Alcohol and Substance Abuse in Parentally Bereaved Youth

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ABSTRACT

Objective: Little is known about the role of parental bereavement regarding alcohol and substance abuse. Our aim was to examine whether the incidence of alcohol and substance abuse is higher in parentally bereaved youth and, if so, what might explain this increased incidence.

Method: In a longitudinal population-based study conducted between November 2002 and December 2012, the incidence of alcohol and substance abuse or dependence (ASAD) during a period of 5 years was examined (using *DSM-IV* criteria) in 235 youth whose parents died of suicide, accident, or sudden natural death and 178 demographically similar nonbereaved youth.

Results: In a period that covered 5 years subsequent to the death, bereaved youth had an increased incidence and earlier time to onset of ASAD relative to nonbereaved controls (incident rate ratio = 2.44; 95% CI, 1.17–5.56). Additionally, youth over the age of 13 years (hazard ratio [HR] = 6.68; 95% CI, 3.22–13.89; $P < .001$), those who developed a disruptive behavior disorder (HR = 7.55; 95% CI, 1.83–31.22; $P = .005$), and those who had greater functional impairment (HR = 0.93; 95% CI, 0.90–0.95; $P < .001$) were at increased risk for ASAD. However, after adjusting for the above-noted variables, the relationship between parental bereavement and pathological youth alcohol and substance use was not statistically significant (HR = 1.73; 95% CI, 0.79–3.81; $P = .17$).

Conclusions: Bereaved youth are at greater risk for ASAD than their nonbereaved counterparts, especially adolescent boys with disruptive behavior disorders. The effect of bereavement was explained by its overall impact on greater functional impairment in bereaved offspring. Interventions that help to improve offspring functioning and that prevent or attenuate the development of disruptive behavior disorders have the potential to prevent ASAD in bereaved youth.

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There is an increased risk for adverse psychiatric sequelae, namely, depression and posttraumatic stress disorder (PTSD), after the loss of a parent, sibling, or close friend.[1–3] Longitudinal follow-up studies indicate that the period of greatest risk for these conditions is immediately after the loss, whereas after the first year postbereavement, the incidence of new-onset depression is not different from that in controls.[1,4–7] With regard to PTSD, the period of greatest increased risk is immediately after the death, although there may be evidence of an increased period of risk beyond the first year postloss.[4–7]

Less is known about the possible negative long-term effects of bereavement on the development of alcohol and substance abuse or dependence (ASAD). Retrospective studies[8–10] have generally found that parental separation and early childhood adversity have a stronger impact than parental bereavement per se on ASAD in youth. Nonetheless, in a large, longitudinal, developmental epidemiologic study,[11] parentally bereaved youth were found to show higher rates of alcohol and substance abuse symptoms than their nonbereaved counterparts. Further, a natural record linkage study[12] found that youth whose parents died by suicide were at greater risk than nonbereaved youth to develop substance abuse. Even less has been done with respect to the relationship of complicated grief to alcohol and substance abuse, with only 1 report[13] documenting such a relationship in a sample of bereaved bipolar patients.

Thus, there is some (albeit mixed) support for a relationship between parental loss and subsequent ASAD. There are multiple pathways that might theoretically explain such a relationship. First, parents who die prematurely have higher than expected rates of ASAD, and, consequently, their children are at increased risk through both genetic and environmental mechanisms.[1] Second, some of the sequelae of parent loss, such as depression, PTSD, and health risk behaviors, are associated with an increased risk for ASAD.[14–17] Third, surviving parents may be burdened by depression, PTSD, and economic and child-rearing responsibilities and may be less effective in monitoring and supervising their children, resulting in increased risks for externalizing disorders and ASAD.[1,18–20]

Our controlled, longitudinal study of youth who lost a parent to suicide, accident, or sudden natural death provides an ideal opportunity to address the following questions: (1) Is ASAD a sequela of parental bereavement? (2) What are other child, parental, and contextual factors that predict onset of ASAD? (3) Does parental bereavement convey an increased risk for ASAD, even after controlling for other predictors of the onset of ASAD in this sample? and (4) What are the predictors of the onset of ASAD within the bereaved sample?

