Creativity in familial bipolar disorder

Diana I. Simeonova, Kiki D. Chang *, Connie Strong, Terence A. Ketter

Department of Psychiatry and Behavioral Sciences, Division of Child and Adolescent Psychiatry, Stanford University School of Medicine, 401 Quarry Road, Stanford, CA 94305 5540, USA

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Abstract

Studies have demonstrated relationships between creativity and bipolar disorder (BD) in individuals, and suggested familial transmission of both creativity and BD. However, to date, there have been no studies specifically examining creativity in offspring of bipolar parents and clarifying mechanisms of intergenerational transmission of creativity. We compared creativity in bipolar parents and their offspring with BD and bipolar offspring with attention-deficit/hyperactivity disorder (ADHD) with healthy control adults and their children. 40 adults with BD, 20 bipolar offspring with BD, 20 bipolar offspring with ADHD, and 18 healthy control adults completed the Barron–Welsh Art Scale (BWAS), an objective measure of creativity. Adults with BD compared to controls scored significantly (120%) higher on the BWAS Dislike subscale, and non-significantly (32%) higher on the BWAS Total scale. Mean BWAS Dislike subscale scores were also significantly higher in offspring with BD (107% higher) and offspring with ADHD (91% higher) than in healthy control children. Compared to healthy control children, offspring with BD had 67% higher and offspring with ADHD had 40% higher BWAS Total scores, but these differences failed to reach statistical significance when adjusted for age. In the bipolar offspring with BD, BWAS Total scores were negatively correlated with duration of illness. The results of this study support an association between BD and creativity and contribute to a better understanding of possible mechanisms of transmission of creativity in families with genetic susceptibility for BD. This is the first study to show that children with and at high risk for BD have higher creativity than healthy control children. The finding in children and in adults was related to an enhanced ability to experience and express dislike of simple and symmetric images. This could reflect increased access to negative affect, which could yield both benefits with respect to providing affective energy for creative achievement, but also yield liabilities with respect to quality of interpersonal relationships or susceptibility to depression.

Keywords: Creativity; Bipolar disorder; Mood disorders

1. Introduction

Many eminently creative individuals have been retrospectively diagnosed with mood disorders, suggesting relationships between creativity and affective disorders. Jamison (1989) described several research paradigms used to study relationships between mood disorders and creativity. A common approach uses historical and biographical studies to provide anecdotal evidence for high rates of affective illness in eminently creative individuals, suggesting artists and writers may have a 2–3-fold more psychosis, mood disorders and suicide compared to people in less creative professions (Ludwig, 1992, 1994, 1995; Jamison, 1993; Post, 1994, 1996; Schildkraut et al., 1994).

Another approach involves assessing living artists and writers. For instance, in a sample of 30 writers, 80% were found to have had an episode of affective...
illness at some time in their lives (Andreasen, 1987). Also, a higher incidence of mania and hypomania was found in writers (43%) compared to a control group (10%). First-degree relatives of writers also reported higher rates of psychopathology, especially major depressive disorder, as well as higher levels of creativity (20%) compared with the relatives of controls (8%). Furthermore, another study reported that 38% of a group of 47 British prizewinning writers were diagnosed with affective disorders (Jamison, 1989).

Yet another approach examines relationships between creativity and affective illness by measuring creativity in patients with mood psychopathology. Richards and colleagues (1988) examined “everyday creativity” using the Lifetime Creativity Scale (LCS), which assesses creative accomplishments based on vocational and avocational activities. Richards reported that adults with cyclothymia and first-degree relatives of patients with bipolar disorder (BD), but not patients with BD themselves, had significantly higher LCS scores compared to controls. This study is notable in having examined creativity in a general clinical sample by using a standardized measure rather than identifying creativity by eminence.

Another psychometric scale that has been used to study creativity is the Barron–Welsh Art Scale (BWAS) (Barron and Welsh, 1952; Barron, 1963). The scoring of this instrument is based on “like” and “dislike” responses to figures of varying complexity and symmetry compared to preferences of creative individuals. Although there is some controversy regarding the exact type of creativity assessed by this measure (Ridley, 1977a,b, 1979), due to its perceptual preference this test may be viewed as a measure of enhanced perceptual creativity (Santosa et al., 1999) or ability to experience and mobilize negative affect (Strong and Ketter, 2002; Strong et al., 2003). Santosa and colleagues (Santosa et al., 1999) compared BWAS scores and various measures of temperament in euthymic adults with BD, euthymic adults with unipolar depression, healthy controls, and creative controls recruited from graduate programs in fine arts, creative writing, and product design. Patients with BD and creative controls scored similarly and significantly higher than healthy controls and unipolar depressed adults on the BWAS Total score, with these differences being driven by particularly robust increases in BWAS Dislike subscale scores, reporting a greater dislike of symmetrical and simple figures than the controls.

The aforementioned research suggests relationships between creativity and affective illness, specifically BD. However, definitions of and metrics used to assess noneminent creativity remain controversial, adding to the complexity of studying this phenomenon. Nevertheless, the question of whether or not BD is linked to creativity may be better addressed by studying noneminent creativity in patients with BD rather than eminently creative individuals, as such an approach should yield more generalizable findings relevant to common clinical populations.

