

Adolescent Siblings of Individuals with an Autism Spectrum Disorder: Testing a Diathesis-Stress Model of Sibling Well-Being

Gael I. Orsmond · Marsha Mailick Seltzer

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Abstract The purpose of this study was to test a diathesis-stress model of well-being for siblings who have a brother or sister with an autism spectrum disorder (ASD). Data were collected from 57 adolescents and their mothers. Sisters reported higher levels of depressive and anxiety symptoms than brothers. Having a family history of ASDs was associated with depressive, but not anxiety, symptoms. A high level of maternal depression was also associated with more depressive and anxiety symptoms. A diathesis-stress model was partially supported, primarily through the findings that sibling sub-threshold autism characteristics were associated with depressive and anxiety symptoms in siblings, but only in the presence of a high number of stressful life events.

Keywords Autism · Siblings · Diathesis-stress · Well-being

Introduction

There is substantial and increasing interest in understanding the impacts of autism on family members (National Research Council 2001). Although there is considerable research on how mothers of children with autism adapt, there is comparatively little known about how siblings

adjust, especially over the life course. The current literature presents contradictory findings as to whether or not siblings of individuals with an autism spectrum disorder (ASD) are at risk of negative outcomes. In this paper, we examine the levels of depressive and anxiety symptoms in adolescent siblings who have a brother or sister with an ASD, using a diathesis-stress model (Ingram and Luxton 2005; Rende and Plomin 1992; Zuckerman 1999) to understand how genetic vulnerabilities and environmental stress may interact to place certain siblings at risk.

In a meta-analytic review on the well-being of siblings of individuals with an ASD, Yirmiya et al. (2001) concluded that there is insufficient data to support the notion that siblings of children with an ASD are at increased risk for negative psychiatric outcomes. Among the few substantive findings, the summative evidence indicated that siblings of individuals with autism were at greater risk of psychiatric conditions when compared with siblings of individuals with Down syndrome, but not when compared with siblings of individuals with psychiatric conditions or no disability. There was also clear evidence that siblings of individuals with an ASD were at greater risk of ASDs and similar sub-threshold impairments. More recent research has continued to present mixed findings. Several studies report siblings to be well-adjusted (e.g., Hastings 2003a, 2007; Kaminsky and Dewey 2002; Pilowsky et al. 2004), while others indicate increased risk of behavior problems (Hastings 2003b; Ross and Cuskelly 2006; Verte et al. 2003) and social impairment (Constantino et al. 2006) in siblings, especially in the presence of demographic risk (Macks and Reeve 2007).

The research on siblings of children with autism has not generally taken life stage into account. All of the studies that have included adolescent siblings have also included children as young as 5–8 years in the same sample (Bagenholm

G. I. Orsmond (✉)
Department of Occupational Therapy, Boston University,
635 Commonwealth Ave., Boston, MA 02215, USA
e-mail: gorsmond@bu.edu

M. M. Seltzer
Waisman Center, University of Wisconsin-Madison, Madison,
WI, USA

and Gillberg 1991; Gold 1993; Kaminsky and Dewey 2002; Mates 1990; Roeyers and Mycke 1995). Past research has not taken into account the developmental context of adolescence, and specifically the challenges to sibling well-being associated with this life stage. In general, siblings without disabilities report greater conflict in their relationship in adolescence compared with their experiences in childhood or young adulthood (Kim et al. 2006; Scharf et al. 2005).

There are some indications that the risk of psychological and adjustment problems for siblings of individuals with ASD increases during adolescence, although study findings are not consistent. Depressive symptoms and behavioral difficulties become more prevalent during adolescence in the general population (APA 2000). Behavioral and emotional difficulties during adolescence are frequently associated with the increase in normative stressors (Compas et al. 1993). With respect to siblings of children with autism, Gold (1993) found that siblings ages 7–12 reported significantly fewer depressive symptoms than siblings ages 13–17. Similarly, in a study that included children and adolescents (mean age = 9.11 years), Rodrigue et al. (1993) reported that siblings of children with autism who were younger in age were reported to have fewer internalizing and externalizing behavior problems than siblings older in age. In contrast, Verte et al. (2003) found that siblings ages 6–11 who had a brother or sister with high-functioning autism had more internalizing and externalizing behavior problems than siblings ages 12–16.

Although there is considerable evidence that adolescent girls are at greater risk of depression than adolescent boys (Allgood-Merten et al. 1990; Cicchetti and Toth 1998; Nolen-Hoeksema et al. 2006), the gender findings with respect to siblings of children with autism are inconsistent. Several studies have found no gender differences in sibling well-being (Gold 1993; Kaminsky and Dewey 2002; Mates 1990; Pilowsky et al. 2004; Rodrigue et al. 1993; Verte et al. 2003), while others have found sisters to have more favorable psychological outcomes than brothers (Ferrari 1984; Hastings 2003a; Ross and Cuskelly 2006). These studies typically have not differentiated siblings in childhood from those in adolescence, and thus the gender difference that typically emerges in adolescence (i.e., that adolescent girls are at greater risk of depression at this stage of life) may have been obscured. Thus, the first aim of our study was to describe the percent of siblings who score above clinical cut-off points on measures of depressive and anxiety symptoms and whether these percentages differ by gender of the non-disabled sibling.

