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ORIGINAL PAPER

The Impact of Child Symptom Severity on Depressed Mood Among Parents of Children with ASD: The Mediating Role of Stress Proliferation

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Abstract S*tress proliferation* (the tendency of stressors to engender additional stressors in other life domains) is explored in a sample of 68 parents of children identified with ASD. Regression analyses showed that parent depression was predicted by both child symptom severity and by stress proliferation and that stress proliferation partially mediated the effect of child symptom severity on parent depression. In addition, informal social support was found to reduce levels of parent stress proliferation and parent depression; however, contrary to the stress buffering hypothesis, the ameliorative effect of support on stress proliferation was shown to be greatest when reported child symptomatology was *less* (rather than more) severe. Study implications for future research and practice are discussed.

Keywords Stress proliferation · Autism · ASD · Parent depression · Social support · Symptom severity

All families undergo experiences that tax their resources and capacity to contend with adversity. Research has shown, however, that non-normative family stressors, such as the disability of a child, can place especially severe and sustained demands on parents, placing them at risk for a variety of poor mental health outcomes (Beresford, 1994; Marsh, 1992; Seligman & Darling, 1997).

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One of the most severe disabilities affecting young children is autism, a disorder that adversely affects nearly every aspect of the child's development (Cohen & Volkmar, 1997; National Research Council, 2001; Wetherby & Prizant, 2000). Relative to other childhood disorders, autism's impact on the family appears to be particularly severe, with parents of children with autism frequently reporting high levels of stress associated with their child's social and communicative deficits, problem behaviors, and level of dependency (Bouma & Schweitzer, 1990; DeMeyer, 1979; Moes, 1995; Rodrigue, Morgan, & Geffken, 1990).

One problematic mental health outcome commonly linked to the demands of parenting a child with autism is depression (Bristol, 1987; Bristol, Gallagher, & Holt, 1993; Bristol, Gallagher, & Schopler, 1988; Dumas, Wolf, Fisman, & Culligan, 1991; Gray & Holden, 1992; Sharpley, Bitsika, & Efemidis, 1997). While research has generally found the parents of children with autism to be at increased risk for depression, compared those of typical children, marked variation in depressive symptomatology has also been noted. In light of these intra-group differences, it is important to understand why some parents of children with autism experience high levels of depressive symptomatology while others do not.

Existing research suggests that much of the stressfulness of parenting a child with autism spectrum disorder (ASD) emanates from factors directly related to the child's disability (Hastings, 2002; Koegel et al., 1992; Konstantareas & Homatidis, 1991). Children with ASD frequently exhibit a wide range of problematic and socially deviant characteristics and behaviors (Schreibman, Heyser, & Stahmer, 1999). Contending with such severe deficits and behavioral problems on a continual, daily basis clearly constitutes a major, chronic source of stress for many parents and

families (Dominic, Cutler, & McTarnaghan, 2000; Gray, 1998; Marcus, Kunce, & Schopler, 1997; Norton & Drew, 1994).

Serious stressors, however, rarely exist in isolation. Instead, sources of stress experienced in one area of life often ''spill over'' into other areas, resulting in an accumulation of multiple stressors in a variety of social spheres, a process Pearlin and his colleagues (Aneschensel et al., 1995; Pearlin, Aneschensel, & Mullan, 1997) have termed *stress proliferation*. According to Pearlin (1999), stress proliferation occurs when an initial (primary) stressor or set of stressors in one domain of life engenders additional (secondary) stressors in other life domains. Examples of the stress proliferation process abound in everyday life: problems at work may result in increased marital tensions at home (Eckenrode & Gore, 1989); separation and divorce frequently lead to financial strain and worry (Shapiro, 1996); childcare demands may exacerbate the stress experienced by working mothers as they attempt to balance work and family responsibilities (Simon, 1995). Because stressors in one area of life have the power to negatively affect activities, roles, and relationships in other spheres to which they are directly and indirectly linked, new sources of stress may be created. When such an expansion of stressors across life domains occurs, affected individuals may become enmeshed in a constellation of difficulties much more complex and severe than would have otherwise been the case had stress proliferation not taken place.