We hypothesized that there would be an increased risk of ASAD in bereaved youth. We also predicted that other factors would convey an increased risk for ASAD, namely, higher rates of ASAD in deceased parents and surviving caregivers and prior and current psychiatric disorders in the offspring, especially externalizing disorders. Further, we posited that more adaptive functional status in the caregiving parent and adaptability and cohesion levels in bereaved families would be protective against ASAD. We anticipated that the impact of bereavement on ASAD would be attenuated

after controlling for these other predictors of this outcome. We also hypothesized that, within the bereaved group, those youth who experience complicated grief would be at higher risk of developing ASAD.

Clinical Points

- Interventions aimed at preventing or attenuating the development of disruptive behavior disorders (oppositional defiant or conduct disorders), depression, and posttraumatic stress disorder may potentially prevent alcohol and substance abuse or dependence in bereaved youth.
- Treatment of psychiatric illness in caregivers and interventions to improve their parenting efficacy and overall functioning are also likely to be protective against the development of alcohol and substance abuse or dependence.

METHOD

Sample

The bereaved sample consisted of 235 offspring (34.4% were bereaved by suicide, 23.8% were bereaved by accidental death, and 41.7% were bereaved by sudden natural death) and 147 adult caregivers, with 178 youths and 98 adult caregivers included in the nonbereaved comparison group. Because we were interested in new-onset ASAD after bereavement, 12 offspring with a history of ASAD prior to bereavement or the equivalent time period for controls were excluded, 7 from the bereaved group and 5 from the nonbereaved group (2.9% vs 2.7%, $\chi^2_1 = 0.01$, $P = .91$). Retention for the study from the baseline assessment over the next 3 follow-up timepoints was 89.8% at 21 months, 83.8% at 33 months, and 71.4% at 62 months after the death. Subjects lost to follow-up were more likely to be bereaved (76.3% vs 23.7%, $\chi^2_1 = 7.3$, $P = .009$) and showed greater functional impairment as assessed with the Children's Global Assessment Scale[21] and Global Assessment Scale[22] (mean [SD] = 74.3 [13.0] vs 79.3 [11.5]; $t_{389} = 2.5$, $P = .01$). Additionally, the caregivers of subjects lost to follow-up were more likely to be diagnosed with PTSD (29.4% vs 14.4%; $\chi^2_1 = 5.3$, $P = .02$), to be more functionally impaired as assessed with the Global Assessment Scale (mean [SD] = 71.06 [13.8] vs 77.9 [11.4]; $t_{367} = 3.3$, $P = .001$), and to have had a lower socioeconomic status as assessed with the Hollingshead scale[23] (mean [SD] = 30.5 [8.6] vs 36.9 [8.2]; $t_{244} = 2.86$, $P = .005$). The deceased probands of participants lost to follow-up had a higher rate of borderline personality disorder (14.3% vs 3.5%, Fisher exact test, $P = .008$). At the time of last assessment, this sample was 49.7% female, 84.5% white, 11.1% African American, and 4.4% biracial and had a mean age of 17.6 years (SD = 3.7) and socioeconomic status of 44.3 (SD = 12.1), which corresponds to medium business, minor professional, and technical occupations. There were no differences between the 2 offspring groups on any of these characteristics (P values > .06).

