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Research report

Psychosocial functioning in offspring of parents with bipolar disorder

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ABSTRACT

Background: Offspring of parents with bipolar disorder are at increased risk for a range of psychopathology, including bipolar disorder. It is not clear if they also have impairments in their psychosocial functioning.
Methods: We compared the psychosocial functioning of three groups of children enrolled in the Pittsburgh Bipolar Offspring Study (BIOS): offspring of probands with bipolar disorder (n = 388), offspring of probands with other types of psychopathology (n = 132), and offspring of healthy probands (n = 118). Psychosocial functioning was assessed at study intake using the schedule of the Adolescent Longitudinal Interval Follow-Up Evaluation (A-LIFE), the Child Behavior Check List (CBCL) and the Children's Global Assessment Scale (CGAS).
Results: Offspring of probands with bipolar disorder exhibited impairments in various aspects of psychosocial functioning. On all measures, they had worse functioning in comparison with offspring of healthy probands. Offspring of probands with bipolar disorder generally exhibited more impairment than offspring of probands with nonbipolar psychopathology. After adjusting for proband parent functioning and the child's Axis I psychopathology, functioning of offspring of probands with bipolar disorder was similar to that of offspring of healthy probands.
Limitations: Data are cross-sectional and therefore do not allow for causal conclusions about the association between parental psychopathology, child psychopathology and offspring psychosocial functioning.
Conclusions: Offspring of parents with bipolar disorder exhibit impairments in psychosocial functioning which appear largely attributable to proband parent functional impairment and the child's own psychopathology. As such, interventions to improve parental functioning, as well as early interventions to treat the child's psychopathology may help reduce the risk for long-term functional impairment in offspring.

1. Introduction

Bipolar disorder (BP) is one of the leading causes of disability worldwide with lifetime prevalence in the general adult population estimated at about 4.4% (Merikangas et al., 2007). It is a chronic, devastating disorder that leads to profound impairments in nearly every domain of functioning (Judd et al., 2005; Keck et al., 1998), and impairment persists even during periods of illness remission (Fagiolini et al., 2005; Morriss, 2002; Merikangas et al., 2007).

The study of child and adolescent offspring of parents with BP is one way to study the natural history of this disorder, in terms of understanding the antecedents, progression as well as the impact of the disorder (Hammen et al., 1987; Beardslee et al., 1998). The single largest risk factor for the development of BP is a positive first degree family history of the disorder and in up to 60% of persons who develop BP onset occurs

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before age 21 (Leboyer et al., 2005; Pavuluri et al., 2005; Birmaher et al., 2007). Several recent studies report increased rates of psychopathology among offspring of parents with BP. Increased rates of problem behaviors like aggression, rule breaking and attention problems as measured by the Child Behavior Check List (CBCL) have been reported (Dienes et al., 2002; Giles et al., 2007) as well as increased risks for mood disorders, anxiety disorders, ADHD, disruptive disorders and co-morbidity (Singh et al., 2007; Pavuluri et al., 2005; Birmaher et al., 2007, 2009; Goldstein et al., 2010). In majority of BP offspring, onset of first mood symptoms has been described by early to mid adolescence (Birmaher et al., 2009; Duffy et al., 2009). Thus not only is it important to categorize symptoms and psychopathology in these high-risk offspring, it is also important to examine problems with their adaptive functioning and its determinants.

Since much of the burden associated with BP in adulthood derives from depressed mood states (Judd et al., 2005), it is likely that the literature on offspring of parents with major depressive disorder (MDD) holds important implications for the offspring of parents with BP. There is now substantial evidence showing that offspring of depressed MDD parents experience significant psychosocial difficulties in a variety of domains such as academic, peer, and family functioning as compared with healthy controls (Beardslee et al., 1998; Goodman and Gotlib, 2002; Weissman et al., 1997) which persist even in the absence of offspring psychopathology (Lewinsohn et al., 2005). This increased risk of impaired functioning among offspring of parents with MDD has been attributed in part to environmental factors such as marital and parenting difficulties, chronicity and severity of parental illness (Beardslee et al., 1998), low family cohesion and high family conflict — all of which have also been described among families in which a parent has BP (Chang et al., 2001; Romero et al., 2005; Du Rocher Schudlich et al., 2008). However, it is not clear whether offspring of BP parents are similarly impaired.