Our next aim was to examine the contribution of genetic vulnerability to sibling well-being in this population. The variability noted in past research on sibling well-being (e.g., Yirmiya et al. 2001) suggests that we need to pay greater

attention to understanding factors that put siblings at greater risk or, alternatively, that serve as protective factors. One of these potential factors is the genetic predisposition siblings may have to ASDs and related impairments. There is a general consensus that there is a genetic basis to autism (see Rutter 2000; Wassink et al. 2004 for reviews). Siblings of individuals with an ASD are at greater risk for ASDs themselves as well as a range of related impairments, often referred to as the “broader autism phenotype” (BAP; Bauminger and Yirmiya 2001). Therefore, any consideration of the impact on a non-diagnosed sibling who has a brother or sister with ASD must take the BAP into account when interpreting the data, and acknowledge its potential to increase the vulnerability of siblings who do not have an ASD.

Bauminger and Yirmiya (2001) proposed that a diathesis-stress model might account for variation in sibling functioning in families of individuals with autism, with the genetic vulnerability (diathesis) interacting with environmental stress to influence sibling outcomes. This model has not been directly tested for siblings of individuals with an ASD, but has been fruitful in examining the expression and development of depression in adolescents in the general population (Brozina and Abela 2006; Lewinsohn et al. 2001) and adults with intellectual disability (Esbensen and Benson 2006). The present study adopts this transactional model that takes into account both the genetic and environmental influences operating in siblings of individuals with ASD. In our conceptualization, the diathesis was represented by genetic vulnerabilities in the form of BAP characteristics in the sibling or mother and family history of ASDs. Stress was represented by three variables that assessed environmental and family stress to the sibling: behavior problems in the brother or sister with an ASD, sibling life events, and maternal depressive symptoms.

Prior research on siblings of individuals with intellectual disability has shown that the presence of behavior problems negatively impacts sibling relationships (Greenberg et al. 1999; Seltzer et al. 1997; Orsmond and Seltzer 2007). Surprisingly, although there is considerable evidence that problematic behaviors in children with autism negatively impact parental well-being (Seltzer et al. 2001), few studies have examined this association in siblings of individuals with an ASD. While Orsmond et al. (2009) found that behavior problems in the brother or sister with an ASD were negatively associated with sibling engagement and positive affect in the sibling relationship for both adolescents and adults, Hastings (2003a) found that behavior problems in the brother or sister were not predictive of sibling behavioral adjustment for siblings during childhood or adolescence. However, more recent longitudinal research by Hastings found that earlier behavior problems in a brother or sister with a developmental disability

(including autism, Down syndrome, or intellectual disability) predicted later adjustment problems in siblings (Hastings 2007). Thus, we hypothesized that behavior problems in the brother or sister with an ASD would negatively influence sibling well-being, and we explored whether behavior problems interacted with sibling genetic vulnerabilities to predict sibling well-being.

There is considerable evidence that stressful life events are related to the development and recurrence of emotional or behavior problems in adolescents (Allgood-Merten et al. 1990; Hammen 2005; Ingram and Luxton 2005; Tiet et al. 2001). Most episodes of major depressive disorder are preceded by stressful life events (Hammen 2005). But, not everyone who experiences stressful life events develops a depressive episode. Therefore, much of the contemporary research on depression has focused on how personal vulnerabilities, most frequently in the form of a negative attributional style of thinking, interact with stressful life events toward the development of depression (Lewinsohn et al. 2001). In the current study, we anticipated that stressful life events would interact with genetic vulnerabilities (e.g., family history of ASDs, BAP characteristics in the sibling or mother) to predict higher levels of depressive and anxiety symptoms in adolescent siblings.

Moreover, having a parent with depression is a major risk factor for depression and other mental and behavioral health problems in childhood and adolescence, with both genetic and psychosocial processes likely influencing the outcome (Petersen et al. 1993). Although genetic transmission may serve as a potential mediator of childhood and adolescent depression, much research has focused on the stress associated with living with a depressed parent (Langrock et al. 2002). Depression may influence the emotional availability a parent has for the child and interactions with both children and spouse. These are factors that have been shown to impact the well-being of children and adolescents (Petersen et al. 1993). Considerable research has shown that mothers of children with autism are at increased risk for depressive symptoms (e.g., Abbeduto et al. 2004; Olsson and Hwang 2001). In the current study, we anticipated that maternal depressive symptoms would interact with genetic vulnerabilities associated with ASDs to predict sibling depressive and anxiety symptoms.

Thus, there were two research aims to this study: (a) to determine the percent of siblings who score above clinical cut-off points on measures of depressive and anxiety symptoms and whether these percentages differ by gender of the non-disabled sibling, (b) to examine how genetic vulnerabilities associated with ASDs interact with environmental stress to impact sibling well-being. Understanding these factors will help us identify the conditions under which

adolescents with a brother or sister with an ASD are at greater risk of negative outcomes.