Recruitment

The deceased probands were between the ages of 30 and 60 years, had biological offspring between the ages of 7 and 25 years, and had died within 24 hours of the precipitating event from suicide, an accident, or sudden natural cause. Families in which there were multiple deaths or injuries were excluded. Bereaved families were recruited through coroner's records (49.7%) and by newspaper advertisement (50.3%); 71% of eligible bereaved families were recruited. Control families were recruited using random-digit dialing and by advertisement. Controls were frequency matched with the bereaved families on the age, race, sex, and neighborhood of the proband. Both biological parents needed to be alive, and the family could not have been exposed to the death of any first-degree relative within the previous 2 years. Slightly over half (55%) of the potential controls agreed to participate. This study was approved by the University of Pittsburgh Institutional Review Board. After a complete description of the study, caregivers' consent was obtained for their participation, and either assent or consent was obtained from their offspring. Further details on recruitment procedures were described previously.[1,5,24] The study was conducted between November 2002 and December 2012.

Assessment

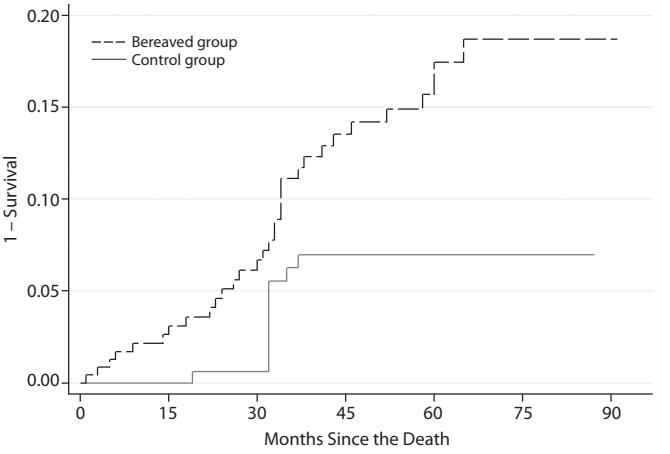
Participants were interviewed by master's degree–level interviewers with a background in psychology or social work at 9, 21, 33, and 62 months after the death, with parallel timing for the nonbereaved controls. Socioeconomic status and household income were rated using the Hollingshead scale.[23] Psychiatric disorders were assessed using the Structured Clinical Interview for *DSM-IV* Axis I Disorders (SCID-I) for offspring 18 years and older and caregivers and the Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime Version for offspring younger than 18 years.[25,26] Personality disorders were assessed in those 18 years and older using the Structured Clinical Interview for *DSM-IV* Axis II Personality Disorders (SCID-II).[27] Psychiatric assessment of the proband was conducted using a psychological autopsy procedure.[28] Functional status was assessed using the Children's Global Assessment Scale[21] for child or adolescent offspring or the Global Assessment Scale[22] for young adult offspring and caregiver. Complicated grief was assessed using the Inventory of Complicated Grief in caregivers.[29] In offspring, the Inventory of Complicated Grief–Revised for Children[30,62] which consisted of 28 items, was used. A history of abuse, including onset and duration, was obtained using a measure based on the Abuse Dimensions Inventory.[31] The severity of PTSD symptoms was assessed by the Child PTSD Symptom Scale Interview[32] in children and the parallel PTSD Symptom Scale Interview for adults.[33] Self-reported depression, anxiety, and suicidal ideation were assessed in offspring using the Mood and Feelings Questionnaire,[34] Screen for Child Anxiety Related Emotional Disorders,[35] and Suicidal Ideation Questionnaire-JR, respectively.[36] Parallel measurements in adults were obtained using the Beck Depression Inventory,[37] Beck Anxiety Inventory,[38] and Adult Suicidal Ideation Questionnaire.[39] Intercurrent life events were assessed using the Life Events Checklist[40] in offspring younger than 18 years

and the shortened Social Readjustment Rating Scale[41,42] of Holmes and Rahe in offspring 18 years and older. Factors that might buffer the impact of bereavement were also assessed in offspring. Family cohesion and self-esteem were assessed using the Family Adaptability and Cohesion Evaluation Scale II[43] and the Weinberger Adjustment Inventory[44] self-esteem subscale, respectively. Social support and coping style were assessed using the Survey of Children's Social Support[45] and Kidcope,[46] respectively, in offspring younger than 18 years. These domains were assessed by the Multidimensional Scale of Perceived Social Support[47] and the Ways of Coping questionnaire[48] in offspring 18 years and older.

