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The Use of Structural Equation Modeling in Generative Research: Toward the Design of a Preventive Intervention for Bereaved Children

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Describes a generative study of processes which may lead to symptomatology in children who have experienced the death of a parent. Based on existing literature, four putative mediating variables were identified: parental demoralization, family warmth, negative family events, and positive stable family events. Structural equation modeling techniques were used to compare several potential causal models involving these variables. The results were most consistent with a model in which bereavement was not directly related to child symptomatology, but rather its effects were transmitted through these four mediational mechanisms. The implications of the results of the structural modeling for the design and evaluation of preventive interventions are discussed briefly.

Evaluation researchers reviewing a variety of areas have noted the relative infrequency with which prevention and treatment programs have developed an articulated mechanism of operation through which the intervention is expected to result in a positive outcome (Lipsey, 1990; Sechrest, West, Phillips, Redner, & Yeaton, 1979). For example, through what processes would having young elementary school children form occasional "magic circles" in school classrooms discourage substance abuse during adolescence (Moskowitz, Schaps, & Malvin, 1982)? Even in those cases in which a "theory" of the intervention exists, there may be little, if any, empirical base supporting the mechanisms proposed by the theory. At the same time, basic

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psychosocial research on the development of mental health problems is often conducted without direct concern for the later development of treatment or prevention programs. The divergent foci of these lines of research too often result in separate literatures on etiology and intervention that do not directly speak to the concerns of the other or benefit from advances in the other (Higginbotham, West, & Forsyth, 1988).

In contrast with the current typical practice, several authors in the area of prevention research have advocated the development of preventive interventions that are based on a strong empirical and theoretical foundation (Cowen, 1982; Lorion, 1983; Lorion, Price, & Eaton, 1989; Price, 1982). For example, Lorion (1983) made the following statement:

> In the absence of knowledge of a disorder's causes and/or of the individual, familial, and environmental conditions for its manifestations, the initiation of a primary prevention effort appears premature. Similarly, if one is ignorant of the preliminary manifestations of a target disorder, unable to systematically detect their presence, incapable of altering their evolution, one is unprepared to attack a problem at the secondary level. (p. 257)

These authors have offered general guidelines about the issues that need to be addressed by generative research (Cowen, 1982) that are designed to provide an empirical foundation for later interventions. They have also outlined some of the general approaches that should be considered as researchers move from problem analysis and generative research, to intervention design, to the field trial, and eventually to the dissemination of the intervention. However, to date there are few specific illustrations of any of these steps in the published literature.

The goal of the present article is to provide an illustration of how generative research can be conducted so that it can later directly inform the design and evaluation of a preventive intervention. The basic research reported here later served as a foundation for the design of a preventive intervention with bereaved children (Sandler, West, et al., 1991). The present generative research is unique in that it uses the prior literature to develop a preliminary model of the bereavement process in children which was then tested using structural equation modeling techniques.

Briefly, for readers unfamiliar with these techniques, structural equation modeling refers to a family of statistical procedures for testing whether obtained data are consistent with a theoretical model (Bentler, 1980; Bollen, 1989; Jöreskog & Sörbom, 1979; Kenny, 1979; Loehlin, 1987). They are particularly useful when the phenomenon under investigation involves a complex system of interrelationships among variables, as might be presumed to be the case in the development of symptomatology in a child following the death of his or her parent. No claim is made that these techniques by themselves "prove" that a theory is correct. Rather, these techniques are useful in de-

termining whether a theory provides a *plausible* account of the data and in identifying ways in which a theory does not fit the data and may need to be modified. A particularly valuable application of these techniques is to situations in which competing substantive theories have been offered to account for a phenomenon (Dignam & West, 1988). Substantive theories and alternative explanations based on methodological artifacts that do not provide an adequate account of the data set can be ruled out, leaving a much smaller set of plausible explanations to be investigated in future research.

The process of developing and refining structural equation models also serves an important, but often overlooked, *heuristic* role in many areas of "soft" psychology. To utilize this approach, the relationship among the constructs of a theory as well as between each construct and its measurement operations must be precisely specified. The researcher is thus forced to provide a clear and unambiguous specification of the theory (Loehlin, 1987). This clear specification of an empirically plausible theoretical model provides a strong starting point for the design and evaluation of preventive interventions.

A MEDIATIONAL MODEL OF THE DEVELOPMENT OF PSYCHOLOGICAL SYMPTOMATOLOGY

To utilize structural equation modeling techniques in our generative study, we needed to specify a model of the development of psychological symptomatology in bereaved children. Although there have been several scholarly reviews of the correlates of psychological symptomatology of bereaved children (Bowlby, 1980; Osterweis, Solomon, & Green, 1984), researchers to date have not proposed and tested models of the processes through which parental death leads to increased mental health problems in children. Consequently, it was necessary to develop our own preliminary model of these processes based on the existing literature. Below we discuss the primary model that serves as the focus of our research and the literature upon which it is based. Later in this article we discuss three alternatives to this model that are important for conceptual or methodological reasons, or both. The models address processes following the death of a parent that are potentially modifiable. Each model has somewhat different implications for the design of preventive interventions.

