Personality traits in children of parents with unipolar and bipolar mood disorders

ROTHEN, Stéphane, et al.

Abstract

BACKGROUND: Using family study data, the following questions regarding the mechanisms of association between personality traits and mood disorders were addressed: 1) Is there an association between unipolar and bipolar mood disorders and personality traits in probands? 2) Are personality traits associated with depression in their 9 to 17 year-old children? 3) Is there an association between parental mood disorders and personality traits in offspring? 4) Are parental personality traits associated with the risk of depression in offspring? METHODS: The study included 50 probands with bipolar and 37 with unipolar mood disorder, 34 healthy controls as well as 178 of their children between 9 and 17 years. Diagnoses were made according to a best-estimate procedure based on a semi-structured interview (DIGS), medical records and family history information. Personality traits were assessed using the Eysenck Personality Questionnaire in adults and the Eysenck Personality Questionnaire Junior in offspring. RESULTS: Personality traits, and in particular Neuroticism, were found to be associated with mood disorders in currently […]

Reference


DOI : 10.1016/j.jad.2008.05.013
PMID : 18585789

Available at:
http://archive-ouverte.unige.ch/unige:1452

Disclaimer: layout of this document may differ from the published version.
Personality traits in children of parents with unipolar and bipolar mood disorders

Rothen Stephane1,2, Vandeleur Caroline Louise2, Lustenberger Yodok1,2, Jeanprêtre Nicolas1, Ayer Eve1, Fornerod Daniel1, Gamma Franziska1, Teichmann Tania1, Halfon Olivier1, Ferrero François2, Preisig Martin1

1 Department of Psychiatry, University Hospital Center and University of Lausanne
2 Department of Psychiatry, University Hospital of Geneva

Acknowledgments
This research was supported by grants from the Swiss National Foundation (#32-40677.94, #3200B0-105969, #32-47315.96, # 32-061974.00) and by a grant from GlaxoSmithKline.

The SNF as well as GSK had no further role in study design; in the collection, analysis and interpretation of data; in the writing of the report; and in the decision to submit the paper for publication.

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

Address for correspondence
Stéphane ROTHEN
DP-CHUV, Site de Cery, 1008 Prilly. Switzerland.
Phone: +41 21 643 6608
Fax: +41 21 643 6469
e-mail : stephane.rothen@chuv.ch
Abstract

Background: Using family study data, the following questions regarding the mechanisms of association between personality traits and mood disorders were addressed: 1) Is there an association between unipolar and bipolar mood disorders and personality traits in probands? 2) Are personality traits associated with depression in their 9 to 17 year-old children? 3) Is there an association between parental mood disorders and personality traits in offspring? 4) Are parental personality traits associated with the risk of depression in offspring?

Methods: The study included 50 probands with bipolar and 37 with unipolar mood disorder, 34 healthy controls as well as 178 of their children between 10 and 17 years. Diagnoses were made according to a best-estimate procedure based on a semi-structured interview (DIGS), medical records and family history information. Personality traits were assessed using the Eysenck Personality Questionnaire in adults and the Eysenck Personality Questionnaire Junior in offspring.

Results: Personality traits, and in particular Neuroticism, were found to be associated with mood disorders in currently affected as well as remitted probands and offspring. However, there was no association between mood disorders in parents and personality traits in their children, and conversely, parental personality traits were not associated with the risk of depression in offspring.

Limitations: 1) Relatively small proportion of offspring who were still unaffected but likely to subsequently develop mood disorders; 2) cross-sectional design.

Conclusions: The findings were best compatible with the complication or scar hypothesis, which assumes the occurrence of abnormal personality traits as a consequence of previous depressive episodes.

Keywords: Personality Traits, Mood disorders, EPQ, EPQ-J, Depression
Personality traits in children of parents with unipolar and bipolar mood disorders.