To date, the concept of stress proliferation has not been employed in the study of families of children with disabilities such as ASD. It should be noted, however, that the construct is somewhat similar to the concept of *stress pile-up* employed in McCubbin & Patterson's (1983) Double ABC-X Model of family stress. In Hill's (1958) original ABC-X Model, the characteristics of the family stressor (A), the family's crisis-meeting resources (B), and the family's definition of the stressor (C) interact, with each contributing to the prevention or precipitation of a family crisis (X). In their revised Double ABC-X Model, McCubbin & Patterson included the notion of the accumulated impact (pile-up) over time of various life stresses on family adjustment. Despite their similarity, the two concepts differ in important ways. While *stress pile-up* refers broadly to the impact of life stress *in general* on family adaptation, *stress proliferation* refers more specifically to the emergence of new sources of stress causally linked to a temporally prior, primary stressor (or set of stressors). Analytic attention to the stress proliferation process is thus important because it draws attention to the complex processes through which initial stressors expand and exert their concerted effect on psychological well-being and adjustment.

Parenting a child with ASD clearly places caregivers in a situation where stress proliferation may occur. Particularly illustrative in this regard are studies that describe the severe family disruptions frequently produced by the child with ASD (Hastings, 2002; Henderson & Vandenberg, 1992; Konstantareas & Homatidis, 1991; Moes, 1995). Research suggests that, for many parents, providing care to the child with ASD becomes a central commitment overriding and altering other life priorities (Gray, 1998). Similar to family members caring for persons with other severe disabilities and health conditions (Aneshensel et al., 1995; Marsh, 1992; Seligman & Darling, 1997), for parents of children with ASD, life may come to revolve around the disabled child, with interactions both inside and outside the home being constantly altered to accommodate their child's needs (DeMeyer, 1979; Gray, 1998; Weiss, 1991). Parents, particularly mothers, may find it necessary to reduce or end outside employment to provide care, thereby reducing family income at the same time they must contend with the high out-of-pocket costs of evaluating and treating their child's disorder (Freedman, Litchfield, & Warfield, 1995; Jarbrink, Frombonne, & Knapp, 2003). Due to the child's odd, disruptive, and unpredictable behavior, normal family activities may also be curtailed (Fox, Vaughn, Wyatte, & Dunlap, 2002) and family members may find it difficult to bring outsiders into the home, increasing the family's sense of social isolation (Koegel et al., 1992; Marcus et al., 1997). Marital discord may also result as parents disagree about how best to manage the cacophony of difficulties emanating from the child's pervasive developmental disorder (Fisman, Wolf, & Noh, 1989). Thus parents rearing children with ASD must often contend, not only with their child's communicative, social, and behavioral deficits, but also with a bevy of additional stressors arising in the wake of their child's disorder. Because many of these secondary stressors occur in key life domains, such as marriage, family, and work, they are likely to exert a major influence on parent well-being.

Use of the stress proliferation construct may help us better understand why difficulties, such as depression, occur more frequently in some parents of children with ASD than in others. While research has consistently found that the parents of more highly impaired children with ASD report higher levels of psychological distress (Bristol, 1987; Bristol, Gallagher, & Holt, 1991; Hastings, 2002; Koegel et al., 1992; Konstantareas & Homatidis, 1991), the relationship between child symptom severity and parent distress may be at least partially mediated by stress proliferation. According to Baron and Kenney (1986), a *mediator* can be thought of as a third variable that carries at least some of the effect of one variable on another. In the present case, it is suggested that stress proliferation may

mediate the relationship between child symptom severity and parent depression (i.e., rearing a child with more severe impairments increases stress proliferation, which, in turn increases parent depression). Thus, a major goal of this study was to investigate the relationship between child symptom severity, stress proliferation, and parent depression, and in particular, the possibility that the influence of child symptom severity on parent depression may be, at least in part, indirectly transmitted through stress proliferation.

Attention to the stress proliferation process may also help us take a more expansive view of the points at which social support and other psychosocial resources benefit those coping with severe stressors, such as those associated with parenting a child with ASD. While research has clearly documented the role of social support in mitigating caregiving stress (Albanese, San Miguel, & Koegel, 1995; Bereford, 1993; Boyd, 2002; Dunst, Trivette, & Jodry, 1997; Gill & Harris, 1991), the process by which support exerts its effects is less clearly understood. One theory (the ''stress buffering hypothesis'') suggests that social support's benefits occur primarily when stress is high, presumably because individuals are able to manage low levels of stress on their own (Cobb, 1976; Cohen & Willis, 1985; Turner & Turner, 1999). This theory, however, may not be applicable in situations where exposure to severe stressors limits, rather than facilitates, effective use of support (Curtrona, 1986; Morrow, Hoagland, & Carnrike, 1981; Osborne & Rhodes, 2001). Evidence suggests that the demands of parenting a severely disabled child with ASD can, at times, overwhelm and isolate parents (Fox et al., 2002; Marcus et al., 1997; Moes, 1995). Thus, under these circumstances, one might expect that the impact of social support on stress proliferation and on depression would vary depending on the severity of the child's autistic symptoms; however, in this case, the effect of social support would be most pronounced when symptomatology was *less*, rather than more, severe. Thus, a second goal of this study was to examine the potential role of social support in reducing stress proliferation and depression among the parents of children identified with ASD as well as the possibility that child symptom severity may alter (i.e., *moderate*) the impact of social support on stress proliferation and depression.