Our primary model specifies that parental death leads to disruptions of the postbereavement family environment and these problems in the family environment in turn lead to increased psychological symptomatology in the children. Based on a review of the existing empirical evidence and preliminary empirical work (Sandler, Gersten, Reynolds, Kallgren, & Ramirez,

1988), we identified four aspects of the bereaved child's family environment that might play such a mediational role: increased psychological symptomatology of the surviving parent, decreased stability of positive family events, decreased acceptance of the children by the surviving parent, and increased negative events in the family. Our criteria for identifying plausible mediators was that there be existing empirical evidence to support each of the causal paths in the model. That is, (a) parental death should have been shown to be associated with a disruption of this aspect of the family environment and (b) the family environment variable, in turn, should have been shown to be associated with children's psychological symptomatology.

There is considerable evidence that the death of a spouse is one of the most stressful events that can occur to the surviving spouse and often leads to increased psychological problems in the survivor (Osterweis et al., 1984). Research with nonbereaved samples has found that parental psychological symptomatology is related to increased mental health problems of children (Morrison, 1983). Research in a community sample of bereaved families has found that parental psychological symptomatology is related to a wide array of parent-reported adjustment problems in bereaved children (Van Eerdewegh, Bieri, Parilla, & Clayton, 1982).

Several studies have reported that parental death is often followed by multiple changes in the family environment (Rutter, 1966). For example, Birtchnell (1980), in a retrospective study of female psychiatric patients whose mother had died before they were 8 years of age, reported that 50% had more than one mother replacement figure and 22% had also experienced the death of their father before they reached 20 years of age. One quarter lived with their father throughout their childhood and reported having a good relationship with him. Adam (1982) found that a history of family instability following the death or divorce of parents was significantly related to suicidal ideation in a sample of college students visiting a student health service.

As in our research with children of divorce (Sandler, Wolchik, Braver, & Fogas, 1991), we conceptualized two ways in which family instability might be manifest in the family environment: (a) by increasing the occurrence of negative events and (b) by decreasing the occurrence of stable positive events in the family. Negative life events are usually assessed by summing a broad spectrum of negative experiences (Sandler & Guenther, 1985) and were seen as one way to assess the family disruptions which often follow parental death. Numerous studies of samples of nonbereaved children have reported that this total of recent stressful experiences is related to increased psychological symptomatology (Compas, 1987). Elizur and Kaffman (1982) in a prospective study of bereaved children found that recent life stressors were related to higher levels of child disturbance 18 months following the death. In contrast, stable positive events refer to re-

cent positive experiences that continue unchanged from the past. We hypothesized that such stable positive events would be disrupted following the death of the parent. In two samples of children who had recently experienced parental divorce, Sandler, Wolchik, et al. (1991) found that stable positive events were related to lower levels of symptomatology.

A robust finding in the child bereavement literature is that the quality of the relationship between the child and the surviving parent is related to the children's adjustment. Brown, Harris, and BiFulco (1986), in a retrospective study of 139 adult women had lost a mother in childhood, assessed parent–child problems as including indifference or low control. They found that 35% of the women exposed to these types of parent–child problems were depressed as adults, as compared to only 11% of those who experienced neither parent–child relationship problem. Elizur and Kaffman (1983) in a prospective longitudinal study of bereaved children found that strains in the mother–child relationship were the strongest correlates of children's psychological problems 42 months following the death.

GENERATIVE STUDY

The generative study attempted to further develop the empirical and theoretical base that would inform the later design of an intervention for bereaved children (see Sandler, West, et al., 1991). Specifically, we addressed several limitations that characterized the research on bereaved children reviewed in the previous section. First, reports had not been collected from both the bereaved child and the surviving parent to allow for a comparison of their perspectives. Second, only one study had used data from a community sample that was as representative as possible of bereaved families with children in a major metropolitan area (Van Eerdwegh et al., 1982). Third, data were collected at times more proximal to the parent's death than previous research, but some variability in the time elapsed since the death was allowed in order to permit exploration of possible temporal effects. Last, effects of the primary mediational model of the development of symptomatology in bereaved children as well as tests of three alternative models were conducted.

METHOD

Participants and Design

The full epidemiologic population, the sample used in the present study, and analyses to detect potential selection biases are described more

fully in Gersten, Beals, and Kallgren (1991). Briefly, the original epidemiologic population consisted of families having a bereaved spouse/parent and at least one bereaved child between the ages of 8 and 15 residing in Maricopa County, Arizona. Eight was chosen as the minimum age cutoff for two reasons: (a) Children at this age have developed a mature concept of death as universal and irreversible, and (b) available measures of several constructs of interest have not been demonstrated to have adequate psychometric properties for lower age groups.

We employed a variant of a matched risk and comparison group design. To select the sample of bereaved families, an initial random sample of families was selected from the Arizona state death certificate files in which an adult between the ages of 25 and 50 had died 3 months to 2 years before time of sampling, a surviving spouse was present, and the residence was located in Maricopa country which encompasses the Phoenix metropolitan area. This sample was selected subject to the restriction that the time since the death was approximately evenly (rectangularly) distributed across the 2 year period. Initial recruitment letters were sent to families; these were followed by follow-up letters and telephone calls. Of the 182 families for whom the existence of an eligible child was confirmed, 92 agreed to participate and were interviewed. The study sample did not differ from the full epidemiologic population on available demographic measures, with the exception that families having a surviving father participated at a lower than expected rate. Gersten et al. (1991) provide a detailed description and assessment of the recruitment process.