1. Introduction

The bulk of research has revealed associations between unipolar depression and personality traits. This literature has been reviewed recently (Clark et al., 1994; Sass and Junemann, 2003; Christensen and Kessing, 2006). Indeed, unipolar depressives generally exhibited higher scores on neuroticism, self-criticism, dependency and rigidity (Sauer et al., 1997). In contrast, the literature regarding associations between bipolar disorder and personality traits is less consistent. Nevertheless, several studies on bipolar patients found this disorder to be associated with high negative affectivity or neuroticism, high novelty-seeking, high harm-avoidance, self-transcendence, low self-directedness and low cooperativeness (Savitz and Ramesar, 2006).

In children, research on personality traits has shown that extreme withdrawal, neuroticism and negative emotionality in childhood are predictive of later internalizing psychopathology including depression (Savitz & Ramesar, 2006). Preliminary results of studies on the offspring of bipolars suggest that these children may exhibit some abnormal personality traits which could be a premorbid risk factor for the subsequent development of bipolar disorder (Hodgins et al., 2002). The children of bipolars were found to be more active (Kron et al., 1982; Decina, 1983) and aggressive (Worland, 1979; Kron et al., 1982; Decina, 1983). They also revealed more emotional over-control and temper dysregulation (Kuyler, 1980), recently defined as "high emotional reactivity" and "emotional dysregulation" which seem to characterize the irritability and affective storms often observed in bipolar children (Hirshfeld-Becker et al., 2003)

Nevertheless, the nature of the association between mood disorders and personality traits still needs to be elucidated. Klein and colleagues (Klein et al., 2002) describe six potential mechanisms that could underlie this association: 1. Common cause: personality and mood disorders have the same etiological cause; 2. Precursor: personality is an early manifestation of mood disorders; 3. Predisposition: personality increases the risk of developing mood
disorders; 4. **Pathoplasticity:** personality influences the expression of the course of mood disorders; 5. **State dependence:** Mood state colors or distorts the assessment of personality; and 6. **Complications (or scar):** episodes of mood disorders have an enduring impact on personality that persists after recovery.

Besides longitudinal research, the study of patterns of familial aggregation of specific disorders and personality traits is a suitable method to determine the underlying mechanism. Both mood disorders (Sullivan et al., 2000; Craddock and Jones, 2001; Faraone et al., 2003; Smoller and Finn, 2003; Merikangas and Low, 2004; Shih et al., 2004) and personality traits (Scarr et al., 1981; Ahern et al., 1982a; Ahern et al., 1982b; Carmichael and McGue, 1994; Bratko and Marusic, 1997) have been shown to aggregate in families. Adolescent offspring of affected and unaffected probands are of particular interest for assessing the mechanisms of the association between mood disorders and personality traits. Indeed, these offspring offer an excellent opportunity to determine whether personality traits are a premorbid condition for the later onset of mood disorders, given that at that age personality traits are already measurable but most of those who will develop a mood disorder are still unaffected. The expected findings from such an offspring study regarding potential mechanisms underlying the association between mood disorders and personality are presented in Table 1.

Consequently, we addressed the following study questions using data collected within a controlled family study from probands and offspring: 1) Are personality traits, as measured by the Eysenck Personality Questionnaire (EPQ: Eysenck and Eysenck, 1975), associated with Major Depressive Disorder (MDD) and bipolar disorder in probands, and if so, is this association limited to probands who are in a current mood episode? 2) Is there an association between personality traits and depression in the 9 to 17 year-old offspring of probands? 3) Do personality traits of the offspring of affected probands differ from those of the offspring of controls, and if so, is this difference also present in children who have no lifetime history of depression? and 4) Is there an association between personality traits in parents and the risk of depression in their offspring, and if so, is this association also present in the offspring of unaffected parents?
2. Methods

2.1 Sample

The data of the present paper stemmed from a family study on unipolar and bipolar mood disorders. For this paper, the psychiatric proband sample was restricted to patients consecutively recruited from the psychiatric departments of Lausanne and Geneva who had at least one 9 to 17-year old child who had also participated. Probands were required to meet DSM-IV criteria for bipolar disorder (n=50) or MDD (n=37). Control probands (n=34), who did not meet criteria for one of the above-mentioned disorders, were recruited at the orthopedic departments of Lausanne and Geneva. The mean age of the probands was 40.9 years, (s.d: 6.27 years), 52% were mothers, 18% held managerial or professional specialty positions and 95% were citizens of European countries.