Data Analysis

We compared the bereaved and nonbereaved groups on time to onset of ASAD using Kaplan-Meier survival analyses. Cox regression models were also used to estimate the influence of potential predictors of the onset of alcohol and substance abuse by examining 1 variable at a time and then selecting the significant subset of predictors for multivariable analyses using the false discovery rate method with the Yekutieli procedure (*qqvalue* package in STATA).[49] We examined sets of variables related to the onset of ASAD on the basis of temporal order: demographics, antecedent variables (such as history of parental or offspring disorder that antedated the death), baseline variables (such as diagnoses that were present after the death, but not prior to it), and longitudinal correlates (such as new-onset depression, that first emerged at subsequent timepoints). Demographic, antecedent, and baseline variables were used as fixed effects, while longitudinal variables were used as time-varying covariates, meaning that all measurements up to the timepoint of ASAD (or the maximum timepoint in the study for those who did not experience an onset) were included in the model. The final, most parsimonious set of variables was selected using a backward stepwise method and a significance level of .05. We included a cluster effect in all Cox models to account for sibling pairs in the sample. Finally, we repeated these analyses just within the bereaved sample in order to identify factors within the bereaved sample that predicted onset of alcohol and substance abuse.

Figure 1. Kaplan-Meier Curve of Time to Onset of Alcohol and Substance Abuse or Dependence[a]



[a]Incidence ratio, 2.44; 95% CI, 1.17–5.56.

RESULTS

Alcohol and Substance Abuse or Dependence in Bereaved and Controls

There were 42 incident cases of ASAD; 26 met criteria for abuse only, whereas 16 had dependence. Bereaved participants had higher rates of ASAD than controls (13.6% vs 5.6%; $\chi^2_1 = 7.09$, $P = .008$), an increased hazard ratio (HR) for developing ASAD (HR = 2.49; 95% CI, 1.16–5.32; $z = 2.34$, $P = .02$), and an earlier time to ASAD onset (Wilcoxon $\chi^2_1 = 6.66$, $P = .01$; log-rank test $\chi^2_1 = 6.77$, $P = .009$) (Figure 1). The magnitude of the HR conveyed by bereavement was similar for the subgroup with abuse (HR = 2.59; 95% CI, 1.04–6.44; $z = 2.05$, $P = .04$), with dependence (HR = 2.34; 95% CI, 0.62–8.76; $z = 1.26$, $P = .21$), with alcohol abuse or dependence alone (8 incident cases: HR = 2.39; 95% CI, 0.51–11.31; $z = 1.10$, $P = .27$), with substance abuse or dependence alone (22 incident cases: HR = 2.63; 95% CI, 0.97–7.17; $z = 1.90$, $P = .06$), and with both types of abuse or dependence (12 incident cases: HR = 2.60; 95% CI, 0.71–9.56; $z = 1.44$, $P = .15$). Given the small number of incident cases in the subgroups, we only analyzed ASAD as an outcome.

Demographic and Antecedent Predictors

Cox regression models were used to examine the associations between ASAD and pre-death, baseline, and longitudinal variables (Table 1).

Being 13 years or older at the time of parental death (or at 6 months prior to intake for the controls) increased the risk for ASAD (HR = 4.91; 95% CI, 2.29–10.50; $P < .001$). A history of disruptive behavior disorders (DBDs; ie, conduct and/or oppositional defiant disorder) prior to intake/bereavement was associated with an increased risk for ASAD (HR = 5.35; 95% CI, 1.97–14.55; $P = .001$). Similarly, a past diagnosis of major depressive disorder in the proband (meaning prior to death or the equivalent in the controls) significantly increased the risk for offspring ASAD (HR = 2.90; 95% CI, 1.49–5.63; $P = .002$). Caregivers' clinical characteristics were not found to increase the risk for ASAD.