Based on the above review, a series of relationships between child symptom severity, stress proliferation, social support, and parent depression were hypothesized. Specifically, it was hypothesized: (1) that child symptom severity and stress proliferation would both be significantly associated with parent depression; (2) that stress proliferation would account for a significant increase in the explained variance in parent depression beyond that attributable to child symptom severity alone; (3) that the effect of child symptom severity on parent depression would be at least in part *indirect*, due to the mediating role of stress proliferation; (4) that the negative effect of social support on stress proliferation would be greatest when child symptom severity was *less*, rather than more, severe; and (5) that the negative effect of social support on parent depression would also be greatest when child symptom severity was *less* severe.

Methods

Participants and Procedure

The participants in this study were the parents of 68 children attending one of six public school special education programs located in the Greater Boston area. Following study approval by the University's Institutional Review Board, questionnaires and cover letters were mailed to parents' homes by participating schools, followed by a reminder letter three weeks later. Across participating schools, the parents of 110 students receiving educational services for ASD were mailed questionnaires and a response rate of 61% was obtained. In all cases, respondents identified themselves as the parent who was primarily responsible for the care of the child with ASD. Because questionnaires were returned anonymously and because researchers were not given access to school records, no information is available on non-responding parents. Data on child gender, age, and race, however, indicated that study children were demographically similar to students receiving services for ASD at participating schools.

The final parent sample consisted of 60 mothers and 8 fathers. Parent age ranged from 28 to 61 years, with a mean of 38.4 (SD = 6.6) years. In terms of education, most respondents had completed at least some college (mean years of education = 15; SD = 2.4). Total family income varied substantially within the sample, ranging from under $25,000 (13%) to over $100,000 a year (10%). Eighty-eight percent of participating parents were Caucasian, 6% were African–American, 3% were Hispanic, and 3% were of Asian descent.

In terms of child characteristics, 88% were male and 12% were female, with a mean age of 7.2 years (SD = 2.11). Thirty-four percent were reported by the parent to be diagnosed with autism, 56% with PDD-NOS, 4% with an unspecified ASD, and 6% with developmental delay. In terms of communication skills, 22% of the children were reported by the parent to be primarily nonverbal. Seventy-one percent of the children attended a fully segregated special needs program, while 29% attended either a partial or full-day inclusion program.

Instruments

As part of the study questionnaire, participating parents were asked to provide information about themselves and their family, the child with ASD, and the child's school program. Specifically, in terms of the analyses presented here, information was gathered using the following measures.

Severity of Child Autism Symptomatology

Unlike many existing psychopathology rating scales developed primarily for use with disabled persons without ASD (Aman, Singh, Stewart, & Field, 1987; Matson, Kadzin, & Senatore, 1985; Reiss, 1988), the index utilized in this study was developed to assess the severity of symptoms *deemed by parents of children with ASD* to be particularly problematic to them. Index items were selected based on the content analysis of data generated through a series of exploratory interviews with a separate sample of 22 parents of children with ASD (Benson, 2000, unpublished manuscript). The resulting index asked respondents to estimate on a 4-point scale (0 = never to 3 = often), how frequently their child currently exhibited 18 different symptoms, characteristics, or behaviors commonly observed in children with ASD, including receptive and expressive communication difficulties, hyperactivity, rapid mood swings, shrieking or screaming, tantrums, social withdrawal, lack of eye contact, self-stimulatory behaviors, repetitive behaviors, sadness or depression, sleep problems, difficulty adjusting to change, noncompliance, limited food preferences, pica, self-injury, and aggression towards others. Possible index scores ranged from 0 to 54. Cronbach's alpha for the index was 0.80, indicating good internal consistency. Criterion validity for the index was also examined utilizing data from a separate sample of 107 parents of children with ASD (Benson, Karlof, & Siperstein, 2004, submitted for publication). Employing those data, the present symptom severity index was significantly and positively correlated ($r = 0.36$; $p < 0.001$) with the *teacher-reported* Childhood Autism Rating Scale (CARS; Schopler, Reicher, & Renner, 1988), a widely used and well-validated measure of autism symptomatology.