The sample of children included 178 minor offspring of probands (53% males; mean age 13.2 years, s.d: 2.5 years). Seventy-two of them were children of bipolar probands (54% males; mean age 13.4 years, s.d: 2.5 years), 55 were children of MDD probands (47% males; mean age 13.5 years, s.d: 2.5 years) and 51 were children of controls (59% males; mean age 12.5 years, s.d: 2.6 years). The majority of families included one (n=76) or two (n=35) children, ten families had three or four children.

This research was approved by the institutional review board and parents gave written informed consent for their participation and for that of their children prior to the assessments.

2.2. Assessments

Participants were interviewed by psychologists or psychiatrists who were trained over a three-month period. Training included videotaped interviews which were supervised by a clinically experienced senior psychologist. Interviewers were blind to the disease status of
the other family members. The diagnostic assignment according to DSM-IV was based on a best-estimate procedure including information from direct interviews, family history reports and medical records (Leckman et al., 1982).

Diagnostic information of probands was elicited using the Diagnostic Interview for Genetic Studies (DIGS: Nurnberger et al., 1994). The French translation of this instrument (Leboyer et al., 1995) revealed high kappa coefficients for inter-rater reliability and slightly lower kappas for test-retest reliability for major Axis I diagnoses including mood disorders (Preisig et al., 1999).

Children were assessed using a modified version of the Kiddie-Schedule for Affective Disorders and Schizophrenia (K-SADS-E: Orvaschel et al., 1982b). This modified version was established for the Yale Family Study (Merikangas et al., 1998) and translated into French by our research group. The K-SADS-E has been found to be a reliable and valid instrument for obtaining lifetime diagnoses on prepubertal children (Orvaschel et al., 1982a) and adolescents (Gammon et al., 1983; Chambers et al., 1985).

Family history information was collected using the French version of the Family History-Research Diagnostic Criteria (FH-RDC) (Andreasen et al., 1977). The validity of the French version of this instrument was previously tested through the assessment of agreement between the direct interview and family history methods for a series of diagnoses in adults (Rougemont-Buecking et al., 2008; Vandeleur et al., 2008). The validity in children is in preparation.

Personality traits in probands were assessed using the EPQ, a 90-item scale which measures Neuroticism (emotional instability versus stability), Extraversion (outgoing disposition versus introversion) and Psychoticism (toughness versus tenderness/agreeableness and conscientiousness). The French version of the instrument was validated by its originator (Eysenck et al., 1980). Using 3 different French samples, the authors reported Cronbach alpha coefficients of 0.78 to 0.87 for Neuroticism, 0.72 to 0.82 for Extraversion and 0.60 to 0.72 for Psychoticism, respectively. For the measurement of personality traits in children the junior version of the EPQ (EPQ-J: Eysenck & Eysenck, 1975)
was applied, which is comprised of 81 items. Our group has previously established and validated its French translation. Validation was based on confirmatory factor analysis, which revealed a sufficient fit of an oblique factor solution testing the three original EPQ dimensions as well as the Lie scale (Rothen et al., 2008). Cronbach alpha coefficients were generally satisfactory for all the factors: 0.84 for Neuroticism and 0.74 for both Extraversion and the Lie scale, and 0.69 for Psychoticism.

2.3. Data analysis

1. Intra-individual associations between disorders and personality traits in probands

These associations were determined using multiple linear regression models, which included the specific personality trait as the dependent variable and the diagnostic status (subdivided into current and past episodes) as well as sex and age as the independent variables. For these analyses, 39 probands were excluded because of missing data on personality traits or currency of episodes.