While problems with psychosocial functioning have been clearly established among youth with BP (Rucklidge, 2006; Goldstein et al., 2009), relatively few studies to date have examined social functioning in youth at risk for BP, and findings have been inconsistent. Some studies report no differences in various aspects of functioning between offspring of parents with BP and controls (Petti et al., 2004; Reichart et al., 2007; Linnen et al., 2009). Others report poorer overall functioning in offspring of parents with BP (Hodgins et al., 2002; Singh et al., 2007; Hennin et al., 2005; Ostiguy et al., 2009). Others have approached this issue by studying the premorbid functioning of adults with BP, also with varied results. Cannon et al. (1997) reported greater premorbid social impairment in adults who developed BP in adulthood as compared with healthy controls. Alternatively, Kutcher et al. (1998) reported good to excellent premorbid functioning in adolescents with BP, though the study did not employ a control group.

Limitations of most of the studies in this area to date include small sample sizes, measurement of social functioning limited to specific domains, and in some cases, samples that do not control for comorbid psychopathology. We therefore aim to evaluate the psychosocial functioning of offspring probands with BP as compared with offspring of probands with non-BP psychopathology and healthy probands using a large, well-characterized sample and multiple assessments of psychosocial functioning.

Based on the existing literature that shows that persons with BP suffer multiple impairments in daily life, and that offspring of parents with BP are at increased risk of not only BP but other disorders as well, we hypothesized that: 1) offspring of probands with BP will display greater impairment in every domain of psychosocial functioning examined (work, recreation, satisfaction, and interpersonal) as compared with offspring of healthy probands; 2) psychosocial impairment across domains will also be greater in offspring of probands with BP as compared with offspring of probands with other forms of psychopathology; and 3) psychosocial impairment in offspring of probands with BP will be largely attributable to offspring psychopathology and thus functioning among healthy offspring of probands with BP will be similar to healthy offspring of controls.

2. Methods

2.1. Subjects

The methods for BIOS have been described in detail elsewhere (Birmaher et al., 2009). Briefly, probands with BP were recruited through advertisement (53%), adult BP studies (31%), and outpatient clinics (16%). Probands were required to fulfill the Diagnostic and Statistical Manual, Version-IV (DSM-IV) criteria for BP-I or -II. Exclusion criteria included current or lifetime diagnoses of schizophrenia, mental retardation, mood disorders secondary to substance abuse, medical conditions, or medications, and living more than 200 mi away from Pittsburgh. With the exception of children who were unable to participate (e.g., mental retardation), all offspring aged 6 through 18 years from each family were included in the study. Control probands consisted of healthy probands and probands with non-BP psychopathology from the community and were group matched by age, sex and neighborhood using the area code and first 3 digits of the telephone number and zip code of the parents with BP.

2.2. Procedures

2.2.1. Proband clinical and demographic factors

The sample consists of 388 offspring of 233 probands with BP, 132 offspring of 77 control probands with a lifetime diagnosis of a non-BP Axis I disorder, and 118 offspring of 65 control probands without a lifetime Axis I diagnosis. One control family from the original sample was removed because the parent developed BP.