Stress Proliferation

Stress proliferation was measured using the stress subscale of the *Effects of the Situation Questionnaire* (ESQ) developed by Yatchmenoff et al. (1998). Utilizing a 4-point scale (0 = not at all to 3 = a lot), respondents were asked to rate the degree to which 17 areas of their lives had been either limited or made more difficult *because of their child's ASD*. These areas included physical health, work, finances, relationships with a spouse, other children, family members and friends, relations between children, the respondent's view of him- or herself as a parent, family activities, social activities or hobbies, time for day-to-day activities such as shopping and preparing meals, involvement in organizations or groups, religious or spiritual life, and the respondent's sense of order or structure in his or her life. Total index scores ranged from 0 to 3 and represent the mean ratings for stressors impacting the respondent. In the present study, Cronbach's alpha for the stress subscale of the ESQ was 0.91, indicating excellent internal consistency.

Parent Social Support

A slightly modified version of *Family Support Scale* (FFS; Dunst, Trivette, & Hamby, 1994) was used to measure parent social support. Using a 5-point scale (1 = not at all helpful to 5 = extremely helpful), respondents were asked to rate each of 17 possible sources of support, including family members, friends, coworkers, other parents, teachers, and non-school professionals (one FSS item assessing the helpfulness of early intervention (EI) was not used since no children were using EI services at the time of the study). The FSS has demonstrated good reliability and validity in a variety of studies of families with disabled children (Krauss, 2000). In the present study, two types of parent social support were derived from the FSS based on scoring procedures developed by McConachie and Waring (1997). These were: (1) a weighted score for the helpfulness of *informal support* received from family, friends, parents, and other nonprofessionals, and (2) a weighted score for the helpfulness of *formal support* received from school personnel, public and private agencies, and other professionals. Subscale scores ranged from 1 to 5 and represent the mean helpfulness ratings for sources of informal and formal support available to each respondent. In the present study, both support subscales demonstrated adequate internal consistency (informal support alpha = 0.73; formal support alpha = 0.70). It is noteworthy that scores for social support subscales were only modestly correlated ($r = 0.22$; $p < 0.05$), suggesting that the two types of support represent related, but distinct, forms of family assistance.

Parent Depression

Parents' level of depressive symptomatology was ascertained using the *Center for Epidemiologic Studies-Depression Scale* (CES-D; Radloff, 1977), a self-administered questionnaire designed to measure depressive symptomatology in the general population. Using a 4-point scale (0 = rarely/none of the time to 3 = most/all of the time), respondents were asked to estimate how frequently during the past week they had experienced 20 different

depressive symptoms, including feeling sad, restless, fearful, or hopeless about the future. Total scores for the CES-D ranged from 0 to 60, with higher scores reflecting a higher degree of depressive symptomatology. The CES-D has a recommended cutoff point (a score of 16 or greater) for the identification of ''probable cases'' of clinical depression and has been shown to be a sensitive screening device for depression, even among psychiatric populations (Myers & Weissman, 1980). In this study, Cronbach's alpha for the CES-D was 0.92.

Sociodemographic Characteristics

Several parent and child sociodemographic characteristics were also included as control variables in the analysis. Parent demographics included gender, age, race/ethnicity, and years of education. Child demographics included gender and age.

Results

Descriptive Analyses

Table 1 displays descriptive statistics for each study variable. What is perhaps most striking about these findings is the high mean score reported by parent respondents on the depression index. Participating parents received a mean score of 16.4 on the CES-D, which is above the recommended cutoff used to identify probable cases of depression. Forty-five percent of parents surveyed scored above the cutoff point, suggesting that nearly half of the parent sample reported depressive symptoms severe enough to warrant a clinical diagnosis of depression. Equally noteworthy, however, is the wide variation evidenced in parents' scores on the CES-D, as indicated by the measure's high standard deviation (11.4) and observed range (0–42). Thus, while participating parents reported, on average, a high level of depression, marked within-group variation in severity of depressive symptomatology is also apparent from these data.