2. Intra-individual associations between disorders and personality traits in offspring

These associations were assessed using generalized linear models (mixed model; (Searle, 1971), which also included the specific personality trait as the dependent variable and the diagnostic status as well as sex and age as the independent variables. These models can be applied to correlated measurements, therefore accounting for the lack of independence of the observations due to the fact that the number of children varied across families.

3. Inter-generational associations between disorders in probands and personality traits in offspring

To test these associations, similar generalized linear models were applied, including the specific personality trait in children as the dependent variable and the lifetime diagnostic
status in probands, sex and age of children as well as sex of probands as the independent variables.

4. **Inter-generational associations between personality traits in probands and diagnostic status in offspring**

These associations were assessed again employing generalized linear models (generalizing estimate equations, GEE: Liang and Zeger, 1986), which included the diagnostic status in children as the dependent variable and dichotomized personality scores (using the median in probands as the cutoff), sex and age of children as well as sex of probands as the independent variables. For these analyses, 58 children needed to be excluded because of missing data on personality traits in their parent.

Statistical analyses were performed using the Statistical Analysis System (SAS, version 9.1.3). For the multiple regression analyses and generalized linear models, PROC REG, PROC MIXED and PROC GENMOD were employed.

3. **Results**

1. **Intra-individual associations between disorders and personality traits in probands**

Table 2 presents intra-individual associations between the three EPQ personality dimensions and mood disorder diagnoses in probands. Unipolar and bipolar probands who were currently depressed or in remission scored significantly higher on Neuroticism than controls, with currently depressed individuals having the highest scores. Although the Neuroticism scores of bipolars in a manic episode were in a similar range to those of bipolars in remission, their scores did not differ significantly from those of controls. Regarding Extraversion, only currently depressed unipolar probands revealed significantly decreased scores as compared to controls, whereas with respect to Psychoticism, bipolars scored higher regardless of the presence or type of mood episodes.

INSERT TABLE 2 ABOUT HERE
2. Intra-individual associations between disorders and personality traits in offspring

Among offspring, only one of them reported a manic episode. For this reason, associations between manic symptomatology and personality traits in offspring could not be assessed. However, similarly to the findings in probands, increased levels of Neuroticism were found in offspring in both depressive episodes and remission (Table 3). Again, those who were currently depressed revealed the highest scores. However, we did not observe an association between depression and Extraversion, whereas currently depressed offspring scored higher on Psychoticism than offspring without a lifetime history of depression.

INSERT TABLE 3 ABOUT HERE

3. Inter-generational associations between disorders in probands and personality traits in offspring

Table 4 reveals no influence of parental psychopathology on any of the levels of the three personality dimensions in offspring. This finding was also confirmed when the sample was split according to the lifetime history of a depressive episode in the offspring.

INSERT TABLE 4 ABOUT HERE

4. Inter-generational associations between personality traits in probands and diagnostic status in offspring

Conversely, personality scores in probands (above vs. below the median) were not associated with an increased risk of depression in offspring, regardless of the type of parental pathology or the specific parental personality trait (Table 5).

INSERT TABLE 5 ABOUT HERE
4. Discussion

The present study examined the nature of the associations between mood disorders and personality traits using data from a controlled family study, which also included 9 to 17 year-old offspring. Samples of young offspring of affected parents are particularly suitable for the study of premorbid personality characteristics given that disorders are not yet manifest at this age, but a sizable proportion of these children will later develop disorders. The high risk design allows us to study both the intra-individual temporal sequence of the manifestations of mood disorders and abnormal personality traits and the inter-generational patterns of cross-transmission of mood disorders and personality traits. The combination of these two sets of information can provide precious clues to the mechanisms underlying the intra-individual associations between mood disorders and personality traits.