Method

Participants

Data were collected from 57 siblings ages 12–18 during the fourth wave of an ongoing longitudinal study of families of adolescents and adults with an ASD (Seltzer et al. 2003). Families participating in the larger study were recruited via agencies, schools, diagnostic clinics, and the media. All of the individuals with an ASD in these families were over the age of ten at the start of the study and met the criteria for an ASD according to an independent diagnosis and confirmed by the Autism Diagnostic Interview-Revised (ADI-R; Lord et al. 1994). The larger study included both adolescents and adults with an ASD, so only a subset of the families included adolescent siblings at the fourth wave of data collection, which were the focus of the present analyses. Of the 85 adolescent siblings available for participation in the study, 57 participated, for a response rate of 67.1%. Eleven mothers did not want their non-disabled child to participate in the study, and 17 siblings who were contacted declined to participate in the study. The families of the 57 siblings who participated did not differ in background characteristics (e.g., income, maternal employment) from the families of the 28 siblings who did not participate, nor did characteristics of the son or daughter with an ASD (e.g., severity of autism symptoms, behavior problems) differ between these two groups. The sibling who was closest in age to their brother or sister with an ASD was recruited, while those who had significant disabilities that would have made it difficult for them to participate (such as cerebral palsy or intellectual disability) were excluded from this study.

The siblings included in this analysis ranged in age from 12 to 18 and averaged 16 years of age ($SD = 1.7$). Most (65%; $n = 37$) were sisters. Ninety percent were younger than their brother or sister with an ASD, and 74% lived with their brother or sister with an ASD. The siblings with an ASD ranged in age from 14 to 25 ($M = 19.5$, $SD = 2.5$), and 70.2% ($n = 40$) were brothers. Most of the brothers or sisters with an ASD spoke in phrases of three words or more, according to the ADI-R (70.2%), and over half (59.6%) had co-morbid intellectual disability. Families were asked to indicate an income range which best represented their income in the past year (e.g., \$0–9,999, \$10,000–19,999, etc.). Although a range of family income was represented, almost half (47.4%) of the families had an income above \$70,000 in 2004. The mothers of these children ranged in age from 39 to 58 during this round of

data collection ($M = 48.09$, $SD = 4.18$), and 78.9% were married.

Procedure

A letter describing the adolescent sibling study was sent to mothers of potential participants. If the mother was willing to have her adolescent son or daughter participate, she returned a prepared form. Trained staff then conducted 45-min phone interviews with the adolescent siblings. Following the phone interview, siblings completed a brief mailed survey. Seven siblings did not complete the mailed survey; only data from the phone survey were available for these siblings. All siblings received a small gift for participating in the study.

Data collected from mothers during the same round of data collection were also used. Mothers completed a self-report questionnaire and participated in an interview in their home or another location. The data described below comes from the self-report questionnaire. Data from seven mothers are missing as they did not complete the questionnaire.

Measures

Depressive Symptoms

Depressive symptoms in adolescent siblings and their mothers were measured with the Center for Epidemiological Studies-Depression scale (CES-D; Radloff 1977) via self-report written questionnaire by the siblings and mothers. The CES-D is a well-validated and reliable measure of depressive affect (Radloff 1991) that frequently has been used with adults (Steinhausen et al. 2006; Zimmerman and Coryell 1994) as well as adolescents (Dierker et al. 2001; Field et al. 2001; Lewinsohn et al. 2001; Schoenbach et al. 1982) and has been standardized for high school populations (Radloff 1991). Test–retest reliability, internal consistency, and concurrent validity are good (Schoenbach et al. 1982). Scores range from 0 to 60 and a score of 16 or higher is indicative of depressed mood in adults, although higher cut-off scores have been recommended for adolescents (Radloff 1977, 1991; Roberts et al. 1991). The mean for siblings in the present sample was 14.52 ($SD = 10.35$; range from 0 to 49). The mean for mothers in the present sample was 11.59 ($SD = 8.86$; range from 0 to 42). Cronbach's alpha was .91 for both siblings and mothers.

Anxiety Symptoms

Adolescents completed the Revised Children's Manifest Anxiety Scale (RCMAS; Reynolds and Richmond 1978,

1997) via written questionnaire. The RCMAS consists of 37 yes–no items and has three subfactors: physiological manifestations of anxiety, worry and oversensitivity, and problems with fear/concentration. A total score can also be calculated, with higher scores indicative of greater anxiety. Sex-specific norms are available (Reynolds and Richmond 1997) based on a sample of 5,000 school-aged children and adolescents. Numerous studies have shown the RCMAS to have good internal consistency, convergent and divergent validity with adolescent samples (Dierker et al. 2001; Muris et al. 2002). Total scores for this sample ranged from 0 to 24 with a mean score of 9.39 ($SD = 5.88$). Cronbach's alpha for this sample was .87.

BAP Characteristics in Siblings and Mothers

Mothers completed the social interaction subsection of the Development, Social Interaction, and Mood Questionnaire (DSIM; Magnusson et al. 2005) on the adolescent sibling and on herself. The DSIM was developed using adapted items from the Autism Spectrum Quotient (Baron-Cohen et al. 2001), the Social Communication Questionnaire (Berument et al. 1999) and from criteria for the BAP outlined by other researchers (Bolton et al. 1994; Le Couteur et al. 1996; Piven et al. 1997). This subsection of the DSIM contained 38 items scored on a 4-point scale (0 'definitely disagree' to 3 'definitely agree') rating social communication, rigid and repetitive behaviors, and cognitive attention (e.g., "I like getting acquainted with new people" and "I find it difficult to imagine being in the place of someone else"). Scores for siblings ranged from 3 to 58 ($M = 24.70$, $SD = 13.70$), with an alpha reliability coefficient of .90. Scores for mothers ranged from 0 to 54 ($M = 28.46$, $SD = 11.88$), with an alpha reliability of .86. Five mothers did not complete this measure regarding the non-disabled sibling who was the focus of the present study.