Table 1 Descriptive statistics of study variables

	Mean/ percentage[a]	Median	Standard deviation	Observed range
Study variables				
Parent depression	16.4	14.0	11.4	0–42
Child symptom severity	17.1	17.1	6.1	3–33
Stress proliferation	1.7	1.8	0.8	0–3
Informal social support	2.5	2.4	0.8	1–5
Formal social support	2.5	2.3	1.1	1–4.8
Control variables				
Parent age	38.4	37.5	6.6	28–61
Parent years of education	15.0	16.0	2.4	9–21
Parent race/ethnicity				
White	88.2%			
Non-white	11.8%			
Parent gender				
Male	11.8%			
Female	88.2.8%			
Annual family income				
Under $24,999	13.2%			
$25–$49,999	29.4%			
$50–$74,999	30.9%			
$75–$99,999	16.2%			
$100,000 or more	10.3%			
Child gender				
Male	88.2%			
Female	11.8%			
Child age (in months)	88.0	86.0	35.0	39–18

[a] For categorical variables only

Bivariate Analyses

The hypothesis that child symptom severity and stress proliferation would be significantly related to parent depression was tested through the use of bivariate correlations. Child symptom severity was significantly and positively correlated to both stress proliferation ($r = 0.40$, $p = 0.001$) and to parent depression ($r = 0.58$, $p = 0.000$). Thus hypothesis one was supported.

Additional correlational analyses were conducted to assess the bivariate relationships among study variables and to determine which variables to include in the regression analyses presented below. As expected, child symptom severity was found to be significantly and positively associated with stress proliferation ($r = 0.30$, $p = 0.013$). Also, as expected, informal parent support was significantly and negatively related to both stress proliferation ($r = -0.24$, $p = 0.05$) and to parent depression ($r = -0.37$, $p = 0.002$). Surprisingly, however, neither the correlation of formal parent support with stress proliferation ($r = 0.05$, $p = 0.69$) or with parent depression ($r = -0.15$, $p = 0.21$) was found to be statistically significant. Finally, only one parent sociodemographic characteristic, years of education, was significantly related to stress proliferation ($r = 0.34$, $p = 0.005$), while no parent or child sociodemographic variables were significantly associated with parent depression in the bivariate analyses.

Multivariate Analyses

Multiple regression was employed to examine the remaining five hypotheses. In each set of analyses, study data were checked diagnostically for violations of regression assumptions (normality, independence, linearity, and homoscedasticity) and no serious violations of assumptions were detected.

Hypothesis two, that stress proliferation would uniquely account for a significant increase in explained variance in parent depression beyond that attributable to child symptom severity, was tested through the use of hierarchical regression (Cohen, Cohen, West, & Aiken, 2003). To test this hypothesis, two regressions were conducted. In the first regression, child symptom severity was entered as the sole independent variable; in the second regression, stress proliferation was added to the model. As shown in Table 2, in the first regression (Model 2A), child symptom severity was found to be a highly significant and positive predictor of parent depression, accounting for 16% of the variance in the dependent variable. In the second regression (Model 2B), stress proliferation was also found to be a significant, positive predictor of parent depression, accounting for an additional 22.9% of the variance in the dependent variable beyond that explained by child symptom severity alone, a highly significant increase in R^2. Thus hypothesis two was supported.

In addition to positing a direct relationship between child symptom severity and parent depression, it was also hypothesized that at least some of child symptom severity's effect on parent depression would be *indirect*, mediated by stress proliferation. According to Baron and Kenny (1986), mediation can be said to exist if the following conditions are met: (1) the independent variable significantly affects the dependent variable in the absence of the hypothesized mediator; (2) the independent variable significantly affects the mediator; (3) the mediator exerts a significant unique effect on the dependent variable; and (4) the effect of the independent variable on the dependent variable is significantly reduced when the mediator is included in the model.

Each of these four conditions was examined in turn. As indicated in the analysis above, both child symptom severity and stress proliferation were significant predictors of parent depression. In addition, child symptom severity was found to be significant predictor of stress proliferation. Finally, as shown in Table 2, when both child symptom severity and stress proliferation were included as independent variables (Model 2B), the effect of symptom severity on parent depression was substantially smaller than when symptom severity was as the only independent variable in the model (Model 2A). Use of the ''Sobel test'' (McKinnon & Dwyer, 1993; Sobel, 1982) indicated this to be a statistically significant reduction (Z-value = 2.32, $p = 0.02$). Thus the third hypothesis, that the relationship between child symptom severity and parent depression would be partially mediated by stress proliferation, was supported.