The results of the present study confirmed these associations in both adults and offspring. Specifically, the findings were in line with those of previous research, which evidenced an association between current depressive episodes in unipolars and increased levels of Neuroticism in both adults (Kendler et al., 1993; Ormel et al., 2004). A similarly strong association between current depressive episode and Neuroticism was also observed in bipolar probands. As Neuroticism was also associated with the two types of mood disorders in remitted probands, the observed association could not be entirely attributable to the well-documented state dependence (5th mechanism) of personality features (Akiskal et al., 1983; Reich et al., 1987; Morgado et al., 1991; Kendler et al., 1993; Ormel et al., 2004). The fact that remitted bipolars also revealed increased levels of Neuroticism is of particular interest given the contradictory findings of previous research regarding associations between this disorder and personality traits (Goodwin and Jamison, 1990). As compared to Neuroticism, the present data support weaker and less consistent associations between mood disorders and Extraversion or Psychoticism. In accordance with previous studies (Farmer et al., 2002; Jylha and Isometsa, 2006; Fanous et al., 2007), Extraversion was negatively associated with current depressive episodes in unipolar adults. However, this association was not observed in remitted patients and adolescents. Regarding Psychoticism,
previous studies reported inconsistent results (Van et al., 1998; Rashed et al., 2001; Farmer et al., 2001; Sariusz-Skapska et al., 2003). Interestingly, in this study, Psychoticism was increased in bipolar probands, regardless of the type or the presence of a current mood episode which could suggest a certain specificity of the latter personality trait with respect to the polarity of mood disorders, whereas in children, Psychoticism was associated with current depressive episodes.

The patterns of familial aggregation of personality traits and mood disorders of the present study together with the finding of associations between personality traits and mood disorders not only in patients exhibiting current episodes but also in remitted patients is best compatible with the 6th mechanism, i.e. the complication or scar hypothesis (Kendler et al., 1993; Rohde et al., 1994), which assumes the occurrence of abnormal personality traits as a consequence of previous depressive episodes. Indeed, the present data only supported increased Neuroticism and Psychoticism scores in offspring who already had a history of depression, whereas there was no evidence for the existence of cross-aggregation between personality traits and unipolar or bipolar mood disorder; i.e. personality scores of offspring did non differ according the mood disorder status of probands, and conversely, the risk of mood disorders in offspring was independent of personality traits in probands. The lack of evidence of cross-aggregation undermines the hypotheses that personality traits and mood disorders share a common cause (1st mechanism) or that abnormal personality traits are a precursor of mood disorders (2nd mechanism), as these hypotheses assume that offspring of affected probands exhibit increased levels of abnormal personality traits even if they are not yet affected by a mood disorder. Conversely, the observation that the risk of depression in offspring was independent of the level of personality traits in probands was not consistent with the predisposition / pathoplasticity hypotheses (3rd and 4th mechanisms), which assumes increased levels of abnormal personality traits and, subsequently, an increased risk of depression in the offspring of parents with abnormal personality traits, even if the parents are not affected by a mood disorder.
However, this conclusion should be interpreted in light of several limitations, which could explain contradictions with results of previous studies suggesting that abnormal personality traits could be precursors or a predisposition for mood disorders (Clark et al., 1994; Watson et al., 1994; Shea et al., 1996; Ormel et al., 2004; Christensen & Kessing, 2006) or share common pathogenetic factors with mood disorders (Kendler et al., 1993; Kendler et al., 2006). First of all, the size of the subgroup of not yet affected children, who are most informative regarding the assessment of potential cross-aggregation, was small, which was partially due to the fact that already more than 20% of all offspring reported a depressive episode. For this reason, despite the low mean age of the sample, the proportion of those who will subsequently develop a mood disorder may be small and therefore, even if these adolescents exhibited increased levels of pathological personality traits, their impact on the mean personality scores of the entire sample of still unaffected offspring would be modest.