After receiving Institutional Review Board approval and obtaining consent from parents and assent from children, parents were assessed for psychiatric disorders, family psychiatric history, and other demographic and clinical variables. DSM-IV lifetime psychiatric disorders for proband parents were ascertained through the Structured Clinical Interview-DSM-IV (SCID) (Spitzer et al., 1992) plus the ADHD, disruptive behavior disorders (DBD) and separation anxiety disorder (SAD) sections from the Schedule for Affective Disorders and Schizophrenia for School-Age Children, Present and Lifetime Version (K-SADS-PL) (Kaufman et al., 1997). Proband

functioning was assessed using the Global assessment of functioning (GAF) scale which measures social, occupational and overall functioning on a scale of 1–100. Sociodemographic data including marital status at intake were assessed with a general information form. Socioeconomic status (SES) was ascertained using the Hollingshead scale (Hollingshead, 1975).

2.2.2. Offspring diagnoses and functioning

Parents were interviewed about their children and the children were directly interviewed for the presence of lifetime psychiatric symptoms using the K-SADS-PL. All diagnoses were made using the DSM-IV criteria. The Petersen Pubertal Developmental Scale (PDS) (Petersen et al., 1988) was used to evaluate pubertal development.

Offspring psychosocial functioning at intake was assessed using three scales — the Psychosocial Functioning Schedule of the Adolescent Longitudinal Interval Follow-Up Evaluation-Baseline (A-LIFE) (Leon et al., 1999), the Child Behavior Check List (CBCL) (Achenbach, 1991), and the Children's Global Assessment Scale (CGAS) (Schaffer et al., 1983). The A-LIFE measures very specific areas of functioning (interpersonal, academic, work, and recreation) while the CGAS measures global overall functioning on a health-illness continuum and both are interviewer rated. The CBCL was included as a comparison report measure of parents about their offspring's abilities, and social and school functioning.

Adolescents were interviewed about their own functioning using the A-LIFE, and parents were interviewed about their child. For younger subjects (<12), the child and parent were interviewed together. Summary ratings were then assigned for each item. The instrument examines functioning in 4 domains: 1) work (including employment, academic, and household), 2) interpersonal relations (including relatives and friends), 3) recreational activities and hobbies (e.g., reading, spectator or participant sports, listening to music, socializing, and community organizations), and 4) global satisfaction. Ratings reflect functioning during the worst week of the preceding month, as follows: 1 (very good), 2 (good), 3 (fair/slightly impaired), 4 (poor/moderately impaired), and 5 (very poor/severely impaired). The total instrument score is the sum of the impairment scores in each of the 4 domains (for work and interpersonal relations, the most impaired sub-domain score is used to calculate the total), and ranges from 4 to 20. Intraclass coefficient (ICC) for all domains of the A-LIFE computed in the context of another large naturalistic pediatric study conducted by our group ranged from 0.81 to 1.00. This was calculated by using Shrout–Fleiss method to compute ICCs for minimum and maximum scores over 6–7 months. The average of the ICCs was taken as the final instrument reliability.

Interviewers also rated the child's global functioning using the CGAS which can be measured in 3 domains: current (within the last month), most severe past (lifetime) and highest past year. Reliability and validity of this scale have been established (Schaffer et al., 1983). Scores above 90 indicate superior functioning; scores between 70 and 90 indicate good functioning in all areas; scores between 50 and 70 indicate impairments in one or more areas while scores below 30 indicate inability to function in most situations and need for supervision to ensure safety.

Finally, parents completed the CBCL (1991 version) which is a questionnaire to be completed by parents of 6–18 year olds on their child's behavioral and emotional problems, and competence. In this study, we report on the competence section. Both the behavior problem scale and the social competence scale have been shown to have adequate test–retest reliability and to significantly discriminate between clinic referred and nonclinic referred children. The 20 competence items of the CBCL obtain parental reports of the amount and quality of their children's participation in sports, hobbies, games, activities, jobs and chores, and friendships; how well the child gets along with others and plays alone; and school functioning. Competence items can be scored on four scales: Activities, Social, School and Total competence. Total competence comprises the sum of the three specific scale scores and higher scores indicate more favorable functioning. Raw scores were used for analyses in this study.

