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Family Risk and Related Education and Counseling Needs: Perceptions of Adults with Bipolar Disorder and Siblings of Adults with Bipolar Disorder

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Abstract

Genetics and mental health professionals increasingly provide education and counseling related to risk for psychiatric illness, but there is insufficient evidence about patient perceptions and needs to guide such interventions. Affected individuals and relatives may perceive increased family risk and have interest in genetic education and counseling. Our objectives were to explore perceptions of family vulnerability, perceived control, and coping strategies related to familial risk and needs from genetic counseling. Our methods included conducting semi-structured interviews (n=48) with individuals with BPD and unaffected siblings. Content analysis generated descriptive data that provide guidance for clinical interventions and themes to evaluate in future studies. The results showed that participants perceived increased personal and family risk, attributing BPD to genes and family environment. Causal attributions were often uncertain and at times inconsistent. Participants wished to modify psychiatric risk to relatives, but were uncertain how to do so; despite the uncertainty, most parents reported risk-modification efforts. Efforts to cope with family vulnerability included monitoring and cognitive distancing. Participants endorsed the usefulness of education and psychological support, but described more ambivalence about receiving risk assessment. Educational and supportive interventions around family risk for BPD should focus on perceptions of cause and vulnerability, reproductive decision making, and early intervention and risk modification in young relatives. Psychological support is an important component. Providers should evaluate patient coping strategies, which could facilitate or hinder genetic counseling interventions, and should not assume interest in quantitative risk assessment.

Keywords

genetic counseling; psychiatric; bipolar disorder

INTRODUCTION

Accompanying our growing understanding of the complex, heritable nature of major psychiatric disorders is a parallel need to educate and counsel patients and families about the cause of psychiatric illness and about risk factors that may contribute to illness occurrence in families [Austin and Honer, 2005; Duffy et al., 2000; Finn and Smoller, 2006]. Available studies suggest that well over half of individuals with bipolar disorder (BPD) and schizophrenia, as well as their close relatives, are interested in genetics education and

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counseling [DeLisi and Bertisch, 2006; Lyus, 2007; Quaid et al., 2001; Schulz et al., 1982]. Psychiatric genetic counseling will be most effective when informed by collaboration between genetics and mental health professionals.

Affected individuals and relatives tend to hold common perceptions of BPD and schizophrenia, including moderate to high illness burden, with higher levels of burden reported by relatives than by affected individuals [Schulz et al., 1982; Smith et al., 1996]. Their perceived causes of the disorders include genetic/biological and environmental factors [Meiser et al., 2007; Meiser et al., 2005; Schulz et al., 1982; Targum et al., 1981]. There are limited data about perceptions of familial risk, and the results are inconsistent. Two studies found that individuals with schizophrenia or BPD underestimated risk for family recurrence, while siblings and spouses overestimated risk [Schulz et al., 1982; Targum et al., 1981]. Three other studies indicate that most individuals appreciated their increased family risk, and in fact often overestimated risk [Austin et al., 2006; Quaid et al., 2001; Trippitelli et al., 1998]. These studies provide little insight, however, into diagnosed individuals' and relatives' perceptions of the origin of the increased risk to relatives.

In a subset of diagnosed individuals and relatives, perceptions of psychiatric disorders appear to affect reproductive decision making. In an exploratory qualitative study, Meiser and colleagues [2005] found that about half of participants indicated that their dense family histories of BPD affected their reproductive decisions. Many reported having children, however, before they were aware of the role of genes in BPD. In a follow-up study in a similar population, 35% of non-parents were less willing to have children because of their strong family histories [Meiser et al., 2007]. Other studies have reported effects on reproductive plans in ~20-40% of individuals with psychotic disorders and their relatives [Austin et al., 2006; Lyus, 2007].

The existing data about perceptions of BPD and schizophrenia facilitate the development of expectations among a population that might seek psychiatric genetic counseling. The data fall short, however, of suggesting how to focus and structure interventions related to psychiatric risk in the family. In this qualitative study of individuals with BPD and siblings of individuals with BPD, we aimed for a deeper understanding of illness perceptions and their effects on perceived illness vulnerability in the family, reproductive decision making, and coping. We included siblings in the study because they seek genetic counseling services [Austin and Honer, 2007] but are not well represented in studies to date. Our clinical experience suggests that results from quantitative studies oversimplify the appraisal and coping processes of families dealing with psychiatric disorders.