Another limitation is the cross-sectional design, which reduces the ability to accurately establish the sequence of manifestations of mood episodes and personality traits. Therefore, a study combining the family study with a longitudinal design would be the most promising future approach to further investigate the pathways between personality and mood disorders, which are still poorly understood. Such a study should only include young offspring who do not yet exhibit mood disorders at baseline evaluation.
Reference List


Table 1: Expected findings regarding theoretical pathways

<table>
<thead>
<tr>
<th>Pathways according to Klein et al. 2002</th>
<th>Criteria</th>
<th>Expected findings</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>1. Common cause</strong></td>
<td>Sequence of occurrence</td>
<td>- There is no specific sequence regarding the onset of the mood disorder and the occurrence of abnormal personality traits.</td>
</tr>
<tr>
<td></td>
<td>Disorder and personality traits in offspring by parental characteristics</td>
<td>- Children of affected parents without abnormal personality traits exhibit abnormal personality traits even if they are not affected by a mood disorder; - Children of parents with abnormal personality traits without a mood disorder are at an increased risk of a mood disorder even if they do not exhibit abnormal personality traits.</td>
</tr>
<tr>
<td><strong>2. Precursor</strong></td>
<td>Sequence of occurrence</td>
<td>- Abnormal personality traits typically precede the onset of a mood disorder.</td>
</tr>
<tr>
<td></td>
<td>Disorder and personality traits in offspring by parental characteristics</td>
<td>- Children of affected parents without abnormal personality traits exhibit abnormal personality traits even if they are not (yet) affected by a mood disorder; - Children of parents with abnormal personality traits without a mood disorder are at an increased risk of a mood disorder even if they do not exhibit abnormal personality traits.</td>
</tr>
<tr>
<td><strong>3. Predisposition + 4. Pathplasticity</strong></td>
<td>Sequence of occurrence</td>
<td>- Abnormal personality traits typically precede the onset of a mood disorder.</td>
</tr>
<tr>
<td></td>
<td>Disorder and personality traits in offspring by parental characteristics</td>
<td>- Children of parents with abnormal personality traits without a mood disorder are at an increased risk of a mood disorder only if it is preceded by abnormal personality traits; - Children of affected parents without abnormal personality traits do not exhibit abnormal personality traits.</td>
</tr>
<tr>
<td><strong>5. State dependence</strong></td>
<td>Sequence of occurrence</td>
<td>- The manifestation of abnormal personality traits is limited to the duration of mood episodes.</td>
</tr>
<tr>
<td></td>
<td>Disorder and personality traits in offspring by parental characteristics</td>
<td>- Children of affected parents without abnormal personality traits during mood episodes are at an increased risk of abnormal personality traits only when they are in a current episode; - Children of unaffected parents with abnormal personality traits are not at an increased risk of a mood disorder.</td>
</tr>
<tr>
<td><strong>6. Complications (or scar)</strong></td>
<td>Sequence of occurrence</td>
<td>- The occurrence of abnormal personality traits typically follows the onset of a mood disorder.</td>
</tr>
<tr>
<td></td>
<td>Disorder and personality traits in offspring by parental characteristics</td>
<td>- Children of affected parents without abnormal personality traits during episodes are at an increased risk of abnormal personality traits only after the onset of a mood disorder; - Children of unaffected parents with abnormal personality traits are not at an increased risk of a mood disorder.</td>
</tr>
</tbody>
</table>
Table 2: Associations between mood episodes and personality traits in probands