To construct the control group, an approximate 20% sample of bereaved children was continuously selected throughout the study to serve as target children to be matched. Control children were then recruited through systematic contacting of households in the same neighborhood as the target child. Potential control families were screened to eliminate families reporting parent divorce, alcoholism, or death during the target child's lifetime as well as chronic illness of the child. The control family was selected that had a child of the same gender and within 3 years of age of the study child. This selection procedure led to samples of bereaved (n = 92) and control (n = 20) families that were well matched on gender of target child, age of target child, neighborhood of residence, and the Duncan socioeconomic index of the family.[2]

[2]This study was part of a larger study focusing on the effects of parental death, parental divorce, parental alcoholism, and chronic child illness on symptomatology. Community comparison samples were selected in an identical manner and with the same exclusion criteria for each of the risk groups.

Measures

Measures of child psychopathology and family environment were collected during separate 1.5-hour interviews of the parent and the child. Demographic information including parent gender, target child gender, age of child, family SES, and ethnic background were also collected.

Child Psychological Symptomatology

Parent Reports. The Child Behavior Checklist (Achenbach & Edelbrock, 1983) was administered to the parent. Three scales were derived from this measure corresponding to the dimensions of anxiety (19 items, $\alpha = .80$), depression (17 items, $\alpha = .72$), and conduct disorder (19 items, $\alpha = .82$). Previous work by Gersten, Beals, West, and Sandler (1987) showed evidence of the convergent and discriminant validity of these measures.

Child Reports. The Child Assessment Schedule (CAS; Hodges, Kline, Stern, Cytryn, & McKnew, 1982; see also Gersten et al., 1991) was adapted to a structured interview format and shortened. Of interest here are the dimensions of anxiety (17 items, $\alpha = .70$), depression (20 items, $\alpha = .80$), and conduct disorder (15 items, $\alpha = .79$). The three dimensions were correlated (median $r = .35$), and showed similar internal consistencies and correlational structures across age and gender. In addition, the children completed the Revised Manifest Anxiety Scale (Reynolds & Richmond, 1978, $\alpha = .87$) and the Child Depression Inventory (Kovacs, 1981, $\alpha = .87$), standardized measures that have also been shown in previous research to have satisfactory psychometric properties. Since no standardized measure for child reports of conduct disorder could be located, a new measure of conduct disorder was developed. A total of 28 items ($\alpha = .86$) reflecting conduct disorder were extracted from the Child Behavior Checklist (Achenbach & Edelbrock, 1983) and adapted to be appropriate for a child respondent.

Family Environment Variables

Parent Reports. The parent completed the Family Cohesion subscale from the Family Environment Scale (FES; Moos & Moos, 1981, $\alpha = .62$). In addition, the parent completed the PERI Demoralization Scale (Dohrenwend, Shrout, Egri, & Mendelsohn, 1980), a 41-item self-report scale of nonspecific psychiatric distress ($\alpha = .93$). The parent also completed the General Life Event Scale for Children, a parent report measure of

child life events (Sandler, Miller, West, & Hepworth, 1988). This 38-item life event scale was developed to assess the significant events which occur to children. The parent rated (a) whether or not an event has occurred and then (b) whether this event has occurred more than usual, less than usual, or whether there has been no change. Measure of total negative events and stable positive events (positive events for which there has been no change in frequency of occurrence) are derived from this scale.

Child Reports. The child completed the Child Report of Parental Behavior Inventory (Schaefer, 1965), using only the 18 items that represented the child's perception of his or her acceptance versus rejection by the parent ($\alpha = .85$). Note that the parent report (FES Cohesion) was not fully parallel to the child report in that it reflected the warmth of the entire family rather than just the parent's relation with the target child. The child also completed the General Child Life Events Schedule for Children, a parallel child report form of the life event scale to that completed by the parent. Once again, two measures were derived: stable positive events and negative events. Finally, no report of parental demoralization was collected from the child.

RESULTS

Symptomatology of Bereaved and Control Children

Before examining the results of the structural equation modeling, it is important to probe the extent to which parental death is a risk factor, with bereaved children showing higher levels of symptomatology than the control children. An overview of the results for the parent and child reports for the bereaved and matched control samples are presented below (see also Gersten et al., 1991).

Based on the structured interview (CAS) with the child, bereaved children were higher in total symptomatology ($p < .05$), with the scale for conduct disorder reaching ($p < .04$) and the scale for depression approaching ($p < .07$) statistical significance. On the child self-report measures, the mean of the z scores of the measures of anxiety, conduct disorder, and depression differed significantly ($p < .05$), although only the Kovacs (1981) measure of depression approached statistical significance ($p < .08$) among the individual scales. Finally, the overall parent report measure score (Achenbach & Edelbrock, 1983) showed a significant difference ($p < .05$), with the depression scale again approaching statistical significance ($p < .07$). On all measures, the means were in a direction indicating higher levels

of symptomatology for the parental death than for the community comparison group.[3]

Testing a Model of the Development of Symptomatology

The research reviewed earlier suggested a general perspective emphasizing the role of disruptions in the child's postbereavement family environment. According to this perspective, the death of a parent was expected to result in such disruptions as (a) increased negative family events, (b) decreased stable positive family events, (c) decreased acceptance of the child by the surviving child, and (d) increased depression of the surviving parent. Each of these changes, in turn, would be expected to lead to an increase in symptomatology in the child. Thus, this perspective suggests that these disruptions following parental death mediate the effects of parental death on child symptomatology.