Longitudinal Predictors

Within offspring, a new-onset diagnosis of DBDs (HR = 16.16; 95% CI, 5.75–45.47; $P < .001$), poorer functional status (HR = 0.93; 95% CI, 0.91–0.95; $P < .001$), and higher numbers of negative life events (HR = 1.20; 95% CI, 1.10–1.30; $P < .001$) were related to the onset of ASAD.

Controlling for demographic characteristics, the most parsimonious set of the above-noted variables associated with onset of ASAD was age ≥ 13 years at the time of intake/parental death (HR = 6.68; 95% CI, 3.22–13.89; $P < .001$), being male (HR = 2.55; 95% CI, 1.28–5.06; $P = .007$), DBD onset after baseline (HR = 7.51; 95% CI, 1.82–31.01; $P = .005$), and more impaired functional status in offspring (HR = 0.93;

Table 1. Univariate Cox Regression of Antecedent and Baseline Correlates of Time to Onset of Alcohol and Substance Abuse or Dependence

	Hazard Ratio	95% CI	*z*	*P*	*q*[a]
Offspring					
Demographics					
Bereaved	2.49	1.16–5.32	2.34	.02	0.27
Age ≥ 13 y at time of parent's death	4.91	2.29–10.50	4.10	< .001	0.002
Gender, male	2.03	1.04–3.97	2.07	.04	0.37
Race, white	0.39	0.18–0.87	−2.31	.02	0.27
History of psychopathology					
MDD	2.28	1.09–4.75	2.20	.03	0.33
DBDs[b]	5.35	1.97–14.55	3.28	.001	0.04
ADHD	3.10	1.24–7.73	2.42	.02	0.26
Anxiety	1.26	0.43–3.71	0.42	.67	> 0.99
PTSD	3.74	0.41–34.30	1.17	.24	> 0.99
Clinical and psychosocial correlates					
Functional status[c]	0.93	0.91–0.95	−6.64	< .001	< 0.001
Physical or sexual abuse	3.23	1.08–9.68	2.09	.04	0.37
Health risk behaviors	0.83	0.70–0.98	−2.21	.03	0.31
Family adaptability and cohesion	0.96	0.93–0.99	−2.35	.02	0.27
Life events	1.20	1.10–1.30	4.27	< .001	< 0.001
Self-reported depression	1.22	0.91–1.62	1.35	.18	> 0.99
Self-reported anxiety	1.28	0.85–1.92	1.19	.24	> 0.99
Suicidal ideation	1.02	0.99–1.04	1.55	.12	0.89
Aggression	1.01	1.00–1.03	1.69	.09	0.69
Negative coping—efficacy	1.52	1.04–2.20	2.18	.03	0.33
Negative coping—frequency	1.22	0.75–2.00	0.78	.43	> 0.99
Self-esteem	1.01	0.95–1.07	0.28	.78	> 0.99
Social support	0.88	0.65–1.19	−0.83	.41	> 0.99
New-onset psychopathology (up to the time of alcohol and substance abuse onset)					
MDD	2.42	1.08–5.44	2.14	.03	0.34
PTSD	5.53	1.80–16.94	2.99	.003	0.07
DBDs[b]	16.16	5.75–45.47	5.27	< .001	< 0.001
ADHD	2.45	0.62–9.67	1.28	.20	> 0.99
Proband					
History of psychopathology					
MDD	2.90	1.49–5.63	3.14	.002	0.05
Anxiety	1.45	0.70–3.00	1.01	.31	> 0.99
Bipolar	0.64	0.16–2.52	−0.64	.52	> 0.99
PTSD	2.73	1.21–6.16	2.42	.02	0.26
Alcohol/substance abuse	1.59	0.76–3.30	1.23	.22	> 0.99
Personality disorders	2.68	1.32–5.41	2.74	.006	0.12
Adult caretaker					
History of psychopathology					
MDD	0.77	0.33–1.80	−0.60	.55	> 0.99
Anxiety	1.02	0.50–2.05	0.05	.96	> 0.99
Bipolar	2.89	0.96–8.72	1.88	.06	0.49
PTSD	0.48	0.12–1.90	−1.04	.30	> 0.99
Alcohol/substance abuse	1.99	0.99–3.97	1.94	.05	0.45
Personality disorders	3.95	1.01–15.42	1.98	.05	0.44
New-onset psychopathology (up to the time of alcohol and substance abuse onset)					
MDD	1.10	0.37–3.29	0.17	.87	> 0.99
Anxiety	0.48	0.07–3.34	−0.74	.46	> 0.99
PTSD	1.65	0.41–6.58	0.71	.48	> 0.99
Alcohol/substance abuse	3.11	0.85–11.35	1.72	.09	0.68
Functional status[c]	0.96	0.93–0.99	−2.88	.004	0.09