Completion rates for the 3 psychosocial functioning instruments used were as follows: 97% (A-LIFE), 90% (CBCL) and 95% (CGAS). The lower response rate on the CBCL as compared to the other instruments may be due to the fact that parents complete this questionnaire and it is not clinician-administered. There was moderate to large correlation ($r = 0.3–0.5$ — all p-values < 0.01) between the instruments used to assess offspring psychosocial functioning (i.e., current CGAS, most severe past CGAS, A-LIFE, and CBCL).

Bachelor's- or Master's-level interviewers completed all assessments after intensive training for all instruments and after ≥80% agreement with a certified rater. The overall SCID and K-SADS K statistics for psychiatric disorders were ≥0.8. To ensure blindness to parental diagnoses, the interviewers who met with the parent to assess parental psychopathology were different from the interviewers who assessed their children. All data (adult, child, and family) were presented to a child psychiatrist who was also blind to the psychiatric status of the parents for diagnostic confirmation.

2.3. Data analysis

The demographic and clinical characteristics and psychosocial measures among the three groups of offspring were compared using parametric and nonparametric methods as appropriate. Similar methods were used to analyze the demographic and clinical characteristics of the biological parent probands. Using the SAS 9.2 software, two level mixed effects models were developed in a hierarchical manner for each of the psychosocial functioning scores to compare groups of factors that influence the outcomes. The models included random intercept to allow the means to vary randomly across families with variance components as the structure for the covariance matrix and restricted maximum likelihood (REML) as the estimation method. The intraclass correlation coefficients (ICCs) for variance attributable to intrafamilial correlation range from 0.07 (A-LIFE Recreation score) to 0.48 (CBCL Activities score), with a mean ICC of 0.22 across measures. Therefore we adjusted first for intra-family correlations, then for significant demographics, then probands' functioning as measured by the Global assessment of functioning (GAF) scores, and finally offspring's clinical characteristics (offspring diagnoses were dichotomized as present/absent). No further post hoc adjustments for multiple

comparisons were applied (all tests were 2-sided with a significance level set at 0.05).

3. Results

3.1. Proband demographics

The socio-demographic data of probands in the study has been described in detail elsewhere (Birmaher et al., 2009). Briefly, 80% of probands with BP were female (mothers), and significantly less likely to be married (48.5%) than probands with non-BP psychopathology (55.1%) or healthy probands (78.5%; $p<0.001$). They were also significantly more likely to be Caucasian (88.4%) than probands with non-BP psychopathology (73.1%) or healthy probands (78.5%; $p<0.05$).

3.2. Proband clinical characteristics

Of the probands with non-BP psychopathology, 50.6% had lifetime rates of MDD, 37.7% had anxiety disorders, 50.6% had substance use disorders, 14.3% had DBD, 5.2% had ADHD and 3.9% had eating disorders. 88.4% of probands with BP had at least one lifetime comorbid non-BP disorder. Probands with BP also had worse current and past social functioning than the other two groups (most severe past GAF scores: probands with BP (33.2 ± 13.6) vs. probands with non-BP psychopathology (59.7 ± 11.5) vs. healthy probands (78.3 ± 9.7) $p<0.001$; current GAF scores were: probands with BP (61.9 ± 12.5) vs. probands with non-BP psychopathology (77.4 ± 10.9) vs. healthy probands (87.5 ± 7.0) [$p<0.001$]).

3.3. Offspring demographics

There were no significant age or sex differences between the 3 offspring groups. The offspring of probands with BP were significantly more likely to be Caucasian and less likely to be living with both parents than the other 2 groups (all $p<0.05$).

3.4. Offspring psychopathology

As compared with the other two groups, offspring of probands with BP had higher rates of nearly every lifetime Axis-I disorder examined (BP spectrum disorder, depression, anxiety, ADHD, and DBD) (all p-values <0.05; see Table 1).