Although many of the individual relationships within this perspective have received empirical support, no explicit formal theory currently exists that precisely specifies the full network of relationships among these many variables. Given that there is no strong conceptual basis for expecting differential effects of each of the family environment variables on the three symptomatology constructs, we chose to initially examine a model in which parental death is assumed to cause each of the mediators. Each of the mediators (family environment, measures of parental depression, parental acceptance of children, negative events, stable positive events) was, in turn, assumed to have separate effects on each of the measures of child symptomatology (anxiety, depression, conduct disorder; see Figure 1). The parental report and child interview (CAS) and self-report measures were treated as separate indicators of the latent constructs of anxiety, depression, and conduct disorder. This procedure has the advantage of creating theoretically error-free estimates of each child's level on these constructs.[4]

Previous research comparing child and parent child reports of child symptomatology have shown correlations only in the .2 to .3 range between

[3]The statistical power of these comparisons was markedly reduced by the substantial difference in n (92 vs. 20 for the full sample) and variance on several of variables between the bereaved and the community comparison groups. Moreover, we preferred to conduct overall tests using equally weighted rather than empirically weighted composite variables under these conditions (cf. Hakstian, Roed, & Lind, 1979; Wainer, 1976). Univariate tests of risk group differences in symptomatology were conducted using all available data in the present article and listwise deletion of cases having any missing data in Gersten, Beals, and Kallgren (1991), leading to small discrepancies in the reported significance levels.

[4]Note that the failure to find differences between the bereaved and comparison samples on any of the measures of anxiety precludes the possibility of obtaining a true mediational effect for this outcome (cf. Judd & Kenny, 1981).

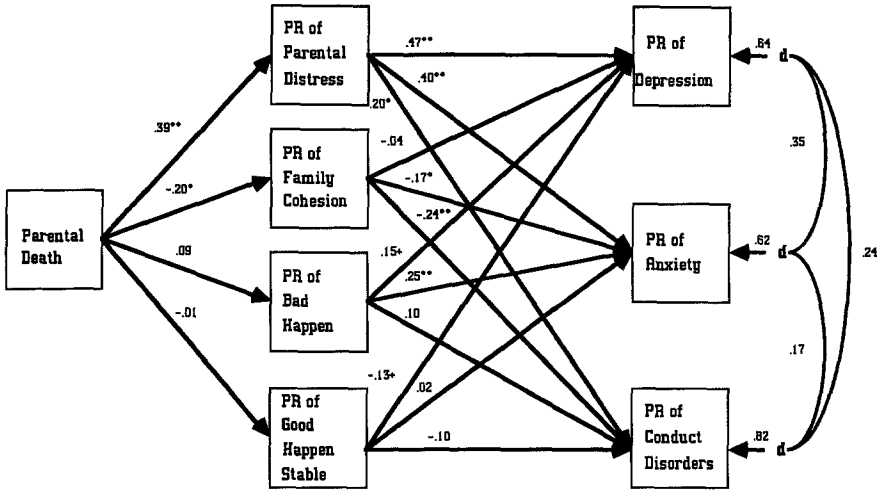

Fig. 1. Structural model showing relationships between bereavement and proposed mediators and between the mediators and symptomatology for the parent informant data. Note: ** $p < .01$; * $p < .05$; + $p < .10$, for tests of path coefficients. (Correlated disturbances between the mediators were estimated but are not shown in the figure.)

the two informants (Achenbach, McConaughy, & Howell, 1987). Our results were consistent with this finding, with a median *r* of .27 between parent and child reports of the same dimension of symptomatology. Similarly, the median *r* for the three measures of family environment on which parallel parent and child measures were obtained was .22. Separate tests were conducted for each of the cross-informant correlations for the symptomatology and family environment measures comparing (a) mothers who were above versus below the mean of the sample on the PERI demoralization measures and (b) children who were 8–12 years of age or less versus 13–15 years of age. These tests showed that the cross-informant correlations did not differ as a function of either the level of depression of the parent or the age of the child (all *p*s = ns), suggesting that these two factors are not biasing the parent and child reports, respectively. The low levels of agreement between the parent and child do, however, suggest the importance of testing the models separately for the two informants.