MATERIALS AND METHODS

Recruitment

Our sampling targeted a range in participants' age, time since diagnosis, density of family history, and parental status. We recruited participants through the National Alliance for the Mentally Ill and the ClinicalTrials.gov websites, and through snowball strategies. Consistent with qualitative methodology, we discontinued recruitment when we achieved thematic saturation [Miles and Huberman 1994], indicated by recurring findings and little new information.

Sample

Participants were 18 years of age or older, diagnosed with BPD (per self report) or a sibling of a diagnosed individual, and spoke English fluently. Participants did not have to be sibling pairs, i.e., affected individuals were included without the participation of unaffected siblings, and visa versa.

Interviews

The first author conducted one-time qualitative interviews by telephone between 08/06 and 05/07. The interviews averaged about 60 minutes, and were audiotaped and transcribed. The interviews included two sections. The first was semi-structured and focused on appraisals and risk perception. The second, more structured portion of the interview employed clinical vignettes, which allowed us to capture participants' perceptions of the education and support model often used by genetics professionals during consultations for psychiatric disorders.

Analysis

The first author analyzed the transcribed interviews, using NVIVO® to facilitate coding. The first author reviewed all interview transcripts and summarized each interview, cataloguing themes of interest. We initially developed codes from existing literature and later refined them through data immersion and reconciliation with a second coder. Coded interviews and interview summaries were analyzed to reveal shared themes. We employed an inductive approach to the interpretation of findings that reflected the perspectives of participants, relying less on inference and more on summarization [Beeson, 1997; McAllister, 2001]. We randomly selected one-third of the transcripts to be coded by an independent coder (GWH). Inter-coder reliability was high (> 0.90).

The study was approved by the National Human Genome Research Institute's Institutional Review Board.

RESULTS

Participants

Forty-eight participants completed interviews; 25 were diagnosed with BPD and 23 were siblings of diagnosed individuals. Participants' characteristics are shown in Table I. Nineteen siblings had affected sisters (83%). Seven siblings (30%) had a psychiatric diagnosis or significant mood/anxiety symptoms, consistent with the increased rate of affective and anxiety disorders in close relatives of individuals with BPD. [insert table I.]

Most participants reported more than one psychiatrically-symptomatic relative within three generations. This suggests self-selection into the study of individuals with a stronger family history than is typical in BPD.

We found few differences between the major themes identified in siblings and in affected individuals. Thus, reported themes represent compiled findings, with discrepancies noted. Figure 1 provides a schematic representation of the findings of the semi-structured perceptions portion of the interview. [insert figure 1]

Perceptions of Bipolar Disorder

Burden—Most participants perceived BPD as significantly burdensome to the affected individual and family members. Affected individuals often described a moderate to strong effect of BPD on their past and current daily function, life plans, and relationships. Many expressed fears about their ability to control symptoms or maintain their desired life course in the future.

Many affected individuals discussed how earlier and better diagnosis and treatment might have reduced their life burden. Mothers especially wished that they had been diagnosed earlier. This was related to a perception that they could rear their children more effectively when treated and educated about the illness.

Perceived Cause—Participants' causal attributions were fairly consistent, though most interviewees expressed some uncertainty about cause. The large majority indicated that genetic, biological, and environmental factors together caused BPD to run in families. Most perceived genes as a primary causal factor, though the relative importance of genes and environment varied. As stated by one participant, "I do believe that it is there in the genes. And I personally believe that it can remain dormant in a person unless there is trauma in the life or stressors that just bring it out." (F, affected, 49yo) Another participant described the cause in this way: "For some reason, there's just the sickness that runs in my family. And like I said, I don't know if it's because of the way our brains are wired or if it's because of the environment people grow up in or combination of both, like, I don't know what it is." (F, sibling, 30yo)

Some participants described the cause of the illness in their family differently depending on the topic of discussion, suggesting some ambiguity in their causal attributions. Participants often looked to their own family histories to justify the importance of genetics. The few participants without a strong family history also endorsed the importance of genetics, though they noted the inconsistency with their own family history.