Family History of ASD

Mothers were queried whether anyone in their family had "problems similar to their son or daughter" with an ASD. If so, the mother was asked to describe the problem, and indicate who that person was. The mothers' descriptions were coded independently by two psychologists as to whether or not the description corresponded with a known or suspected ASD. Descriptions that were coded as positive for a family history included diagnosed ASD, suspected ASDs, and difficulties that involved significant social impairment. The two raters agreed on all but one of the cases (98% agreement). The one family on which there was disagreement reported "obsessiveness" in relatives on both the mother's and father's side and was categorized as positive for family history of ASD after group discussion.

In 22 families (39%) there was a family history of ASD according to the mother's report. In 11 of these families, the family history of ASD was on the mother's side, in 6 families it was on the father's side, and 3 families had a family history of ASD on both the mother and father's side. In two additional families, there was another child with an ASD (but no other family history of ASD).

Life Events

Siblings completed a life events checklist from the NIMH Methods for Epidemiology of Child and Adolescent Mental Disorders (Lahey et al. 1996). Twenty-five life events were queried. On average, siblings reported three of these life events in the past year ($SD = 2.3$), with a range from 0 to 10. Most frequently, siblings reported a mental or emotional problem in the family ($n = 14$), that their parents lost money ($n = 14$), that a parent started a new job ($n = 13$), that someone in their family died ($n = 13$), or that they witnessed a crime or accident ($n = 12$).

Behavior Problems in Brother or Sister with an ASD

Mothers completed the Problem Behavior Scale from the Scales of Independent Behavior-Revised (SIB-R; Bruininks et al. 1996), which consists of eight behavior problems: behavior that is hurtful to self, unusual or repetitive behavior, withdrawn or inattentive behavior, socially offensive behavior, uncooperative behavior, behavior that is hurtful to others, destructive to property, and disruptive behavior. The mother was asked whether her son or daughter with an ASD exhibited each of these eight behavior problems within the last 6 months, and if so, to rate the frequency and severity of the behaviors. Standardized algorithms (Bruininks et al. 1996) were used to translate frequency and severity ratings into a general summary score where higher scores indicated more severe maladaptive behaviors. Maladaptive behavior scores ranged from 100 to 149 ($M = 112.34$, $SD = 10.33$).

Results

Levels of Depressive and Anxiety Symptoms

Just over one-third (36%) of siblings reported depressive symptoms at or above the clinical cut-off score of 16 on the CES-D (Radloff 1977), compared with 19.2% of their mothers. Researchers have recommended different cut-off scores indicating depression for adolescents (Radloff 1991; Roberts et al. 1991; Rushton et al. 2002) and thus we calculated the percentage of adolescents falling above cut-off scores of 24 and 28 as well. Fourteen percent of siblings

scored 24 or higher on the CES-D and 10% of siblings scored 28 or higher. Sibling and mother CES-D scores were significantly though moderately correlated ($r = .39$, $p < .01$).

Siblings' scores on the RCMAS were converted to standard scores (T scores; Reynolds and Richmond 1978) based on age and gender norms; 8.5% of siblings reported clinically relevant anxiety symptoms (a total anxiety T score greater than 60). Siblings' scores on the CES-D and RCMAS were significantly and highly correlated ($r = .75$, $p < .001$). Four siblings scored 16 or higher on the CES-D and had a T score greater than 60 on the RCMAS.

We also examined whether sibling well-being varied according to whether or not they were currently living with their brother or sister with an ASD. Data were available from 40 siblings who lived with their brother or sister with and ASD and 10 who did not. The two groups did not differ significantly in depressive [$t(48) = 1.38$, $p > .05$] or anxiety symptoms [$t(48) = 1.32$, $p > .05$].

Gender Differences in Sibling Well-Being

Sisters reported significantly more depressive symptoms [$t(47) = 4.18$, $p < .001$] and anxiety symptoms [$t(48) = 2.61$, $p < .05$] than did brothers. Sisters' scores on the CES-D averaged 17.73 ($SD = 10.99$) compared with a mean score of 8.29 ($SD = 4.96$) for brothers. Sisters were also significantly more likely than brothers to score at or above the clinical cut-off score of 16 on the CES-D ($\chi^2 = 10.14$, $p < .05$). Over half (51.5%; $n = 17$) of sisters scored 16 or higher on the CES-D compared with only one (5.9%) of the brothers. Using more stringent cut-off scores, sister were also significantly more likely than brothers to score at or above a cut-off score of 24 ($\chi^2 = 4.19$, $p < .05$; seven sisters and no brothers). Similarly, six sisters and no brothers scored 28 or higher, but this difference was only marginally significant ($\chi^2 = 3.51$, $p = .06$).

Similarly, sisters' total anxiety scores on the RCMAS averaged 10.86 ($SD = 5.93$) compared with 6.53 ($SD = 4.76$) for brothers. Although 16.1% ($n = 5$) of the sisters and 6.3% ($n = 1$) of brothers scored above the clinical cut-off score on the RCMAS, this difference was not statistically different.