Table 2 Hierarchical regression analyses predicting parent depression

Model/predictor	β	p	R^2	F-change
Model 2A			0.162	12.74***
Child symptom severity	0.402	0.001		
Model 2B			0.391	24.50***
Child symptom severity	0.252	0.016		
Stress proliferation	0.502	0.000		
Model 2C			0.436	5.13**
Child symptom severity	0.202	0.050		
Stress proliferation	0.472	0.000		
Informal social support	−0.222	0.027		
Model 2D			0.437	0.79
Child symptom severity	0.198	0.059		
Stress proliferation	0.481	0.000		
Informal social support	−0.225	0.027		
Child symptom severity × Informal social support	−0.028	0.780		

**$p < 0.01$, ***$p < 0.001$

The final two hypotheses posited that the effect of informal parent support on stress proliferation and on parent depression would be greatest when child symptom severity was low. In order to test the first of these hypotheses (hypothesis four), four hierarchical regressions were conducted employing stress proliferation as the dependent variable. Because of its significant bivariate correlation with stress proliferation, parent education was entered as the sole independent variable in the first regression. In the second regression, child symptom severity was added to the model, while in the third regression, informal parent support was included, along with the previous two predictors. In the fourth and final regression, the interaction term, Child symptom severity × Informal parent social support, was added to the model. As suggested by Cohen et al. (2003), all independent variables were ''centered'' over their means to control for multicollinearity between first order and interaction terms in this final model.

The results of these hierarchical regressions are presented in Table 3. As indicated by Model 3A, parent education was a significant predictor of stress proliferation, with higher levels of parent education linked to higher levels of stress proliferation. When child symptom severity was added to the model (Model 3B), it uniquely accounted for a highly significant increase in R^2 of 11% above and beyond that explained by the previous model. As shown in Model 3C, informal parent support by itself was not a significant predictor of stress proliferation. However, as indicated by Model 3D, the interaction between child symptom severity and informal support did make a significant independent contribution to the prediction of stress proliferation, uniquely accounting for an additional 7.6% of explained variance in the final model. This significant interaction term indicates that the effect of informal parent support on stress proliferation varies significantly depending on the level of child symptom severity.

In order to facilitate interpretation of this interaction, a data plot was derived based on the guidelines outlined by

Table 3 Hierarchical regression analyses predicting stress proliferation

Model/predictor	β	p	R^2	F-change
Model 3A			0.116	8.64***
Parent education	0.340	0.005		
Model 3B			0.226	9.26***
Parent education	0.370	0.001		
Child symptom severity	0.334	0.003		
Model 3C			0.228	0.20
Parent education	0.202	0.050		
Child symptom severity	0.472	0.000		
Informal social support	−0.052	0.656		
Model 3D			0.304	6.82***
Parent education	0.346	0.002		
Child symptom severity	0.334	0.004		
Informal social support	−0.022	0.845		
Child symptom severity × Informal social support	0.277	0.011		

***$p < 0.001$

Cohen et al. (2003). Figure 1 shows predicted values (derived from regression equations) for the stress proliferation score at low (1 SD below the mean) and high (1 SD above the mean) informal social support scores for each of three levels of the symptom severity score (1 SD below the mean, mean, and 1 SD above the mean). As Figure 1 indicates, for the parents of low and moderately impaired children, high informal social support decreases stress proliferation, while increasing it in the case of parents of children with high impairment. When tests were performed to assess if any of these the three slopes were different from zero, only the slope for parents with less impaired children was found to be statistically significant (*t-value* = −2.33, $p = 0.032$). Thus hypothesis four, that the effect of social support on stress proliferation would be greatest at lower levels of child symptom severity was supported (for *informal* parent support only).

The fifth and final hypothesis that social support's effect on *parent depression* would be greatest at lower levels of child symptom severity was tested by extending the hierarchical regression analyses reported in Table 2. In a third regression (Model 2C), informal social support was entered as an additional independent variable, along with child symptom severity and stress proliferation, while in a forth regression (Model 2D), the interaction term, Child symptom severity × Informal social support, was added, along with three previous independent variables (all independent variables were centered on their means in the final model). As shown in Model 2C, informal parent support accounted for a significant increment in variance of 4.5% above and beyond that explained by child symptom severity and stress proliferation alone (Model 2B). The inclusion of the interaction term in Model 2D, however, did not significantly increase the variance explained by the prior model, indicating that the significant negative effect of informal support on parent depression did not vary significantly across differing levels of child symptom severity. Thus hypothesis five was rejected.