<table>
<thead>
<tr>
<th>Diagnostic status</th>
<th>Neuroticism</th>
<th></th>
<th></th>
<th>Extraversion</th>
<th></th>
<th></th>
<th>Psychoticism</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (S.D.)</td>
<td>β 1</td>
<td>p</td>
<td>Mean (S.D.)</td>
<td>β 1</td>
<td>p</td>
<td>Mean (S.D.)</td>
<td>β 1</td>
</tr>
<tr>
<td>Currently manic bipolar probands (N=6)</td>
<td>13.2 (8.0)</td>
<td>4.4</td>
<td>n.s.</td>
<td>10.5 (4.1)</td>
<td>-1.2</td>
<td>n.s.</td>
<td>5.5 (3.7)</td>
<td>2.8</td>
</tr>
<tr>
<td>Currently depressed bipolar probands (N=9)</td>
<td>17.2 (6.2)</td>
<td>10.8</td>
<td>&lt;0.001</td>
<td>10.0 (5.7)</td>
<td>-3.1</td>
<td>n.s.</td>
<td>4.2 (3.0)</td>
<td>2.5</td>
</tr>
<tr>
<td>Bipolar probands in remission (N=19)</td>
<td>12.6 (5.6)</td>
<td>6.6</td>
<td>&lt;0.001</td>
<td>11.7 (4.9)</td>
<td>-0.8</td>
<td>n.s.</td>
<td>4.3 (3.0)</td>
<td>2.1</td>
</tr>
<tr>
<td>Currently depressed unipolar probands (N=8)</td>
<td>17.0 (6.5)</td>
<td>10.7</td>
<td>&lt;0.001</td>
<td>7.0 (7.0)</td>
<td>-5.4</td>
<td>&lt;0.01</td>
<td>3.0 (2.2)</td>
<td>0.8</td>
</tr>
<tr>
<td>Unipolar probands in remission (N=13)</td>
<td>11.4 (6.2)</td>
<td>4.3</td>
<td>&lt;0.05</td>
<td>10.7 (4.7)</td>
<td>-1.6</td>
<td>n.s.</td>
<td>3.3 (2.0)</td>
<td>1.0</td>
</tr>
<tr>
<td>Controls (N=27)</td>
<td>6.3 (4.0)</td>
<td>-</td>
<td>-</td>
<td>12.3 (3.9)</td>
<td>-</td>
<td>-</td>
<td>2.4 (1.8)</td>
<td>-</td>
</tr>
</tbody>
</table>

1 Parameters from multiple regression, controlled for sex and age.
Table 3: Associations between mood episodes and personality traits in offspring

<table>
<thead>
<tr>
<th>Diagnostic status</th>
<th>Neuroticism</th>
<th>Extraversion</th>
<th>Psychoticism</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (S.D.)</td>
<td>( \beta^1 )</td>
<td>p</td>
</tr>
<tr>
<td>Currently depressed unipolar offspring (N=6)</td>
<td>12.9 (5.7)</td>
<td>6.9 &lt;0.001</td>
<td>14.1 (6.3)</td>
</tr>
<tr>
<td>Unipolar offspring in remission (N=38)</td>
<td>8.1 (3.9)</td>
<td>2.3 &lt;0.001</td>
<td>16.7 (4.3)</td>
</tr>
<tr>
<td>Offspring without lifetime history of depression (N=134)</td>
<td>5.7 (3.6)</td>
<td>- -</td>
<td>17.6 (3.4)</td>
</tr>
</tbody>
</table>

\(^1\) Parameters from multilevel (mixed) linear models, controlled for sex and age.
Table 4: Personality traits in offspring by proband disorder