Parent Informant

The results of the test of the parent informant model are depicted in Figure 1. The overall fit of the model to the data was excellent, $\chi^2(3) = 0.18$, ns; Bentler and Bonnett (1980) NFI = 1.00.. Examination of the

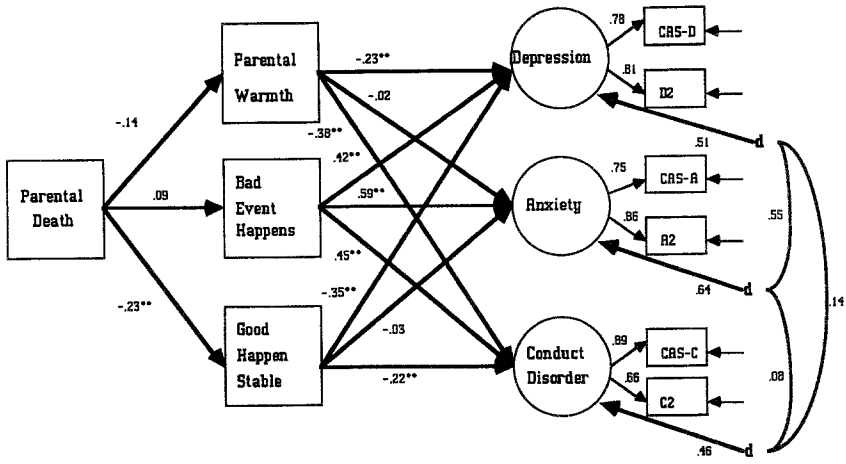

Fig. 2. Structural model showing relationships between bereavement and proposed mediators and between the mediators and symptomatology for the child informant data. Note: ** $p < .01$; * $p < .05$; + $p < .10$, for tests of path coefficients. (Correlated disturbances between the mediators were estimated but are not shown in the figure.)

standardized path coefficients between the parental death group (parental death = 1; control = 0) and the family environment measures indicated that the effects were in the predicted direction: Significant relationships were found between parental (spouse) death and the PERI measure of parental distress as well as the measure of family cohesion. The potential mediators of parental distress, family cohesion, and bad events showed several significant relationships in the predicted direction with the three parent-report measures of child symptomatology. For the parent informant, reports of stable positive events appeared to be largely unrelated to symptomatology. Finally, note that the inclusion of additional direct paths between parental death and child symptomatology does not further improve the fit of the model.

Child Informant

The results of the test of the model for the child informant are depicted in Figure 2. No measure of *parental* depression was collected from the child informant so this variable is not included in the model The overall fit of the model to the child informant data was adequate. $\chi^2(21) = 31.74$, ns; Bentler and Bonnett NFI = 0.93. Examination of the standardized path coefficients showed that the relationships between parental death and the

mediators were in the predicted direction: The relation between parental death and stable positive events attained statistical significance and the relation between parental death and child acceptance approached significance. Note that these were low power tests of the hypothesized relationships given (a) the necessary use of a dichotomous variable (parental death) and (b) the extreme split in the sample between parental death and control subjects (see Footnote 2). Examination of the standardized path coefficients between the family environment measures and the symptomatology measures indicated that all relationships were in the predicted direction, with the relationship between each of the three family environment measures and (a) depression and (b) conduct disorder attaining statistical significance. Only the relationship between negative events and anxiety was significant. Thus, the results were consistent with this portion of the hypothesized model with two exceptions: (a) negative family events appeared to play a less important role in the relation between parental death and symptomatology than was originally hypothesized and (b) the relationship between parental death and anxiety did not appear to have a clear mediational link. This latter result stems directly form the lack of difference between the bereaved and control children on child reports of anxiety. Finally, the addition of direct paths between parental death and each of the three symptomatology constructs did not further improve the fit of the model: model including direct effects, $\chi^2(18) = 29.21$; difference $\chi^2(3)$ between models = 2.53, ns.

Three Alternative Models

As noted in the introduction, structural equation modeling provides the strongest results when alternative models exist that can be competitively tested. Unlike the case in better developed literatures, such as the stress-social support literature where a set of alternative models have been fully specified (Dignam & West, 1988; Wheaton, 1985), it was necessary for us to develop alternative models that were important for either theoretical, methodological, or both reasons. Because the absence of a measure of parental depression in the child informant data precludes tests of the full model and thus makes comparative tests less informative, each of the models described and tested in this section uses only the parent report data.

Recall that for the parent data, the fit of the model depicted in Figure 1 to the parent data was excellent, $\chi^2(3) = 0.18$, ns; Bentler and Bonnett NFI = 1.00. We developed and tested three alternatives to this model. First, we tested a "common cause" model in which the measures of family environment and symptomatology are considered simply as a variety of ef-