Many alluded to concepts of genetic predisposition and environmental triggers. Triggers that participants commonly mentioned were unstable child rearing and poor home environment, traumatic life events, stress, substance abuse, and hormonal changes. A small minority indicated that genes alone or environment alone caused the BPD in their family.

Risk of Mood Disorders to Family Members—We asked participants to rate the risk for mood disorders in close relatives. All affected participants who responded (three declined to answer) and most siblings felt that risk in their families was increased over population risk. Many referenced their own family histories as the indication of increased risk. One participant considered her family history, and the resulting risk in the family, in this way: "I've been concerned because my grandmother committed suicide. My dad had clear-cut bipolar behavior. And I have concerns that knowing, you know, things are inherited. And I don't know which of the bipolar genes...I might have passed on to either of my kids." (F, affected, 49yo) Conversely, some participants noted that, because not all relatives are affected, their family history provides hope for healthy offspring.

We encouraged participants to describe their perceptions of risk in the family in qualitative or quantitative terms. Appropriately, affected individuals perceived the risk for their close relatives to be higher than did siblings. All affected individuals described family risk as higher than for those with no family history, and many were "very concerned" or had families at "great risk." Of the ten affected individuals who used quantitative terms, the reported risk was at least 50% for five, 25 to 50% for four, and 20% for one.

Siblings' risk perceptions spanned a broader range, with several indicating no increased risk to relatives, or uncertainty about whether risk was increased. Most, however, reported increased risk based on their family histories, and several perceived risk as a great deal higher than population risk. Siblings' quantitative assessments varied widely, ranging between 1% and 50%.

A minority of participants made statements reflecting genetic determinism, i.e., expecting children to be affected. Many participants discussed the importance of their partners' mental health and family history to the overall risk for their children.

Perception of family vulnerability

In this study, participant appraisals of BPD as burdensome, risky to relatives, and familial contributed to a perception of overall family vulnerability. One participant described her sense of family vulnerability by saying, “It feels right to me to say that it is hereditary...It’s kind of like I want to say I hope it’s not right because then who knows? Then I’ll start thinking about, ‘Well, I wonder if I’ll be diagnosed eventually,’ or I have twins, I wonder if something’s going to occur with them.” (F, sibling, 29yo)

Though the perception of family vulnerability was common to the study population, many affected individuals did not connect their family histories of mood symptoms/diagnoses to their own mood symptoms until after they were diagnosed with BPD. Though many were symptomatic long before their diagnosis, the diagnostic label itself changed affected participants’ sense of family vulnerability and their perception of shared etiology with relatives. In addition, BPD was perceived as risky and heritable in a way that major depression and anxiety disorders were not. Sibling participants described a sense of family vulnerability that began earlier than did that of affected individuals; in some cases family vulnerability was recognized before the sibling was diagnosed, based on past family history.

Reproductive considerations—Very few affected participants with children reported that they considered the risk for mood disorders when making reproductive plans. Although all but one of the participants were symptomatic before their childbearing years, they reported not being concerned about children until they received the BPD diagnosis (i.e., they did not know to be concerned until after the diagnosis). Several parents felt great responsibility after the diagnosis, and were extremely concerned about risk to children. Only one stated that she would not have had children, had she known about the risk. Most siblings with children also reported not being concerned about risk for mood disorders when making reproductive plans.

In contrast, most participants without children expressed concerns about the risk of psychiatric illness in future children. Some affected individuals were anxious about their ability to provide a healthy home environment. These concerns arose both from fears of the effect of mood symptoms on child rearing, and also because participants described growing up with poor modeling of child rearing. Slightly less than half of affected individuals of reproductive age and slightly more than half of the siblings planned to have children. A small group of affected individuals (n=3) and siblings (n=4) reported not having children because of risk for mood disorders. Said one such participant, “I saw the sickness in my family and myself early enough to realize that I did not want to have children. You know, inflict whatever illness I have on an innocent child.” (F, affected, 43yo)

At-risk family members—Many siblings expressed concern about their own mental health. This threat was perceived to result primarily from genetic risk, but also from being reared in an unhealthy home environment. About half had concerns serious enough to negatively affect their life plans or self image.