3.5. Offspring psychosocial functioning

We first examined whether any of the measures of psychosocial adjustment were related to offspring age and sex. Older age was associated with worse satisfaction, recreation, and work subscale scores on the A-LIFE, and better activities and social subscale scores on the CBCL (all p-values <0.05). Female sex was associated with better scores on work (A-LIFE) and school (CBCL) subscales (p-values <0.05). These covariates were adjusted in the final model.

3.5.1. A-LIFE

Between-group differences in psychosocial functioning are presented in Table 2. The average A-LIFE scores for all groups were in the range of excellent to mildly impaired. Overall, there were significant differences between groups, with effect sizes in the small to medium range. Pair-wise comparisons showed that offspring of probands with BP on average had significantly higher scores on three subscales (satisfaction, recreation and interpersonal) and the total score as compared to the other two groups, indicating worse functioning. On the work subscale, offspring of probands with BP had worse functioning in comparison with the offspring of healthy probands but not in comparison with the offspring of probands with non-BP psychopathology. The

Table 1
Demographic and clinical characteristics in offspring of bipolar probands, offspring of probands with nonbipolar psychopathology and offspring of healthy probands.

	Offspring of probands with BP	Offspring of probands with non-BP psychopathology	Offspring of healthy probands	Statistic	p-value
	n = 388	n = 132	n = 118		
Age (mean ± SD), years	11.9 ± 3.6	11.8 ± 3.7	11.7 ± 3.4	$F=0.19$	0.82
Sex (female %)	48.5% (188)	54.5% (72)	54.2% (64)	$\chi^2=2.15$	0.34
Tanner stage % (IV or V) [a]	52.1% (126)	55.3% (42)	45.9% (34)	$\chi^2=1.4$	0.5
Race (white %)	81.4%[a] (316)	70.5%[b] (93)	78.8%[ab] (93)	$\chi^2=7.09$	0.029
SES (mean ± SD)	34.7 ± 14.4	35.4 ± 12.8	38.8 ± 12.2	$F=2.3$	0.1
Living with both biological parents % (yes)	42%[a] (163)	53%[b] (70)	71.2%[c] (84)	$\chi^2=31.5$	<0.001
BP spectrum	10.3%[a] (40)	0.8%[b] (1)	0.8%[b] (1)	$\chi^2=22.35$	<0.001
BP 1	2.3% (9)	0% (0)	0% (0)	$\chi^2=5.88$	0.053
BP 2	1.3% (5)	0.8%(1)	0% (0)	$\chi^2=1.67$	0.43
BP-NOS	6.7%[a] (26)	0%[b] (0)	0.8%[b] (1)	$\chi^2=15.0$	0.001
Depression	10.6%[a] (41)	6.1%[a] (8)	0.8%[b] (1)	$\chi^2=12.56$	0.002
Anxiety	25.8%[a] (100)	13.6%[b] (18)	7.6%[b] (9)	$\chi^2=22.79$	<0.001
ADHD	24.5%[a] (95)	21.2%[a] (28)	11%[b] (13)	$\chi^2=9.77$	0.008
DBD	19.1%[a] (74)	10.6%[b] (14)	5.1%[b] (6)	$\chi^2=16.35$	<0.001
SUD	4.1% (16)	3.8% (5)	1.7% (2)	$\chi^2=1.55$	0.46
Any Axis I disorder	59.8%[a] (232)	43.2%[b] (57)	24.6%[c] (29)	$\chi^2=47.84$	<0.001

Abbreviations: BP, bipolar disorder; ADHD, attention deficit hyperactive disorder; DBD, disruptive behavior disorders; SUD, substance use disorders; and SD, standard deviation.
All disorders are lifetime.
Different superscripts indicate significant pair-wise comparisons with $p<0.05$.
[a] A Tanner stage of I indicates prepubertal, II to III, midpubertal; and IV to V, postpubertal.

offspring of probands with non-BP psychopathology had significantly worse functioning on the work subscale and total scores as compared with the offspring of healthy probands (for all above noted comparisons p-values<0.05).