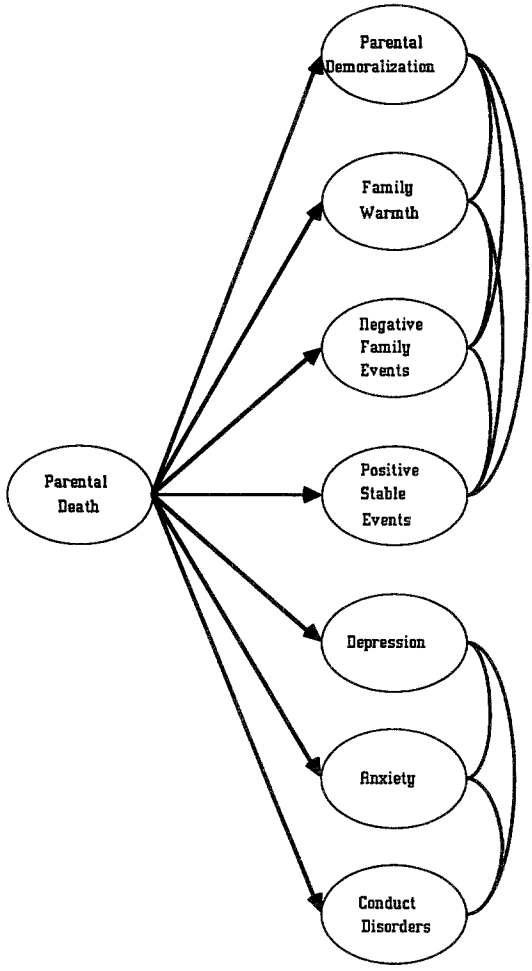

Fig. 3. Common cause model. Note: Bereavement directly causes changes in the family environment and in symptomatology with no mediation.

fects that result from the common cause of parental death. Although this model is not seriously considered in the bereavement literature, this type of model represents a pervasive type of alternative explanation that is important to rule out for methodological reasons in structural equation modeling (Duncan, 1975). A test of the common cause model depicted in Figure 3 in which correlations were allowed between disturbances within the meas-

ures of family environment and within the measures of child symptomatology showed a poor degree of fit, $\chi^2(12) = 71.98, p < .001$. Thus, this model was inadequate to account for the data.

Second, we tested an alternative model that emphasized the effects of the demoralization of the surviving parent on the family environment. According to the parent demoralization model depicted in Figure 4, the death of the spouse causes demoralization of the surviving spouse, which, in turn, leads to decreased family cohesion, increased occurrence of negative events, and decreased occurrence of stable positive events. These latter effects, in turn, lead to changes in symptomatology. In addition, the disturbances of the three family environment measures were allowed to be correlated as were the residuals of the three symptomatology measures.

This model is consistent with some previous research and is a plausible alternative to our primary mediational model. Hilgard, Newman, and Fisk (1960), for example, emphasized the role of the strong surviving parent to maintain a cohesive and protective family environment. Numerous studies report that the death of a spouse leads to increased psychological symptomatology in the surviving spouse (cf. Osterweis et al., 1984), and research has found that parental depression interferes with the quality of parenting (Billings & Moos, 1983). However, this parental demoralization model did not fit the present data nearly as well as the primary mediational model described in Figure 1, $\chi^2(9) = 34.13, p < .001$.

Finally, we tested a third alternative model in which we reversed the causal precedence between the mediators and symptomatology. According

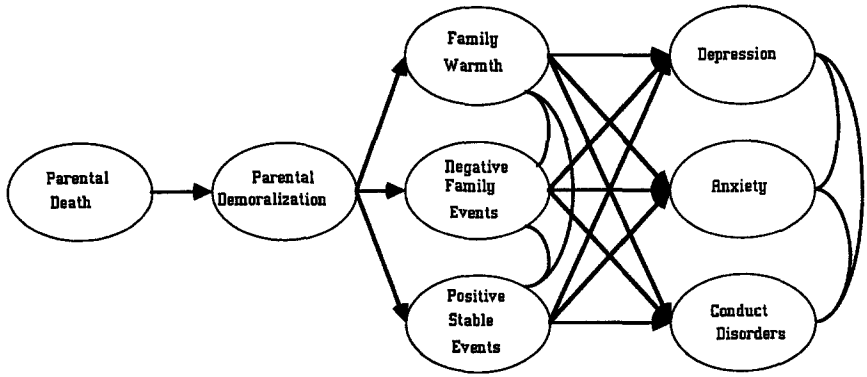

Fig. 4. Parental demoralization model. Note: Bereavement causes parental demoralization, which then causes changes in the family environment, followed by changes in child symptomatology.

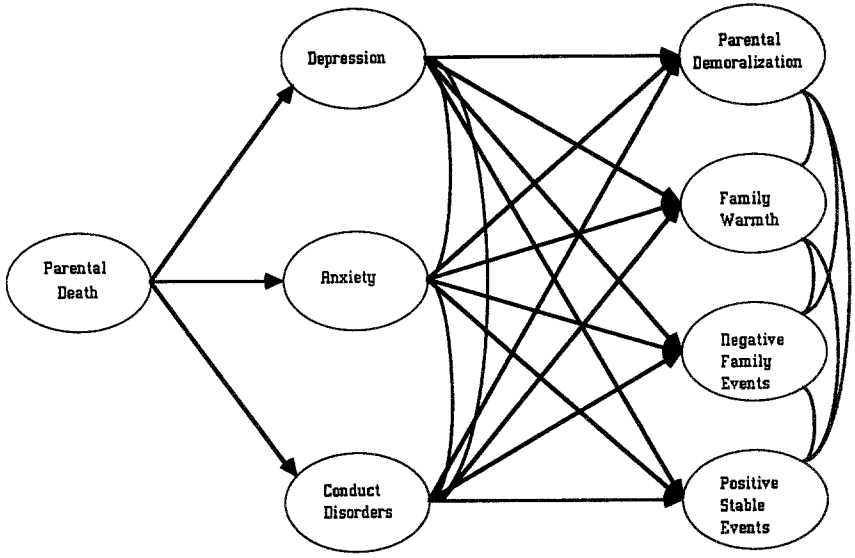

Fig. 5. Reversed causal precedence model. Note: Bereavement causes child symptomatology which then results in changes in the family environment.