Diathesis-Stress Model

To test a diathesis-stress model of sibling well-being, we conducted hierarchical ordinary least squares regression models and created interaction terms to test moderator effects (Baron and Kenny 1986), mean centering all variables before multiplying them together (Aiken and West 1991), and then entering the interaction term into the regression models. We examined three variables conceptualized to represent environmental and family stress to the

sibling: behavior problems in the brother or sister with an ASD, sibling life events, and maternal depressive symptoms. We examined whether these three stress variables operated in conjunction with the diathesis measures to put siblings at greater risk for heightened depressive or anxiety symptoms. These regression models are presented in Tables 1 through 3, with Model A representing the regressions without interaction terms, Model B representing the regression for the diathesis measure of sibling BAP characteristics, Model C representing the regressions for the diathesis measure of maternal BAP characteristics, and Model D representing the regressions for the diathesis measure of family history of ASDs, for each of the three stressors. In all models, we controlled for sibling gender (1 = male, 2 = female) and whether or not the siblings lived together (residential status; 0 = live together, 1 = do not live together). Examination of residuals scatterplots from the regression equations suggested no obvious violations of the assumptions of normality, linearity, and homoscedasticity.

As shown in Table 1, with behavior problems in the brother or sister with an ASD as the measure of stress, gender and family history of an ASD were significant predictors of sibling depressive symptoms. Being female and having a positive family history of ASDs were associated with higher sibling depressive symptoms. Being female and living with the brother or sister with an ASD was associated with higher anxiety symptoms. Controlling for gender and residential status, behavior problems in the brother or sister with an ASD did not have a direct effect on

sibling depressive or anxiety symptoms, and did not interact with sibling genetic vulnerabilities to predict sibling well-being, with the exception of the interaction between Sibling BAP characteristics and behavior problems which predicted sibling depressive and anxiety symptoms at the trend level.

Table 2 presents the regression analyses with sibling life events as the stressor. Sibling BAP characteristics interacted with life events to significantly predict both depressive and anxiety symptoms. We plotted these interactions to interpret the findings, dichotomizing the variables for ease of interpretation and controlling for covariates. As shown in Fig. 1, siblings who had a high number of BAP characteristics (above the mean) and who experienced a high number of life events in the past year (five or more) reported the most depressive symptoms. The findings for sibling anxiety symptoms were similar (see Fig. 2), with siblings who reported the highest number of anxiety symptoms being those who had a high number of BAP characteristics and who experienced a high number of life events in the past year.

Finally, maternal BAP characteristics interacted with sibling life events to predict sibling depressive symptoms and this interaction is shown in Fig. 3. Siblings whose mother reported high BAP characteristics and who reported five or more life events in the past year reported the highest levels of depressive symptoms.

The regression equations for maternal depressive symptoms as the stressor are presented in Table 3.

Table 1 Regression models with behavior problems (BP) as stressor

	Sibling depressive symptoms				Sibling anxiety symptoms			
	Model A	Model B	Model C	Model D	Model A	Model B	Model C	Model D
Step 1								
Gender	.42**	.46**	.37*	.43**	.30 [†]	.34*	.25	.30 [†]
Residential status	-.20	-.24	-.19	-.20	-.28 [†]	-.33*	-.27 [†]	-.28 [†]
Diathesis								
Sibling BAP	.19	.23	.21	.20	.21	.26	.24	.21
Maternal BAP	.05	.10	.06	.01	.20	.26	.21	.20
Family history of ASD	.37*	.32*	.35*	.40*	.17	.12	.15	.16
Stress								
BP in brother/sister with ASD	.11	-.02	.08	.16	.04	-.11	.01	.03
R^2	.41**				.29 [†]			
$F(6, 31)$	3.57**				2.14 [†]			
Step 2: Interaction terms								
Sibling BAP × BP		.27 [†]				.30 [†]		
Maternal BAP × BP			.14				.15	
Family history ASD × BP				.15				-.02
Total R^2		.46**	.42*	.43*		.36*	.31 [†]	.29
$F(7, 30)$		3.69**	3.16*	3.18*		2.42*	1.94 [†]	1.78

[†] $p < .10$; * $p < .05$; ** $p < .01$

Table 2 Regression models with sibling life events (LE) as stressor

	Sibling depressive symptoms				Sibling anxiety symptoms			
	Model A	Model B	Model C	Model D	Model A	Model B	Model C	Model D
Step 1								
Gender	.40**	.36**	.45**	.39**	.29 [†]	.25	.32*	.28 [†]
Residential status	-.25 [†]	-.24 [†]	-.28*	-.21	-.30 [†]	-.29 [†]	-.32*	-.27
Diathesis								
Sibling BAP	.21	.34*	.26 [†]	.18	.22	.34*	.25	.19
Maternal BAP	.05	.12	.20	.06	.19	.26 [†]	.29 [†]	.21
Family history of ASD	.31*	.29*	.29*	.35*	.14	.12	.13	.18
Stress								
Life events	.21	.08	.16	.14	.11	-.01	.08	.05
R ²	.44**				.30 [†]			
F(6, 31)	4.01**				2.26 [†]			
Step 2: Interaction terms								
Sibling BAP × LE		.39*				.36*		
Maternal BAP × LE			.40**				.26	
Family history ASD × LE				.14				.13
R ²		.54**	.56***	.45**		.39*	.36*	.31 [†]
F(7, 30)		4.97**	5.23***	3.52**		2.74*	2.38*	1.96 [†]