The large majority of parent participants were concerned about their children’s risk for psychiatric illness. All affected individuals and the large majority of siblings described monitoring their children’s moods. About half of affected participants and 25% of siblings reported that one or more children were symptomatic or diagnosed with a mood disorder. Many also mentioned concerns about nieces and nephews. About a quarter of diagnosed individuals identified young relatives who reminded them of themselves, thus warranting additional concern and monitoring.

Desire to end the cycle of illness in the family

Many participants discussed cycles of psychiatric illness, and often substance abuse, in their families. One participant described the illness cycle in her family in the following way: “Well, it’s been catastrophic...for my family it’s been a repeat of having to visit my mother in hospitals and they’ve just been devastated by it emotionally and just couldn’t take it. And for me, it’s just been a nightmare because I never know that I’m getting sick.” (F, affected, 59yo) The cycles were attributed to the familial nature of the disorder. Participants felt responsible for ending the illness cycle in their families. This was perceived as more feasible than for past generations because of increased symptom awareness, earlier diagnosis, better treatments, and parents striving for better home environments.

Participants reported that witnessing the illness had strong effects on the family. This was most evident when affected participants discussed symptomatic children, who often were highly resistant to being diagnosed with the condition that led to such burden in the family. Siblings also described resistance to being labeled with the same illness as affected relatives.

Modifying Risk for Mood Disorders

While participants expressed some responsibility for and hopefulness about ending the illness cycle in the family, they were uncertain about concrete steps they could take to affect such change. A majority of participants felt unable to, and/or unsure about, how to significantly reduce risk to young relatives. Even so, most discussed the importance of healthy family and home environments, and had undertaken efforts (or planned to undertake efforts) to improve their child rearing and/or intervene to assist other young relatives. Environmental modifications were not necessarily perceived as leading to prevention, but instead as potentially leading to better outcomes if the child developed symptoms. As one participant described, “All the normal healthy things that you...can give a child. Plenty of love and support, good education, lots of structure, setting a good example; I think all of those things can all help...but I still think with this particular disease...it’s predominantly genetic. And so you could all those right things and your kids could still become bipolar.” (F, affected, 40yo)

Only a small minority endorsed that environmental modifications could affect a large reduction in risk. Another small minority expressed fatalism about young family members, expecting them to become symptomatic. Many discussed the importance of early symptom identification and treatment to improve outcomes. About half of affected participants reported that their own illness experience allowed them to notice symptoms and guide relatives in seeking treatment.

Coping with Familial Vulnerability—Many participants used monitoring coupled with cognitive distancing to cope with personal and family risk. Monitoring was presented in several ways: as an ability that comes from having lived with the disorder (i.e., participants know how to monitor and seek interventions because of their experiences); as anxiety provoking or burdensome; and most commonly as a responsibility (i.e., feeling of duty to watch for signs and seek treatment). A participant described her monitoring in this way: “I think the pressure that I put on my own self to be very vigilant in watching my kids leaves me anxious sometimes because it’s like, I really want to be sure to catch this if I see it because I was into my 30s before I was diagnosed. I don’t want that for my kids.” (F, affected, 41 yo)

Some participants used monitoring purposefully to reduce worry (i.e., allowing themselves to limit concern until they see troubling symptoms). Some siblings expressed frustration at not knowing what to look for in at-risk relatives. Self-vigilance in siblings included

monitoring moods, seeking advice (e.g., from mental health professionals and affected siblings), and undertaking efforts to improve mental health (e.g., therapy, social support, physical distance from dysfunctional families).