to the reversed causal precedence model presented in Figure 5, parental death leads to increases in child symptomatology, which, in turn, lead to negative changes in the measures of family environment. This model is important for methodological reasons as it addresses the issue of the causal precedence of the putative mediators and the putative outcome variables. This model is also consistent with research showing that children are not simply the victims of a disturbed family environment, but they may also create problems in the family, particularly when they exhibit hyperactive or aggressive behavior. For example, Patterson (1980) has shown that children's adjustment problems can lead to poorer parent–child relations and increased parental psychological distress. However, in the present sample of bereaved families, the reversed causal precedence model failed to provide an adequate account of the data, $\chi^2(4) = 18.29, p < .001$.[5]

[5]A popular type of model in prevention research is the transactional model in which many of the variables are expected to have reciprocal effects (e.g., Sameroff, 1987). In the present case, for example, each of the family environment and symptomatology variables might be postulated to be causes of each other. Structural equation modeling techniques can be used with cross-sectional data to test some models having bidirectional relationships. However, such models must satisfy several technical requirements and assumptions before they can be estimated (see Bollen, 1989; Heise, 1975; James & Singh, 1978; Kenny, 1979). Transactional models do not meet these requirements with cross-sectional data because they postulate far too many causal paths to be estimated from the available data.

Age-Related Differences in Effects

Given that our sample of children spanned the late childhood and early adolescent years, we tested whether the effects obtained in our primary model were moderated by age of the child. We initially formed two groups by dividing the sample into children 8–12 years of age (n = 57) and children 13–15 (n = 52). Following the hierarchical testing procedure outlined by Alwin and Jackson (1981), Box's M test was initially used to compare the covariance matrices for the younger and older groups of children. For the parent informant data, Box's M was 39.57, $\chi^2(36) = 36.46$, ns, and for the child informant data, Box's M was 73.70, $\chi^2(55) = 65.50$, ns. These results indicate that there were no overall differences between the older and younger groups of children.

Post hoc examination of the covariance matrices suggested that the overall test may have failed because the set of correlations involving parental death in particular were similar in the two groups of children. We reran these overall tests with this variable dropped from the covariance matrix (i.e., including all family environment and symptomatology variables) resulting in Box's M = 33.39, $\chi^2(28) = 31.08$, ns for the parent informant and Box's M = 69.50, $\chi^2(45) = 62.51$, $p < .05$, for the child informant.

Given these *tentative* overall differences in the child informant data, a series of analyses were then performed to identify mediator–child symptomatology relations that may be moderated by child age.[6] In each regression equation, the predictor variables were the mediator (CRPBI, negative events, or positive stable events), child age (in years), and the Age × Mediator interaction. Each of the three symptomatology variables (depression, anxiety, conduct disorder) was the composite of the z-scored CAS and z-scored self-report measure (e.g., Kovacs inventory for depression) of the variable in question. The results showed that five of the nine interaction terms were at least marginally significant ($p < .10$). Specifically, these interaction terms were CRPBI × Age for Conduct Disorder, $t(109) = -2.74$, $p < .01$; Negative Events × Age for Anxiety, $t(111) = -1{,}81$, $p = .07$; Negative Events × Age for Conduct Disorder, $t(111) = 2.63$, $p < .01$; Positive Stable Events × Age for Depression, $t(111) = -1.74$, $p = .08$; and Positive Stable Events × Age for Conduct Disorder, $t(111) = -1.87$, $p = .06$. Examination of the mediator–symptom regression lines conditioned on different values of age (Aiken & West, 1991; Cohen & Cohen, 1983) showed

[6]The Alwin and Jackson (1981) hierarchical model testing procedure when applied to the reduced covariance matrix (parental death omitted) indicated that while the factor loadings were invariant across groups, the matrix of path coefficients did differ between the older and younger children.

a similar pattern of results in four of the interactions identified above. In each of these cases, the mediator–symptom relationship was in the same direction, but was higher in magnitude for the older than for the younger children. However, for the interaction of negative events and age in predicting anxiety, this pattern was reversed: The relationship was stronger for the younger than for the older children. Given that specific Age × Mediator interactions were not predicted a priori, the initial Box's M tests failed to detect significant overall differences between the covariance matrices, and that similar interactions were not obtained for the parent report data, the obtained pattern of Age × Mediator interactions must be considered to be *very tentative until they are replicated.*

DISCUSSION

The general model depicted in Figure 1 for the parent informant and the child informant version of the same model depicted in Figure 2 provides the best fit to the data. Across the analyses reported, evidence was provided in the child report data, the parent report data, or both of the importance of parental distress, family warmth, and stable positive events as potential mediators of the child symptomatology measures of depression and conduct disorder. In addition, the results show that the relationship between parental death and symptomatology is adequately accounted for by these mediational paths involving the family environmental variables. The addition of direct paths between parental death and the symptomatology constructs did not increase the adequacy of prediction.