3.5.2. CBCL

Overall there were significant differences between groups on all subscales of the CBCL, with effect sizes in the small range. Pair-wise comparisons showed that offspring of probands with BP had significantly lower scores on all subscales (Activities, Social and School) than the offspring of healthy probands. There was no significant difference in functioning between offspring of probands with BP and offspring of probands with non-BP psychopathology on all CBCL subscales. Offspring of probands with non-BP psychopathology had significantly lower scores than the offspring of healthy probands on the school and total scales of the CBCL (for all above noted comparisons p-values<0.05).

3.5.3. CGAS

Overall, there were significant differences between groups on the CGAS, with medium to large effect sizes. Pair-wise comparisons showed that offspring of probands with BP had significantly lower scores (current, highest past, and most severe past) than the other 2 groups. Offspring of probands with non-BP psychopathology also had significantly lower scores than the offspring of healthy probands on the current and most severe past domains of the CGAS (for all above noted comparison all p-values<0.05).

3.6. Adjustment for demographic and clinical variables

Given that there were demographic and proband GAF score differences between the three groups, we next examined a model in which we incorporated 4 levels of adjustment: Level 1) we adjusted for intra-family correlations; Level 2) we adjusted for significant demographics (age, gender, race, SES, proband's marital status, and offspring living with both biological parents); and Level 3) proband current and most severe past GAF scores. Differences between offspring of probands with BP and healthy probands remained significant only on the satisfaction (R-squared associated with adjustment for demographic and proband functioning = 0.0057) and total A-LIFE (R-squared = 0.005), and global functioning (all CGAS domains; R-squared from 0.007 to 0.01). Further adjusting for Level 4) offspring psychopathology revealed no further differences between offspring of probands with BP and offspring of healthy probands (R-squared associated with additional adjustment for offspring psychopathology ranges from 0 for satisfaction to 0.009 for work-total). At Level 4 adjustment, lower SES and presence of offspring lifetime disorder remained significantly associated with worse functioning as measured on the A-LIFE, CBCL and current CGAS across groups (all p<0.05). Older age also remained significantly associated with worse functioning across groups as measured on the A-LIFE (p<0.05).

Psychosocial functioning scores were also compared between healthy offspring of probands with BP and healthy offspring from the other two groups after adjusting for intra-family correlations (Level 1) and significant demographics (Level 2). Group differences remained significant on the satisfaction and work subscales (A-LIFE) and all domains of the CGAS (all p<0.05).

4. Discussion

Our study aimed to compare the psychosocial functioning in offspring of probands with BP and offspring of controls. Controls were further divided into offspring of probands with non-BP psychopathology and healthy probands to assess for specific effects of parental BP as compared with

Table 2
Mean ± SD scores for psychosocial functioning in offspring of bipolar probands, offspring of probands with nonbipolar psychopathology and offspring of healthy probands.