Many participants described coping through cognitive efforts aimed at reducing emotional distress by distancing at-risk individuals from the threat (cognitive distancing). These coping efforts included identifying “protective” personality traits, lack of “risky” personality traits, or distancing by favorably contrasting the young person’s home environment to that of ill relatives. For example, one participant described her at-risk daughter in this way: “... she’s being raised completely differently than I was raised. And not that my parents were bad, but--so at least that gives her a shot at things maybe being different for her....Even if it doesn’t keep her from like straying down the wrong road. I kind of feel, like, if nothing else, she’ll know where to go when she needs help, even if it’s a little too late.” (F, sibling, 30yo)

Cognitive distancing in asymptomatic siblings included ascribing to themselves, or actively pursuing, different personality traits or life experiences than affected sibling. Siblings also normalized their moods (e.g., “Everyone has ups and downs”) and employed distancing based on age (e.g., “If I were going to get it, I would have had it by now”). Symptomatic sibling participants described their illness as different than that of their diagnosed brothers or sisters; most often, they perceived their illness to be an episodic reaction to severe environmental triggers, while they perceived their siblings with BPD to have a chronic illness.

Perceptions of Genetic Services

We used clinical vignettes (see Table II) describing elements of genetic counseling to gauge participants’ perceptions of clinical services. Participants strongly endorsed the relevance of the first vignette that described education about etiology, the impact of parenting, and early symptoms and intervention. Many remarked on the importance of education about the limited ability of parents to cause or prevent BPD.

There was moderate endorsement of the second vignette, which offered risk assessment based on family history. Participants raised concerns about the validity of the risk information and were uncertain about what one would do with such information. Some questioned the professional credibility of the fictional genetic counselor. A small minority were very negative about any predictive information. Many participants requested concurrent psychological support related to illness adjustment, living with personal risk and/or risk to young relatives, and family-based interventions to address effects of BPD on family function.

Participants’ primary perceived benefits of the services described in the vignettes were the ability to monitor at-risk individuals and identify symptoms early. Many participants identified times in their own lives when genetic counseling may have been useful, including at diagnosis, when making reproductive plans, and when concerns for children arose.

DISCUSSION

The quantitative design allowed us to identify new and unexpected themes while building on established concepts, and to provide nuance, complexity, and context [Beeson, 1997; Hull et al., 2001]. Themes developed from this study have implications for clinical care related to education and counseling about familial risk.

The perception of family vulnerability in this study is consistent with a framework developed by Walter and colleagues [Walter et al., 2004]. Using a meta-ethnographic

approach, the researchers reviewed studies exploring understanding about family risk in various complex disorders. The resulting theoretical framework informs how individuals with familial risk develop and manage their sense of vulnerability. The framework includes three key constructs, salience (that is, worthiness of attention, e.g., acknowledgement that the disorder runs in the family), personalizing processes (applying personal models of inheritance and disease causation, e.g., explanations about why the disorder runs in the family), and a personal sense of vulnerability (i.e., sense of personal and family risk). The constructs are fluid and interdependent, and lead to coping and control efforts. The consistency of our results with the framework suggests that the process leading to perceived family vulnerability in our BPD population resembles that of other populations with complex disorders, and that interventions developed around family vulnerability in common disease may be useful across sub-specialty.

Comparing our results to existing psychiatric literature, as in other studies our participants perceived BPD to be burdensome to affected individuals and relatives [Schulz et al., 1982; Smith et al., 1996] and to be caused by genetic and environmental risk factors [Meiser et al., 2007; Meiser et al., 2005; Schulz et al., 1982; Targum et al., 1981]. Using a qualitative design allowed us to capture uncertainty about causal attributions; though participants were able to describe attributions readily, they often were not confident about those they put forward. For some participants, reported causal attributions varied during the interview, suggesting the attributions may be weakly held and dynamic. This invites an opportunity for providers to explore and clarify patient perceptions of etiology.

Most participants perceived an increased risk to close relatives. Affected individuals reported risk to family members to be quite high, consistent with other studies [Austin et al., 2006; Lyus 2007], while siblings reported a wider range of risks. As our population primarily included families with more than one affected relative, the increased risk perception may have some credibility.

Participants identified a strong sense of family and personal vulnerability originating from the familial nature (i.e., genetic and family environmental factors) of BPD. The diagnostic label's strong effect on affected participants' perceptions was unexpected, especially because many reported multi-generational mood issues and a long course of illness. The diagnostic label likely increased participants' mental association between their own symptoms and their family histories, thus increasing their overall perception of family vulnerability. Siblings appreciated the saliency of their family histories at younger ages than did affected individuals, resulting in stronger than anticipated effects on their personal sense of vulnerability.