[a] *q* Value calculated with the *qqvalue* STATA package.
[b] Conduct and/or oppositional defiant disorder.
[c] Lower scores indicate more impairment.
Abbreviations: ADHD = attention-deficit/hyperactivity disorder, DBD = disruptive behavior disorder, MDD = major depressive disorder, PTSD = posttraumatic stress disorder.

95% CI, 0.90–0.95; *P* < .001) (Table 2). No significant interactions were found. The impact of bereavement on adolescent ASAD after controlling for age, gender, and onset of DBD was not statistically significant (HR = 1.73; 95% CI, 0.79–3.81; *P* = .17).

This same set of variables predicted the onset of ASAD within the bereaved group, without an effect of cause of death: being 13 or older at death (HR = 5.61; 95% CI, 2.54–12.36; *P* < .001), male (HR = 2.77; 95% CI, 1.28–6.01; *P* = .01), more functionally impaired (HR = 0.92; 95% CI, 0.89–0.96; *P* < .001) and being diagnosed with a new onset of a DBD (HR = 7.70; 95% CI, 1.59–37.36; *P* = .01) (Table 3). Counter to our hypothesis, the presence of complicated grief was not related to ASAD (HR = 0.41; 95% CI, 0.11–1.61; *P* = .20). No significant interactions were found between the terms in the model.

DISCUSSION

The aim of this report was to explore the relationship between parental loss due to sudden death and the incidence of ASAD in offspring followed up to 5 years after loss. We found that the bereaved offspring were at around 2.4 times higher risk for ASAD compared to their nonbereaved counterparts. However, the effect of bereavement on ASAD was attenuated (adjusted HR = 1.73) after controlling for the incidence of DBDs and functional impairment.[1,50]

The implications of these results should be considered in the context of the strengths and limitations of this study. As for strengths, this is one of the only prospective, controlled community studies of parental bereavement, with relatively good sample retention over a 5-year period and with periodic assessments across a broad range of salient domains. With regard to limitations, the study group was mostly white and did not include parental homicide, thereby limiting the generalizability of these findings. Specific to the outcome in question, we also do not have fine-grained quantitative assessments of the amount or patterns of alcohol or drug use. Finally, we were not able to conduct analyses for the subgroups of alcohol and drug abuse and dependence given the small number of incident cases in these subgroups.

We found that bereaved youth are at increased risk for ASAD over a 5-year period subsequent to the parent's death, similar to Kaplow et al.[11] However, when we controlled for DBDs and functional impairment in offspring, the effect of bereavement was attenuated and not significant. We have previously documented that functional impairment and its univariate correlates that were also associated with ASAD, namely depression and PTSD, were sequelae of parental bereavement and that functional impairment was also related to other adverse outcomes such as health risk behaviors and

Table 2. Multivariate Cox Regression of Correlates of Time to Onset of Alcohol and Substance Abuse or Dependence for Total Sample

	Hazard Ratio	95% CI	*z*	*P*
Bereaved	1.73	0.79–3.81	1.36	.17
Age ≥ 13 y at time of parent's death	6.68	3.22–13.89	5.09	< .001
Male	2.55	1.28–5.06	2.67	.007
White	0.51	0.24–1.05	−1.84	.07
Functional status[a]	0.93	0.90–0.95	−5.65	< .001
New-onset DBDs[b]	7.51	1.82–31.01	2.78	.005

[a]Lower scores indicate more impairment.
[b]Conduct and/or oppositional defiant disorder.
Abbreviation: DBD = disruptive behavior disorder.