	Offspring of probands with BP	Offspring of probands with non-BP psychopathology	Offspring of healthy probands	Statistic	p-value	η^2
	n = 388	n = 132	n = 118			
A-LIFE						
Satisfaction	1.9 ± 0.8^a	1.7 ± 0.8^b	1.5 ± 0.6^b	F = 13.54	<0.0001	0.05
Recreation	1.8 ± 0.9^a	1.6 ± 0.8^b	1.5 ± 0.6^b	F = 4.89	0.01	0.02
Work	2.3 ± 0.9^a	2.2 ± 0.9^a	1.8 ± 0.7^b	F = 9.57	0.0002	0.03
Interpersonal	2.6 ± 0.9^a	2.4 ± 0.9^b	2.2 ± 0.8^b	F = 8.72	0.003	0.03
Total	8.4 ± 2.3^a	7.8 ± 1.9^b	7.1 ± 1.98^c	F = 16.46	<0.0001	0.05
CBCL raw scores						
Activities	5.1 ± 2.2^a	5.4 ± 2.1^{ab}	5.7 ± 1.9^b	F = 3.81	0.05	0.01
Social	5.6 ± 2.3^a	6.0 ± 2.2^{ab}	6.4 ± 2.3^b	F = 5.26	0.04	0.02
School	4.6 ± 1.3^a	4.5 ± 1.3^a	5.0 ± 1.1^b	F = 5.54	0.009	0.02
Total	15.4 ± 4.5^a	16.0 ± 4.4^a	17.1 ± 4.1^b	F = 6.46	0.02	0.02
CGAS						
CGAS hp	76.6 ± 12.4^a	81.0 ± 12.1^b	84.6 ± 9.6^c	F = 21.78	<0.0001	0.07
CGAS msp	65.0 ± 16.1^a	70.3 ± 16.5^b	78.3 ± 12.5^c	F = 32.10	<0.0001	0.10
CGAS current	74.4 ± 13.2^a	79.4 ± 12.7^b	83.6 ± 10.1^c	F = 25.39	<0.0001	0.08

All means are unadjusted.
p — adjusted for intra-family correlations using a model with random intercept for the family factor.
hp — highest past.
msp — most severe past.
Lower scores on the A-LIFE indicate better functioning while higher scores on the CBCL and CGAS indicate better functioning. Different superscripts indicate significant pair-wise comparisons with p<0.05.

psychopathology in general. Results show that overall, offspring of probands with BP do not function as well as offspring of healthy probands or offspring of probands with non-BP psychopathology across almost all domains measured (satisfaction, recreation, work, school, interpersonal relationships and overall). However, their functioning remains in the mildly impaired range. Goldstein et al. (2009) in the COBY study also reported mild to moderate psychosocial dysfunction among youth with BP. Hence it appears that impairments in functioning may precede onset of illness and gradually worsen over time. In addition to external psychosocial variables, intraindividual difficulties such as poor self esteem and anger regulation described by Rucklidge (2006) among youth with BP may also predate onset of illness and serve as predisposing and maintaining factors for dysfunction. Proband parent functioning and offspring psychopathology emerged as the two most salient variables accounting for the differences in offspring psychosocial functioning between groups. Comparing the healthy offspring of probands with BP and healthy offspring from the other two groups revealed that child psychopathology did not account for all the differences between groups. Thus, functional impairment does not appear to be inherently associated with being the offspring of a parent with BP, but rather, emerges in the face of parental impairment or offspring psychiatric disorder. Regardless of parent group, older offspring from lower socioeconomic families appeared to be at increased risk for psychosocial dysfunction; hence these factors should also be put into consideration when assessing offspring of parents with BP.

Although group differences were evident in this study, the offspring of probands with BP in our sample appeared to be functioning much better than might be expected given the fact that a substantial proportion (60%) had already been diagnosed with lifetime psychiatric disorders. On average they had scores in the range of good to mildly impaired across functional domains, and exhibited only slight impairment in global functioning, which is in keeping with studies that indicate relatively good premorbid functioning for young people who develop BP. This has important implications, as good premorbid functioning is known to increase the probability of better long term outcomes — particularly as compared with those who develop other disorders like schizophrenia (Cannon et al., 1997; Kutcher et al., 1998). It is however to be noted that the average age of the sample at intake was about 11 years. It would thus appear that functioning may not be so impaired at younger ages but may only begin to deteriorate from mid to late adolescence as social and academic demands increase, and offspring experience more consequences of self or parental psychopathology with subsequent life disruptions. Similar findings have been reported in which offspring of parents with BP were observed to develop psychosocial competencies early on in life, only to lose them by early adolescence (Radke-Yarrow et al., 1992). However, another possible explanation for the better than expected functioning among offspring of parents with BP in this study could be the receipt of early psychosocial and pharmacological interventions by approximately 30% of offspring of probands with BP. The exact nature, frequency, and timing of these interventions were not documented and therefore we were unable to examine their association with psychosocial functioning at intake. The rates of interventions among the offspring of parents with BP were however comparable to the rates in the other two offspring groups. Future analyses from the BIOS sample utilizing longitudinal data will be able to address this question.