Most participants with children were not concerned about recurrence risk before reproduction. The reasons for lack of concern differed between the study groups. The majority of affected individuals reported not being aware of the risk. The majority of siblings reported some awareness of risk but little or no concern. Similarly, Meiser and colleagues found that many individuals with BPD were not aware of the risk, though their participants were from high-density families [Meiser et al., 2005]. One can surmise that individuals with strong family histories might have awareness of risk to relatives even without a personal diagnosis, as noted in the sibling population in this study. For most parent participants, the desire to have children likely trumped perceived risk. Studies evaluating reproductive decision making in populations with known genetic risk suggest a similar denial of risk in those who strongly desire children [Shiloh and Saxe, 1989]. The framework developed by Walter and colleagues [Walter et al., 2004] suggests another conclusion, positing that perceptions of control play into salience (i.e., increased attention to outcomes perceived as controllable). Prospective parents likely perceive little control over the risk for

BPD in children, particularly during pregnancy and infancy, which in turn may reduce their perceived saliency of their family history.

Participants indicated concern about and monitoring of children, a robust theme that has not been explored previously. Study participants indicated two strong but conflicting positions—a desire to end the cycle of illness in the family, and limited perceived control over risk to young relatives. Though participants expressed little faith in their efforts to ward off illness by improving child rearing, family communication, and environment, the efforts provided participants a sense of control over risk, and represented active attempts to protect their children's health. These efforts at control may explain why the saliency of the family history remains high when related to children.

Clinical Implications

Participants expressed interest in genetic counseling, including a desire to better understand the cause despite having a reasonable understanding of common disease etiology. Patients are likely to look to their own families when developing causal attributions, and providers can similarly use patients' family histories to educate about etiology. On the whole, participants were more interested in general information and counseling than quantified risk assessment based on family history, which was viewed as having benefits and limitations. Our findings suggest that patients may be most eager to discuss family risk following a BPD diagnosis and when concerns for children arise. Siblings may benefit from counseling around their own risk and about early illness symptoms.

As part of any intervention about family risk, participants advocated for psychological support related to living with risk in the family. Providers should include a strong focus on psychological issues during their genetic counseling sessions and should consider referral for more in-depth counseling. The context and purpose of etiology and risk counseling was important to participants, supporting the practice of having clients set the agenda for a genetic counseling session.

While genetic counseling may appeal to some patients who are considering reproduction, one cannot assume that patients will make reproductive choices based on psychiatric history. This may be because the desire to have children and a limited perception of control over risk decrease the saliency of perceived risk.

Patients and relatives may benefit from discussions about concerns for young children, their ability to provide a healthy home environment, and interventions based on early illness symptoms. Parents are likely to benefit from counseling around their limited control over illness onset in children. Though there are few data to support recommendations for affected individuals, providers may encourage parenting education [Craig, 2004], parental support systems and outside daycare [Lee et al., 2006], and family-based interventions [Beardslee et al., 2003] as ways to attempt to reduce risk in children. Providers should stress the importance of early symptom identification and early treatment, and should empower affected individuals by reinforcing that their illness experience likely has made them more capable of noticing and responding to early symptoms.

Participants commonly used monitoring and cognitive distancing to cope with family vulnerability and limited control. Those who favor monitoring might most benefit from counseling around early symptom identification and risk reduction. Cognitive distancing, on the other hand, may decrease an individual's interest in genetic counseling and may interfere with the cognitive and emotional processing of information provided during the session.

Limitations

This study reveals important new themes and provides direction for professionals who counsel about family risk for BPD. In spite of its strengths, the study is limited in that it is a self-selected group. Participants were recruited from the NAMI and ClinicalTrials.gov websites, suggesting more knowledge about psychiatric illness than expected from the average affected individual or sibling. Participants may have been coping more successfully than average, because participation required organized thought and effort. Responses were subject to recall bias, and the use of hypothetical vignettes was likely imperfect in predicting responses in an actual situation. It is possible that participants' mood at the time of the interview (i.e., whether participants were in a depressed, hypomanic, or manic state) may have affected their perceptions of risk and outlook for the future.