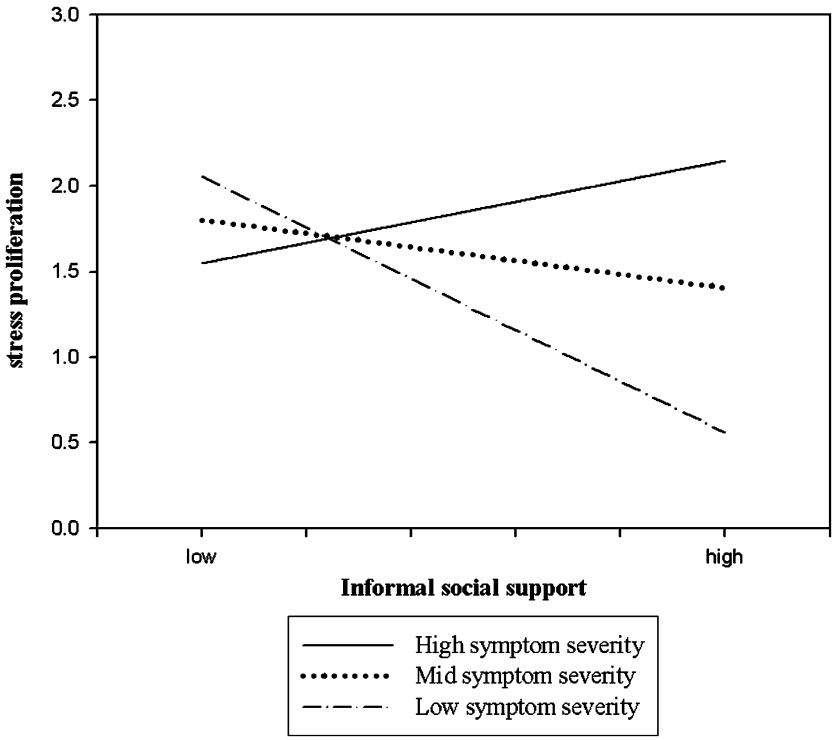

Fig. 1 Interaction between parent reports of child symptom severity and informal social support in the hierarchical regression model predicting stress proliferation

Discussion

Findings from the present study strongly suggest that raising a child with ASD can result in significant psychological distress for parents. This conclusion is supported by the fact that almost half of the parents participating in this study scored at or above the cutoff point used by the CES-D to identify likely cases of clinical depression. While average CES-D scores found in community surveys have been found to vary between 8.0 and 8.5 (Aneshensel, Frerichs, Clark, & Yokopenic, 1982), the mean CES-D score in the present sample was twice that figure (16.4).

Aside from examining parent depression itself, a major goal of the study was to call attention to the stress proliferation process as a potentially important factor contributing to parent psychological distress. Even after controlling for child symptom severity in the regression analyses, stress proliferation was found to be a powerful predictor of parent depression, uniquely accounting for a highly significant increment in the variance beyond that explained by child symptom severity alone. Regression analyses also demonstrated that stress proliferation partially mediated the relationship between child symptom severity and parent depression. Thus while some of the impact of child symptom severity on parent depression was direct, some of symptom severity's effect on depression was *indirect*, with higher levels of child symptom severity resulting in higher levels of stress proliferation, which, in turn, resulted in higher levels of parent depressive symptoms.

Despite its importance, it is important to recognize that stress proliferation may not occur in all situations and, when it does occur, that its effects on well-being may be attenuated by psychosocial resources, such as the availability of various forms of social support (Turner & Turner, 1999). In this study, *informal* parent support was found to significantly decrease parent depression. However, contrary to the stress buffering hypothesis (Cohen & Willis, 1985), informal parent support was found to have its greatest impact on stress proliferation when child symptom severity was at *lower*, rather than higher, levels. While this finding is consistent with research suggesting that the parents of severely impaired children with ASD often find it difficult to seek out and effectively use assistance from others (Fox et al., 2002), other explanations are also possible. For example, the stress buffering hypothesis may not have received an adequate test here since it could be argued that *none* of children with ASD in the present study truly displayed *low* levels of autistic symptoms (such as those one would expect in typically developing children). Therefore, those children in the present sample with less severe symptoms of ASD might be more correctly regarded as moderately impaired, while more severely affected children may be best viewed as extremely impaired. As suggested by Garmezy, Masten, and Tellegen (1984), this implies the existence of a curvilinear relationship between social support and stress proliferation, with informal support exerting a protective effect in *moderately* high-risk, but not *exceedingly* high-risk situations. As noted by Hastings (2003), this potential nonlinear relationship could be profitably explored in future research utilizing a genuine low-risk group (e.g., parents of children with no or very mild disabilities).