However, the measure of negative events was unrelated to parental death in either the parent or child report data. Thus, this potential mediator becomes a clear candidate for deletion from the model, unless strong measurement or theoretical considerations dictate otherwise (Heise, 1975). In the present case, only negative events that would be common across different stress groups (see Footnote 2) and the control groups were measured. A variety of negative life events that were *specific* to the bereaved sample were also measured and found to predict symptomatology in this sample (Sandler, Gersten, et al., 1988). Hence, the particular measure utilized in this study may not adequately represent the construct of negative events for bereaved children.

It is also useful to consider the implications of the failure of the three alterative models to fit the parent informant data. First, had the model presented in Figure 3 provided a good fit to the data, the possibility that parental death leads independently to an increase in symptomatology and a disruption in the family environment would be very plausible. Such a

model would provide no basis for intervention since it provides no explanation of the *process* by which parental death leads to elevated symptomatology in the child (cf. Lorion et al., 1989). Second, had the model presented in Figure 4 provided a good fit to the data, a strong argument could be made that any intervention should be focused solely on changing parental demoralization, a mediator that occupies an early and central role in the hypothesized causal chain. Third, had the model with reversed causal precedence portrayed in Figure 5 provided a good fit to the data, interventions directed toward features of the family environment would be unlikely to have any effect. Rather, any intervention would need to be specifically directed toward reducing the child's symptoms. Thus, the success of the mediational model presented in Figure 1 argues for an intervention that addresses each of the putative mediators, with the possible exception of the occurrence of bad events.

It should be reiterated that we have only shown that the primary mediational model presented in Figure 1 provides an adequate account of the data. In a cross-sectional study, other models will always exist that can provide an equally good account of the data. Some of these models may be rejected on the basis of their inconsistency with strong theory, previous research, or measurement considerations (e.g., if death of the spouse caused *low* levels of distress in the surviving spouse). However, other good-fitting models cannot be rejected on these grounds. For example, a version of the model presented in Figure 4 in which direct paths are added (a) from parental death to family cohesion, bad events, and stable good events and (b) from parental distress to the three measures of symptomatology should provide an equally good fit to the model presented in Figure 1. The major difference between these two models is whether it is assumed that (a) the residuals of the four mediators may be correlated or (b) that parental distress, in addition to parental death, causes lower family warmth, the occurrence of negative events, and destabilizes good events, whose residuals may be correlated. Such models can potentially be clearly distinguished only in a longitudinal study, in a randomized experiment, or in a strong quasi-experiment, in those cases when these designs may be utilized (Dwyer, 1983; Gollob & Reichardt, 1987; Kenny, 1979). Note also that the differential implications of such similar models for intervention design are often minimal.

CONCLUSION

The structural equation modeling approach described above identified four potential variables that appear to mediate between parental death

and child symptomatology. Following this approach, the four variables of parental distress, family warmth, stable positive events, and possibly negative events should be targeted for change in the intervention design. Exactly how these mediators should be changed is not specified by the model; this creative process is left to the intervention designer's experience, ingenuity, and understanding of previous empirical successes in the intervention literature. However, the goal of the initial intervention is to maximize potential impact by creating confounded manipulations that are expected to change as many of the hypothesized mediating processes as possible in a positive direction (Campbell, 1987; Sechrest et al., 1979).

At the stage of the field trial of the intervention, the structural equation modeling offers a framework that is useful in the design of the evaluation. First, the critical "theoretical" components of the intervention have been clearly identified so that the quality of implementation of these components can be assessed in a process evaluation (Sechrest et al., 1979). Second, the work on the measurement of the mediators and symptomatology constructs during the generative phase greatly reduces the effort necessary to identify appropriate measures during the field trial. Finally, the structural equation model serves as the a priori "theory" of the program allowing strong tests of the mediation of program effects (Baron & Kenny, 1986; Judd & Kenny, 1981). The same model as in Figure 1 is tested, with the exception that intervention status (1 = intervention; 0 = control) replaces parental death as an exogenous variable.

Such a test of the model under interventive rather than naturalistic conditions provides an important test of the theory underlying the intervention, potentially contributing to basic psychosocial findings. The mediational analysis in combination with the process evaluation provides information about the success of the intervention in affecting each of the putative mediators and the importance of each of the mediators in producing changes in symptomatology. These analyses can point to components of the intervention that need strengthening or that appear to be superfluous to the achievement of program effects (Higginbotham et al., 1988). They can also point to places where the "theory" underlying the intervention needs reconsideration. Sandler, West, et al. (1991) present the implications of the field trial of our theory-based intervention for bereaved children.

Finally, we emphasize that our program of research is even more iterative than may be apparent from the description above. At times the structural equation modeling and the program design proceeded in parallel with the preliminary results of the modeling informing the program design and insights from the program design informing the modeling. Mediational analysis of the results of the intervention trial have led to revisions in the

model, which, in turn, will be influential in the design of a second generation program for bereaved families. Our hope is that this program of basic research and model-guided intervention development will contribute to the understanding of both the basic processes leading to the development of symptomatology in bereaved children as well as effective interventions that arrest this development.

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