Our recruitment efforts did not capture a broad range in terms of family history; almost all participants had more than one affected family member. This is not surprising given the increased saliency of the subject matter for individuals with more affected family members, and is perhaps reflective of individuals who will seek genetic counseling related to psychiatric illness. Since half of the siblings had concerns about their own mental health, we presume this bias may have generated motivation to participate in the study.

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REFERENCES

- Austin J, Smith G, Honer W. The genomic era and perceptions of psychotic disorders: genetic risk estimation, associations with reproductive decisions and views about predictive testing. *Am J Med Genet B Neuropsychiatr Genet*. 2006; 141:926–928. [PubMed: 16958030]
- Austin JC, Honer WG. The potential impact of genetic counseling for mental illness. *Clin Genet*. 2005; 67:134–142. [PubMed: 15679823]
- Austin JC, Honer WG. The genomic era and serious mental illness: a potential application for psychiatric genetic counseling. *Psychiatr Serv*. 2007; 58:254–261. [PubMed: 17287384]
- Beardslee W, Gladstone T, Wright E, Cooper A. A family-based approach to the prevention of depressive symptoms in children at risk: evidence of parental and child change. *Pediatrics*. 2003; 112:e119–131. [PubMed: 12897317]
- Beeson D. Nuance, complexity, and context: qualitative methods in genetic counseling research. *J Genet Couns*. 1997; 6:21–43. [PubMed: 11660391]
- Craig E. Parenting programs for women with mental illness who have young children: a review. *Aust N Z J Psychiatry*. 2004; 38:923–928. [PubMed: 15555026]
- DeLisi LE, Bertisch H. A preliminary comparison of the hopes of researchers, clinicians, and families for the future ethical use of genetic findings on schizophrenia. *Am J Med Genet B Neuropsychiatr Genet*. 2006; 141:110–115. [PubMed: 16287043]
- Duffy A, Grof P, Robertson C, Alda M. The implications of genetics studies of major mood disorders for clinical practice. *J Clin Psychiatry*. 2000; 61:630–637. [PubMed: 11030482]
- Finn C, Smoller J. Genetic counseling in psychiatry. *Harv Rev Psychiatry*. 2006; 14:109–121. [PubMed: 16603476]
- Hull, SC.; Taylor, HA.; Kass, NE. Qualitative Research. In: Sugarman, J.; Sulmasy, DP., editors. *Methods in Medical Ethics*. Georgetown University Press; Washington, DC: 2001. p. 146-168.
- Lee L, Halpern CT, Hertz-Picciotto I, Martin SL, Suchindran CM. Child care and social support modify the association between maternal depressive symptoms and early childhood behaviour problems: a US national study. *J Epidemiol Community Health*. 2006; 60:305–331. [PubMed: 16537346]

- Lysus VL. The importance of genetic counseling for individuals with schizophrenia and their relatives: Potential clients' opinions and experiences. *Am J Med Genet B Neuropsychiatr Genet.* 2007; 144B:1014–1021. [PubMed: 17525978]
- McAllister M. Grounded theory in genetic counseling research. *Journal of Genetic Counseling.* 2001; 10:233–250.
- Meiser B, Mitchell PB, Kasparian NA, Strong K, Simpson JM, Mireskandari S, Tabassum L, Schofield PR. Attitudes towards childbearing, causal attributions for bipolar disorder and psychological distress: a study of families with multiple cases of bipolar disorder. *Psychol Med.* 2007; 37:1601–1611. [PubMed: 17537283]
- Meiser B, Mitchell PB, McGirr H, Van Herten M, Schofield PR. Implications of genetic risk information in families with a high density of bipolar disorder: an exploratory study. *Soc Sci Med.* 2005; 60:109–118. [PubMed: 15482871]
- Miles, MB.; Huberman, M. *Qualitative Data Analysis: An Expanded Sourcebook.* Sage Publications, Inc.; Thousand Oaks, CA: 1994. p. 337
- Quaid KA, Aschen SR, Smiley CL, Nurnberger JI. Perceived Genetic Risks for Bipolar Disorder in a Patient Population: An Exploratory Study *Journal of Genetic Counseling.* 2001; 10:41–51.
- Schulz PM, Schulz SC, Dibble E, Targum SD, van Kammen DP, Gershon ES. Patient and family attitudes about schizophrenia: implications for genetic counseling. *Schizophr Bull.* 1982; 8:504–513. [PubMed: 7134894]
- Shiloh S, Saxe L. Perception of risk in genetic counseling. *Psychology and Health.* 1989; 3:45–61.
- Smith LB, Sapers B, Reus VI, Freimer NB. Attitudes towards bipolar disorder and predictive genetic testing among patients and providers. *J Med Genet.* 1996; 33:544–549. [PubMed: 8818938]
- Targum S, Dibble E, Davenport Y, Gershon E. The Family Attitudes Questionnaire. Patients' and spouses' views of bipolar illness. *Arch Gen Psychiatry.* 1981; 38:562–568. [PubMed: 7235858]
- Trippitelli CL, Jamison KR, Folstein MF, Bartko JJ, DePaulo JR. Pilot study on patients' and spouses' attitudes toward potential genetic testing for bipolar disorder. *Am J Psychiatry.* 1998; 155:899–904. [PubMed: 9659854]
- Walter FM, Emery J, Braithwaite D, Marteau TM. Lay understanding of familial risk of common chronic diseases: a systematic review and synthesis of qualitative research. *Ann Fam Med.* 2004; 2:583–594. [PubMed: 15576545]