[†] $p < .10$; * $p < .05$; ** $p < .01$

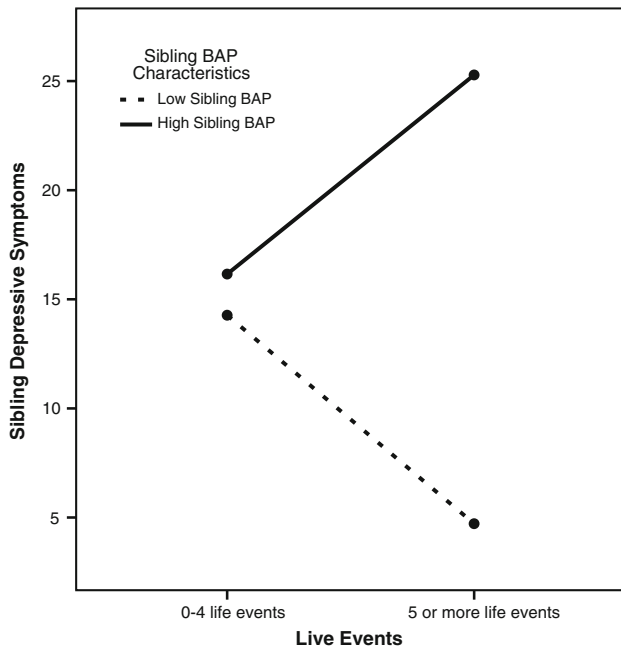


Fig. 1 The interaction of sibling BAP characteristics and sibling life events predicting sibling depressive symptoms

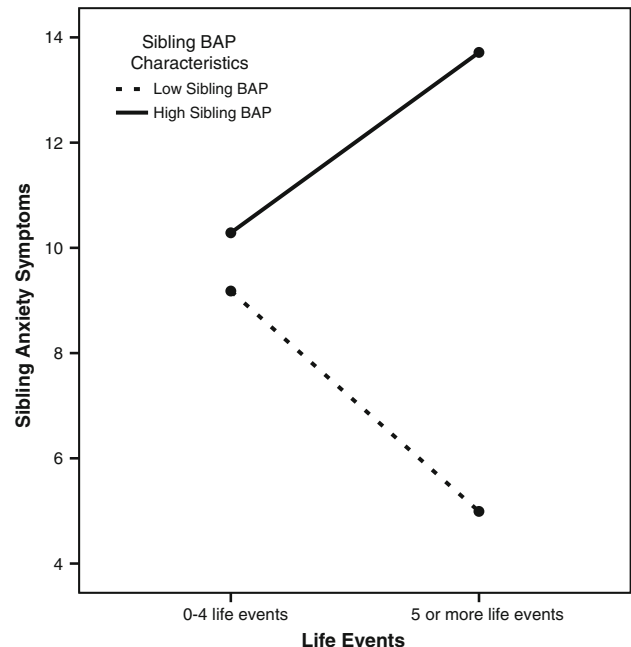


Fig. 2 The interaction of sibling BAP characteristics and life events predicting sibling anxiety symptoms

Maternal depressive symptoms had a significant direct effect on sibling depressive and anxiety symptoms. Siblings whose mothers reported more depressive symptoms reported more depressive and anxiety symptoms themselves. Maternal depressive symptoms were also

marginally significant in interaction with maternal BAP characteristics to predict sibling anxiety. Siblings whose mothers reported high BAP characteristics and high depressive symptoms reported the highest level of anxiety symptoms.

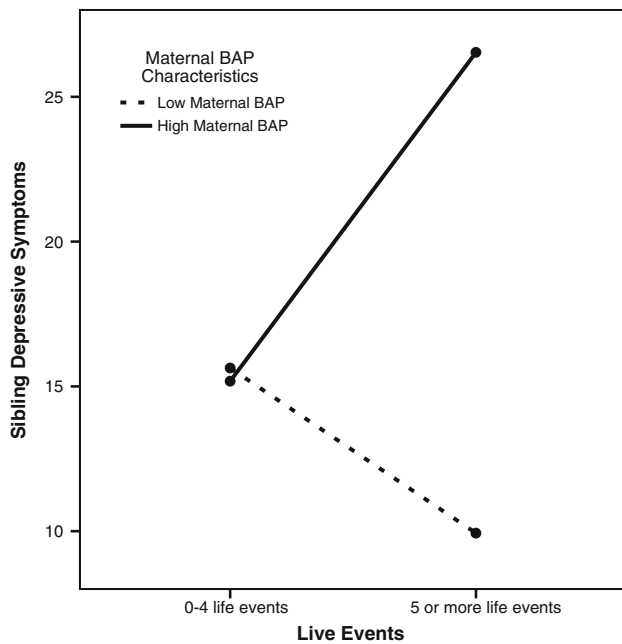


Fig. 3 The interaction of maternal BAP characteristics and life events predicting sibling depressive symptoms

Discussion

Although the stresses of raising a child with autism frequently have been described by parents and verified through research, the impact on siblings has remained unclear (e.g., Yirmiya et al. 2001). In this study, we sought

to clarify the findings by examining siblings within a narrow age range of adolescence (12–18 years of age), and by examining the contribution of genetic and environmental factors and how they might interact to put siblings at risk for negative outcomes.