| Offspring sample                | Diagnostic status of proband | Mean (S.D.) | $\beta^1$ | p    | Mean (S.D.) | $\beta^1$ | p    | Mean (S.D.) | $\beta^1$ | p    |
|--------------------------------|------------------------------|-------------|-----------|------|-------------|-----------|------|-------------|-----------|------|------------------|
| All offspring                  | Bipolar (N=72)               | 5.9 (3.6)   | -0.45     | n.s. | 17.5 (4.2)  | -0.26     | n.s. | 2.0 (1.9)    | 0.15      | n.s. |
|                               | Unipolar (N=55)              | 7.3 (4.4)   | 0.84      | n.s. | 16.7 (4.0)  | -0.76     | n.s. | 2.6 (2.4)    | 0.80      | n.s. |
|                               | Control (N=51)               | 6.2 (4.1)   | -         | -    | 17.6 (2.8)  | -         | -    | 1.9 (2.1)    |            |      |
| Offspring with lifetime        | Bipolar (N=18)               | 8.6 (4.4)   | -0.11     | n.s. | 16.1 (5.7)  | -1.54     | n.s. | 2.1 (1.6)    | 0.94      | n.s. |
| depression                     | Unipolar (N=15)              | 9.1 (4.6)   | -0.99     | n.s. | 16.0 (4.2)  | -0.76     | n.s. | 2.4 (2.5)    | 1.69      | n.s. |
|                               | Control (N=11)               | 8.6 (4.7)   | -         | -    | 17.3 (3.3)  | -         | -    | 1.1 (1.3)    |            |      |
| Offspring without lifetime     | Bipolar (N=54)               | 5.0 (2.8)   | -0.42     | n.s. | 17.9 (3.5)  | 0.06      | n.s. | 2.0 (2.0)    | -0.07     | n.s. |
| depression                     | Unipolar (N=40)              | 6.7 (4.3)   | 1.16      | n.s. | 17.0 (3.9)  | -0.82     | n.s. | 2.7 (2.4)    | 0.56      | n.s. |
|                               | Control (N=40)               | 5.5 (3.6)   | -         | -    | 17.7 (2.7)  | -         | -    | 2.1 (2.2)    |            |      |

$^1$ Parameters from multilevel (mixed) linear models, controlled for sex and age in children as well as sex in probands.
Table 5: Lifetime history of a major depressive episode in offspring by diagnosis and level of personality traits in probands

<table>
<thead>
<tr>
<th>Offspring sample</th>
<th>Level of personality traits in probands</th>
<th>Lifetime history of major depressive episode in offspring</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>%</td>
</tr>
<tr>
<td>Offspring of bipolar probands (n=49)</td>
<td>Neuroticism ≥ median (n=25)</td>
<td>24</td>
</tr>
<tr>
<td></td>
<td>Neuroticism &lt; median (n=24)</td>
<td>17</td>
</tr>
<tr>
<td></td>
<td>Extraversion ≥ median (n=19)</td>
<td>32</td>
</tr>
<tr>
<td></td>
<td>Extraversion &lt; median (n=27)</td>
<td>13</td>
</tr>
<tr>
<td></td>
<td>Psychoticism ≥ median (n=25)</td>
<td>24</td>
</tr>
<tr>
<td></td>
<td>Psychoticism &lt; median (n=24)</td>
<td>17</td>
</tr>
<tr>
<td>Offspring of unipolar probands (N=31)</td>
<td>Neuroticism ≥ median (n=18)</td>
<td>33</td>
</tr>
<tr>
<td></td>
<td>Neuroticism &lt; median (n=13)</td>
<td>31</td>
</tr>
<tr>
<td></td>
<td>Extraversion ≥ median (n=10)</td>
<td>30</td>
</tr>
<tr>
<td></td>
<td>Extraversion &lt; median (n=21)</td>
<td>33</td>
</tr>
<tr>
<td></td>
<td>Psychoticism ≥ median (n=15)</td>
<td>33</td>
</tr>
<tr>
<td></td>
<td>Psychoticism &lt; median (n=16)</td>
<td>31</td>
</tr>
<tr>
<td>Offspring of control probands (N=40)</td>
<td>Neuroticism ≥ median (n=29)</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Neuroticism &lt; median (n=11)</td>
<td>22</td>
</tr>
<tr>
<td></td>
<td>Extraversion ≥ median (n=20)</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td>Extraversion &lt; median (n=20)</td>
<td>25</td>
</tr>
<tr>
<td></td>
<td>Psychoticism ≥ median (n=10)</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Psychoticism &lt; median (n=30)</td>
<td>27</td>
</tr>
</tbody>
</table>

¹ ORs from generalized linear models (GEE), controlled for sex and age of children as well as sex of probands.

§ ORs could not be calculated.