Figure 1.
BPD Perceptions of Study Participants

Table I

Participants' Characteristics

	Diagnosed Participants	Sibling Participants
	N=25	N=23
Mean age of participant in yrs (range)	43 (25-59)	38 (23-81)
Mean reported age at diagnosis in yrs (range) *	32 (17-52)	26 (7-45) (age of affected sib at diagnosis)
Mean time since diagnosis in yrs (range)	12.1 (0-30)	9.9 (0-39) (time since affected sib's diagnosis)
Race	22 (88%)	17 (74%)
Caucasian	2 (8%)	3 (13%)
African American	0 (0%)	1 (4%)
Native American	1 (4%)	0 (0%)
Hispanic	0 (0%)	2 (9%)
Multiracial/other		
Sex		
Male	5 (20%)	6 (26%)
Female	20 (80%)	17 (74%)
# with children	14 (56%)	12 (52%)

* Note, most participants reported that symptoms began many years before the bipolar disorder diagnosis

Table II

Text of Structured Vignettes

<p>In these stories, Susan/Tom has bipolar disorder [for sibling participants: has a brother with bipolar disorder]. He/she has questions about the cause of bipolar disorder and is concerned about it happening again in his/her family. Susan/Tom goes to see a genetic counselor. A genetic counselor is a health care professional who specializes in helping families who have concerns about conditions that can run in the family. After each story I'll ask you to tell me how the information that Susan/Tom learned could be useful or why you think that it is not useful.</p>
<p>1. Susan/Tom goes to a genetic counselor to learn what causes bipolar disorder. The genetic counselor tells Susan/Tom that bipolar disorder is caused by a mix of a person's genes and non-genetic things, like the environment that they live in. The genetic counselor explains that there are many genes involved that may be passed down from both sides of the family. Some things in a person's everyday environment, like being exposed to viruses, also may affect the chance of getting bipolar disorder. The genetic counselor says that, while good parenting helps all children's development, parenting style does not cause, or prevent, bipolar disorder. The genetic counselor and Susan/Tom discuss the importance of finding signs of bipolar disorder early and getting treatment right away. Do you think this information would be useful to Susan/Tom? Why or why not?</p>
<p>2. Susan/Tom goes to the genetic counselor to learn about the chance that bipolar disorder could happen again in the family. The genetic counselor takes a careful family history, and asks Susan/Tom questions about the mental health of family members. The genetic counselor also asks about Susan/Tom's own mental health. The genetic counselor uses the family history information to estimate that there is about a 20% chance, which is the same as a 1 in 5 chance, that bipolar disorder could happen again in a young person in Tom's/Susan's family. The genetic counselor says that this chance might not be exactly right, but is probably close. She hopes the information can give Susan/Tom an idea of how likely it is that others in her/his family may get bipolar disorder. Do you think this information would be useful to Susan/Tom? Why or why not?</p>