Table 3. Multivariate Cox Regression of Correlates to Time to Onset of Alcohol and Substance Abuse or Dependence Among Bereaved Participants

	Hazard Ratio	95% CI	*z*	*P*
Accidental death[a]	0.93	0.37–2.32	−0.16	.87
Suicide[a]	1.42	0.54–3.73	0.71	.48
Age ≥ 13 y at time of parent's death	5.61	2.54–12.36	4.27	< .001
Male	2.77	1.28–6.01	2.58	.01
White	0.56	0.22–1.40	−1.25	.21
Functional status[b]	0.92	0.89–0.96	−4.69	< .001
New-onset DBDs[c]	7.70	1.59–37.36	2.53	.01

[a]Dummy variables for type of parent death. Sudden natural death is the reference category.
[b]Lower scores indicate more impairment.
[c]Conduct and/or oppositional defiant disorder.
Abbreviation: DBD = disruptive behavior disorder.

less optimal attainment of developmental competence.[1,4,14,50] Our findings that offspring depression and PTSD were associated with an increased risk of ASAD are consistent with longitudinal studies and highlight the importance of vigilance to the risk for developing ASAD once a parentally bereaved individual develops depression or PSTD.[16,17,51–53]

While the onset of DBD was among the sequelae of bereavement found in this sample, both community-based epidemiologic and preventive intervention studies find an association between parental bereavement and the onset of DBDs.[11,54] The onset of DBDs, being older than 13 years at the time of parental death, and male gender were all predictors of ASAD, regardless of bereavement status, as has been reported in multiple studies.[17,55–57] It is not the age at which the child lost his or her parent that increases the risk for ASAD, but age per se, since early adolescence is the peak age of incidence for ASAD onset; and age operated similarly in the bereaved and nonbereaved groups to increase risk for alcohol and substance abuse.[58,59] We also did not find that specific aspects of the bereavement experience, such as cause of death, circumstances of the death, or the development of complicated grief contributed to increased risk for ASAD.

Although contextual variables did not survive correction for multiple comparisons or multivariable analyses, we did find that caregiver impairment, negative life events, and sexual or physical abuse were related to offspring risk for ASAD and that family adaptability and cohesion were protective against its development. We have previously demonstrated that better caregiver functioning is protective against the development and maintenance of depression.[4] Also, impairment in the caregiver's function is known to affect parental monitoring and supervision, which in turn can lead to an increased risk for alcohol and substance abuse, whereas family adaptability can protect against ASAD.[19,60] Conversely, interventions that augment parental function and child coping have been shown to protect against both DBDs and alcohol and substance abuse.[60,61]

In summary, bereaved youth are at increased risk for developing ASAD, especially early adolescent boys with DBDs. Prevention of functional impairment through the detection and treatment of depression, PTSD, and DBDs and via improved parenting can attenuate the risk of alcohol and substance abuse in bereaved youth.[60,61] Clinicians who encounter parentally bereaved youth, especially boys older than the age of 13 and those who have symptoms of DBDs, should screen for the presence of ASAD. When youth are recently bereaved, it is important to screen for depression because it is impairing in and of itself and because untreated depression can predispose to the development of ASAD in bereaved youth.

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Editor's Note: We encourage authors to submit papers for consideration as a part of our Focus on Childhood and Adolescent Mental Health section. Please contact Karen D. Wagner, MD, PhD, at kwagner@psychiatrist.com.