventive interventions for community samples of bereaved youth and their families have been shown to reduce grief reactions, the development of clinical interventions to target prolonged grief is likely to be as important for helping bereaved children and adolescents, as has been demonstrated in the treatment of bereaved adults. Also, preventive interventions not only should target bereaved children and adolescents with prolonged grief but also those with increased grief reactions 9 months after the death. Grief reactions in children and adolescents in class 2 gradually diminished during 33 months, but these children and adolescents still showed functional impairment and were at increased risk of incident depression.

One of the most consistently reported findings is that caregivers’ well-being after parental bereavement is a sig-
significant predictor of children's and adolescents' well-being. We found that the combination of complicated grief in the surviving parent and the offspring was particularly potent in predicting incident depression in children and adolescents as long as 3 years after the death. These findings have important clinical implications regarding intervention and prevention efforts. It is imperative to assess the surviving parent and to intervene, when appropriate, to improve the outcomes for parentally bereaved children and adolescents. Adaptations of successful adult treatment approaches to the treatment of prolonged grief in children and adolescents may require interventions that are focused on the family rather than on the individual. Future studies are needed to examine the long-term mental health and developmental outcomes in bereaved children and adolescents, to examine the etiologic and biological pathways by which prolonged grief exerts its effects, and to develop interventions to promote relief from grief in parentally bereaved children and adolescents.

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REFERENCES