While informal parent support was found to play an important role in reducing both stress proliferation and depression in the present study, parent perceptions of the helpfulness of professional sources of social support was *not* found to be a significant factor in reducing either stress or depression. While in some respects counterintuitive, this finding is not surprising given inconsistent research findings regarding the impact of professional support on family well-being. While some studies have pointed to the positive effects of formal social support (Bristol, Gallagher, & Holt, 1993), other studies have suggested that increased interaction with the formal service system may not influence or, in some cases, may actually increase family stress (Brinker, Seifer, & Sameroff, 1994; Rivers & Stoneman, 2003; Shonkoff, Hauser-Cram, Krauss, & Upshur, 1992; Waisbren, 1980). There may also be methodological reasons why formal social support had no effect on parent stress or depression. Because the measure of informal social support used in this study included parent ratings of a variety of professional sources, it is possible that the ameliorative impact of *individual* sources of formal support was diluted when combined into a single measure. Additional research is needed that ''unpacks'' and individually assesses the effects of different components of formal and informal social support on family members.

While drawing needed attention to the importance of stress proliferation, social support, and depression among parents of children with ASD, the limitations of this study also need to be acknowledged. Most importantly, study results are based upon cross-sectional data and, as such, are subject to all the limitations that characterize data of this type. Most relevant here is the fact that, although the stress proliferation construct clearly posits the existence of a cause-and-effect relationship between a primary stressor (i.e., child symptom severity) and the emergence of secondary stressors, in the absence of longitudinal data, one cannot with certainty determine the direction of causality between these factors in the current study [a longitudinal study of family adaptation to ASD currently underway should provide important insight into these causal processes (Benson, Siperstein, Karlof, & Widaman, 2004)].

In addition to its use of cross-sectional data, several additional study limitations should be noted, including the relatively small size and self-selective nature of the sample.

As a result, selection biases may have operated among study participants, which could cause their responses to differ from parents who chose not to participate. In addition, study measures were based solely on parent self-report, a fact that may inflate the level of some associations between variables due to shared-method variance. It should also be noted that it is not known to what extent individual differences (such as unmeasured personality factors) may have influenced parent reports of key study variables such as depressive symptoms, stress proliferation, and social support helpfulness. Future research in this area should include measures which avoid over-reliance on one set of informants.

Contrary to a number of prior studies (Bristol et al., 1988; Olsson & Hwang, 2001; Sloper, Knussen, Turner, & Cunningham, 1991; Trute, 1995), the present study found no significant differences between mothers and fathers in regard to levels of stress or depression. Although it is possible that the inclusion of a larger subsample of fathers may have uncovered gender differences, it should also be noted that all the respondents in this study identified themselves as the primary caregiver of the child with ASD. While research has indicated that fathers generally report less stress due to their child's disability than do mothers, this may be because fathers also tend, in general, to be less involved in care of the disabled child (Konstantareas & Homatidis, 1992; Roach, Orsmond, & Barratt, 1999; Willouhby & Glidden, 1995). Fathers who assume more childcare responsibility, such as those included in this study, may be more likely to be adversely affected by their child's ASD compared to fathers who are less involved. Future research should explore this possibility further.

Findings from the current study have important implications for practice and policy. The high level of depression found in the current study suggests that parents of children with ASD are at increased risk for poor mental health outcomes, not only because of the demands of caring for their child with ASD, but because of other stressors which may be engendered or exacerbated by their child's disorder. Clinical interventions are needed that address both the *direct* and *indirect* consequences of ASD on parents and families. For example, more programs are needed which provide respite and other supportive services to parents. Counseling and mental health services geared to the unique needs of parents of children with ASD are also needed. Based on study findings regarding the importance of help from parents' informal social network, special attention in this regard should be focused on developing services that assist parents in making more effective use of existing sources of support from family members, friends, and other parents of children with ASD. Programs, such as those based on the principles of positive behavior support, may be especially beneficial since their goal is to teach parents how to reduce their child's behavioral difficulties at home and in the community (Dunlap, Newton, Fox, Benito, & Vaughn, 2001).

The key finding flowing from this study is that parent well-being can be both directly *and* indirectly affected by their child's ASD, with stressors expanding and crossing over into areas of the parent's life not directly related to their child's autistic disorder. Further research on the stress proliferation process can contribute greatly both to our understanding of stress and coping in general and to our ability to provide more effective assistance to those grappling with the multifaceted challenges of rearing a child with ASD.

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