Both the A-LIFE and CGAS showed differences in functioning between offspring of probands with BP and offspring of probands with non-BP psychopathology but the CBCL did not. Possible reasons for this could be that while the A-LIFE and CGAS are both interviewer rated the CBCL is a parent rated questionnaire. Youngstrom et al. (1999) found evidence to support dysphoria related bias in caregiver's reports about children's behaviors and emotions. Various forms of psychopathology are associated with dysphoria and this may be responsible for the absence of observable differences between offspring of probands with BP and offspring of probands with non-BP psychopathology in our study.

Similar to offspring of parents with MDD, offspring of parents with BP appear to experience functional difficulties in multiple domains, and parental impairment seems to play an important role in these difficulties. Studies from offspring of parents with MDD have shown that offspring psychosocial impairment is not mediated by offspring psychopathology alone (Lewinsohn et al., 2005) but also by continued parental dysfunction, as well as parent's current symptoms and stressors (Lee and Gotlib, 1991; Hammen et al., 1987). Furthermore, recent studies on offspring of parents with MDD show that when depressed mothers experience symptom remission and improved functioning by 3–6 months following treatment, there is associated symptom reduction and better functioning in their offspring (Swartz et al., 2008; Pilowsky et al., 2008). The efficacy of treatments for depressed youth has also been reported to be reduced in the presence of concurrent maternal impairments (Brent et al., 1998; Garber et al., 2009). The relationship between treatment of a parent with BP and child outcomes could be an area for further study, as well as the mechanisms by which parental and child functioning impact each other, as there is evidence to suggest that functioning may be bidirectional (Forehand and McCombs, 1988).

The above conclusions need to be interpreted within the limitations of the study. We did not control for all confounding variables such as family environment or stressful life events which have been linked with offspring functioning. However, the literature on family functioning is inconsistent and more recent studies are beginning to show that the added effect of child psychopathology may have more impact on family functioning than parent psychopathology alone (Du Rocher Schudlich et al., 2008; Reichart et al., 2004). Also, this is a cross-sectional analysis based on retrospective reports which measured offspring functioning at study intake only, and reliability data for the CGAS was not obtained. Longitudinal follow-up data from the sample will enable us to measure changes in offspring functioning over time through various developmental stages, and monitor the effects of treatment interventions. With prospective data, we will be able to parse out the temporal relationship between psychosocial functioning and the development of problem behaviors and psychopathology. Given that there were multiple levels of adjustment (intrafamily correlations, demographics, proband functioning, and offspring clinical characteristics), we considered the possibility that some power may have been

lost during analyses. A sensitivity analysis was carried out by examining reduced models that excluded parental GAF. In these reduced models the outcomes were significantly different among groups, whereas in the models including parent GAF, no significant differences between groups were detected. As during this process we lost only 2 degrees of freedom and the sample size was large enough, this indicated that the power to detect differences between groups remained adequate.

This study adds to the existing literature on psychosocial impairments among offspring of parents with BP and shows that they do experience some impairment in functioning across various psychosocial domains. Impairments may serve as proxies for current psychopathology and parental functioning, as well as be possible predictors of future psychopathology, course, and outcome. Optimizing outcome in offspring of parents with BP should involve early identification and treatment of psychiatric disorder in them as well as in their parents. Interventions which improve parental functioning should also be a priority. Preventive interventions could be directed toward the early detection of problem areas in functioning before onset of psychopathology especially for younger offspring.

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Conflict of interest

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All other authors declare that they have no conflicts of interest.

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