Our main findings were that brothers in our sample did not report heightened depressive and anxiety symptoms than is reported in the general literature on adolescents, although sisters did report higher levels of both types of symptoms than brothers. Having a family history of ASDs was associated with more depressive, but not anxiety, symptoms. A high level of maternal depressive symptoms was also associated with both more depressive and anxiety symptoms. Moreover, our proposed diathesis-stress model was partially supported, primarily through the findings that sibling BAP characteristics were associated with depressive and anxiety symptoms in siblings, but only in the presence of a high number of stressful life events. Maternal BAP characteristics were also associated with sibling depressive symptoms in the presence of a high number of stressful life events.

In studies with community samples of adolescents, approximately half of adolescents report depressive symptoms on the CES-D above the adult cut-off of 16 (Doerfler et al. 1988; Radloff 1991; Schoenbach et al. 1982). The rates of depressive symptoms reported by siblings in the current study were considerably lower. One-third of siblings scored 16 or higher on the CES-D, and this group was largely sisters. Only 5.9% of the brothers scored 16 or higher. A similar

Table 3 Regression models with maternal depressive symptoms (MCESD) as stressor

	Sibling depressive symptoms				Sibling anxiety symptoms			
	Model A	Model B	Model C	Model D	Model A	Model B	Model C	Model D
Step 1								
Gender	.41**	.41**	.38**	.40**	.29*	.30*	.25 [†]	.31*
Residential status	-.22 [†]	-.22	-.22 [†]	-.23 [†]	-.29*	-.28 [†]	-.28*	-.27 [†]
Diathesis								
Sibling BAP	.12	.12	.14	.14	.14	.15	.18	.11
Maternal BAP	.00	.00	-.02	-.03	.15	.15	.10	.21
Family history of ASD	.36**	.36*	.29*	.35*	.18	.18	.07	.19
Stress								
Maternal CESD	.38**	.38*	.34*	.34*	.37*	.36*	.32*	.44**
R^2	.53***				.42**			
$F(6, 31)$	5.76***				3.74**			
Step 2: Interaction terms								
Sibling BAP × MCESD		.00				.03		
Maternal BAP × MCESD			.18				.29 [†]	
Family history ASD × MCESD				.10				-.18
R^2		.53**	.55**	.53**		.42*	.48**	.44**
$F(7, 30)$		4.78**	5.24**	4.91**		3.11**	4.02**	3.40**

[†] $p < .10$; * $p < .05$; ** $p < .01$

pattern was observed when we used other proposed cut-off scores. In our study, 14% of siblings scored 24 or higher, compared with 12.6% of females and 5.9% of males reported by Rushton et al. (2002). Radloff (1991) reported that 13% of junior high schools students and 18% of high school students scored 28 or above on the CES-D. In our study, only 10% of siblings scored 28 or higher on the CES-D. Thus, in our sample, sisters reported levels of depressive symptoms similar to community samples, while brothers reported considerably lower levels.

Research on general populations of adolescents has also found girls to report more depressive symptoms than boys on the CES-D (Allgood-Merten et al. 1990; Dierker et al. 2001; Doerfler et al. 1988; Roberts et al. 1990). The mean scores for sisters on the CES-D were 17.73 which is very similar to average scores reported by others for adolescents in general (Dierker et al. 2001; Doerfler et al. 1988; Monroe et al. 1999; Tolor and Murphy 1985). Brothers in the current study, however, had a mean score of 8.29 which is considerably lower than the literature suggests for adolescent males. Other researchers have reported scores for males to typically average anywhere from 11 to 16 (Dierker et al. 2001; Doerfler et al. 1988; Monroe et al. 1999; Tolor and Murphy 1985). It may be that the gender differences in depressive symptoms we observed are due to factors that account for gender differences in the general population of adolescents (e.g., differences in cognitive style, hormonal influences, genetics, or life experiences; Nolen-Hoeksema et al. 2006) but it may also be the case that sisters are uniquely impacted by the presence of a brother or sister with an ASD due to caregiving expectations or other social factors.

Fewer than 10% of the adolescent siblings in this sample reported clinically relevant anxiety symptoms on the RCMAS, based on age and gender norms. Siblings' scores on the RCMAS were also comparable to community samples (Dierker et al. 2001; Ollendick et al. 2003; Gullone et al. 2001; Muris et al. 2002; White and Farrell 2001). These studies also reported higher levels of anxiety symptoms in girls than in boys. Similar to our study, Dierker et al. (2001) found adolescents' scores on the CES-D and the RCMAS were significantly and highly correlated.

It is still not clear why some studies on siblings of children with an ASD report negative impacts on sibling well-being (e.g., Fisman et al. 1996; Gold 1993; Hastings 2003a; Rodrigue et al. 1993; Smalley et al. 1995; Verte et al. 2003), while others studies report that siblings are not at heightened risk (e.g., Ferrari 1984; Kaminsky and Dewey 2002; Mates 1990; Pilowsky et al. 2004; Ross and Cuskelly 2006). Factors that possibly contribute to these disparate findings include heterogeneity of siblings' ages in the samples, different comparison groups used, and different measures and reporters used. The studies, however,

differ on all of these factors, and it is difficult to conduct direct comparisons. Limiting our sample to adolescents may have presented a cleaner picture and revealed the gender differences in depressive and anxiety symptoms that typically emerge during adolescence. Also, as we discuss below, perhaps what is more important is to identify the mechanisms that account for this variability.

In examining alternative measures of genetic vulnerabilities, a family history of ASDs was associated with sibling depressive symptoms. Siblings from families where there was a positive history of ASDs reported more depressive symptoms than siblings without a family history of ASDs. This finding was not upheld for anxiety symptoms. As others have suggested, there may be a particular genetic link between depressive disorders and ASDs (e.g., Cook et al. 1994; DeLong 1994). Maternal depressive symptoms were also associated with higher levels of sibling depressive and anxiety symptoms. Considerable research substantiates these findings, both from a potential genetic and an environmental risk perspective (Bouma et al. 2008; Langrock et al. 2002).

Sub-threshold impairments in siblings and mothers (BAP characteristics) also were associated with sibling depressive and anxiety symptoms, but only in the presence of environmental stress. Thus, a diathesis-stress model of sibling well-being was partially supported. The clearest evidence for this model came from the findings regarding sibling BAP characteristics and life events. Siblings who had a higher number of BAP characteristics and who experienced a greater number of stressful life events in the past year reported elevated depressive and anxiety symptoms. Similar to cognitive vulnerability diathesis-stress models of depression (Lewinsohn et al. 2001), it is possible that siblings who have sub-threshold impairments of ASDs are more sensitive to stressful life events than those with few BAP characteristics. Thus, these subtle impairments associated with the BAP may have secondary consequences. Additional research with larger samples is needed to more powerfully test the means by which genetic vulnerabilities interact with life stress to predict sibling outcomes.

Further support for the diathesis-stress model of sibling well-being came from the findings regarding BAP characteristics in mothers. When the mother reported higher BAP characteristics in herself, siblings reported more depressive symptoms if they reported a high number of life events. Although we conceptualized maternal BAP characteristics as a genetic vulnerability of ASDs, and we conceptualized maternal depressive symptoms as an environmental stressor, these demarcations are only conceptual. BAP characteristics in the mother could present family stress and maternal depressive symptoms could reflect a genetic vulnerability to sibling depression. In fact, maternal and sibling depressive symptoms were significantly and moderately correlated.

Research is clear however, that maternal depression changes the family environment and exerts a negative influence on child well-being through this mechanism (Bouma et al. 2008). Moreover, the fact that we found support for our proposed model gives some credence to the way in which we conceptualized the variables. Nevertheless, the critical point is that these factors *in combination* seem to put siblings at risk for negative outcomes. Moreover, identification of such factors is relatively straight-forward and therefore siblings who may be at heightened risk for negative outcomes could be identified and screened.

This preliminary study testing a diathesis-stress model of sibling well-being is unique and potentially important, but some limitations should be noted. We employed relatively brief measures of family history and of BAP characteristics in sibling and mothers. The sibling and mother BAP measure was not correlated with a family history of ASDs, which calls into question the power of the DSIM to characterize BAP symptoms. The DSIM lacks published information about its reliability and validity, although it was developed using items from well-known and validated measures. Moreover, it was developed for use with adults and not specifically adolescents. We note, however, the high internal consistency of the DSIM social interaction scale as used in our sample, and the fact that it was not highly correlated with sibling depressive or anxiety symptoms, as partial support for reliability and validity of the measure. Another limitation is that mothers reported on their own BAP characteristics and on the sibling's BAP characteristics and they may not be able to accurately judge such characteristics, especially if they themselves are affected. Rater bias may have been introduced by this response dependency. Adolescents themselves may be more accurate reporters of their own ASD symptoms, and could be the source of such data in future research.

Using the full family history interview (Bolton et al. 1994) would provide more detailed information about family history. Also, more recently developed measures of the BAP, such as Constantino et al.'s (2003) Social Responsiveness Scale or the Broader Phenotype Autism Symptom Scale (Dawson et al. 2007) have reported reliability and validity. Also, the siblings in our sample were a somewhat select group. We excluded siblings with significant developmental disabilities, and the large majority was younger than their brother or sister with an ASD. We re-ran the analyses excluding siblings who were older than their brother or sister with an ASD; the same pattern of findings was observed. Moreover, the siblings came from families who volunteered to participate in a longitudinal study on family caregiving, and therefore may not be representative of siblings of individuals with ASDs in general.

We also likely lacked the statistical power to fully test all the interaction terms that we investigated. With our

relatively small sample size, we were limited to investigating only key diathesis and stress variables in our regression models. We did find several significantly interaction effects, however, which indicates that such a mechanism is likely present, and may be shown to be even stronger in larger samples. Furthermore, with larger samples, more complex models of sibling well-being that incorporate protective factors may be investigated.

In sum, this set of analyses indicates that sisters who have a sibling with an ASD may be at risk of heightened depressive and anxiety symptoms in adolescence. But, there are also siblings who show considerable resilience. Further research into how the genetic vulnerabilities associated with ASDs place siblings at risk is certainly needed. The use of recently developed screening measures of BAP characteristics (e.g., Constantino et al. 2003; Dawson et al. 2007) may be a first useful practical step to identifying siblings who may be at risk for negative outcomes. As a next step it will be important to identify strategies or resources that might help such siblings cope with stressful life situations. Finally, it is crucial to remember that siblings cope within the context of family relationships, and thus it will be important to examine how the sibling relationship itself, as well as family relationships more broadly, contribute to sibling well-being.

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