Although there is substantial evidence of an association between creativity and BD, the nature of this relationship remains to be established. It is possible that creativity and BD have important genetic components that are transmitted together intergenerationally. However, while familial aggregation and genetic transmission of BD has been well established and demonstrated through family, twin, and adoption studies (for review see Faraone et al., 2003), there only have been limited studies investigating whether creativity is genetically transmitted along with BD. For example, Coryell and colleagues found enhanced educational and occupational achievement in first-degree relatives of patients with BD compared to controls (Coryell et al., 1989). Further, to our knowledge there have been no published genetic studies of creativity in non-BD samples. One hypothesis is that BD “causes” creativity, and that hypomania or mania fuels creative activity. However, BD and creativity could be independently transmitted from parents to children. In addition, it is possible that the genes for BD and creativity are linked and co-segregate through generations, accounting for their co-occurrence in people with BD. Other factors such as family environment might modulate this putative familial co-transmission of creativity and BD.

To clarify the interplay between BD and creativity, it may be helpful to study creativity in patients before their onset of BD, as this would address whether or not hypomania or mania is necessary to be creative. Bipolar offspring are at high-risk for the development of BD and other psychopathology, including behavioral and anxiety disorders (e.g., attention-deficit/hyperactivity disorder (ADHD), oppositional defiant disorder, generalized anxiety disorder) (Todd et al., 1996; Chang et al., 2000; DelBello and Geller, 2001; Wals et al., 2001; Chang et al., 2003a,b). A meta-analysis (Lapalme et al., 1997) reported that bipolar offspring compared with children of healthy parents were at 4-fold greater risk of having a mood disorder and at 2.7 times higher risk of developing any psychiatric disorder. Further, the incidence of bipolar spectrum disorders, such as bipolar I, bipolar II, and cyclothymia, varies between 14% and 50% in this population (Chang et al., 2003a,b). It is unclear which of these high-risk children will eventually develop BD, but several investigators have reported on putative prodromal symptoms of BD in bipolar offspring. ADHD in children of bipolar parents could be an early sign of a later BD (Faraone et al., 1997a,b; Chang et al., 2003a,b). Also, high rates of behavior and attention problems were found to be predictive of the development of affective disorders in bipolar offspring (Carlson and Weintraub, 1993). Thus,
studying bipolar offspring with behavioral and mood problems but not BD may yield trait information before the onset of a first manic episode.

The purpose of this study was to examine one facet of creativity in a group of bipolar adults and their offspring compared to healthy controls using the BWAS. Based on previous findings we predicted that adults with BD would score higher on the BWAS than controls (Santosa et al., 1999) and that this elevation would be driven by higher scores on the BWAS Dislike subscale (Strong et al., 2003). We divided the children into three diagnostic groups: bipolar offspring with BD, bipolar offspring with ADHD, and healthy controls. We hypothesized that bipolar offspring would have higher creativity scores than control children, based on findings of increased creativity in first-degree relatives of writers and patients with affective disorders (Andreasen, 1987; Richards et al., 1988) and on the notion that creativity might be transmitted through genetic and possibly familial mechanisms. We hypothesized that bipolar offspring with BD would have the highest BWAS scores, followed by bipolar offspring with ADHD, and then by healthy controls. We felt that bipolar offspring with ADHD, representing a group of children with possible early signs of BD, would have intermediate BWAS scores. This is consistent with the proposition that genetic contributions to BD and creativity may co-segregate through generations and could be enhanced by family environment, and thus be expressed in the absence of full hypomania or mania. Although hypomania or mania might initially enhance creativity, yielding particularly high BWAS scores in bipolar offspring with BD, the debilitating effect of recurrent manic episodes could undermine creativity. Thus, we predicted that duration of bipolar illness in bipolar offspring would be negatively correlated with BWAS scores.

2. Method

2.1. Design

The clinical sample was drawn from an ongoing phenomenology study of bipolar offspring. Families were recruited from the Stanford Adult Bipolar Disorders Clinic, the Stanford Pediatric Bipolar Disorders Program, physician referrals, local adult bipolar support groups, and from the surrounding community. Control families were recruited via advertisements in the local press. Subjects were enrolled from 40 families having at least one parent with BD and from 18 healthy control families.

This investigation was conducted in accordance with the latest version of the Declaration of Helsinki. The study was approved by the Stanford University Administrative Panel on Human Subjects, and all subjects provided written informed consent prior to participation.

2.2. Assessment and diagnosis

After obtaining oral and written informed consent from parents and oral and written assent from their offspring, semi-structured interviews were conducted. Parents were diagnosed using the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID) (First et al., 1995), and were interviewed for psychiatric history of first- and second-degree relatives following the Family History-Research Diagnostic Criteria (FH-RDC) (Andreasen et al., 1988). Bipolar offspring were assessed using the Affective Disorders Module of the Washington Schedule for Affective Disorders and Schizophrenia for School-Age Children (WASH-U-KSADS) (Geller et al., 1996, 2001). Healthy control children were assessed using the Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime (K-SADS-PL) (Kaufman et al., 1997). Diagnoses were made according to DSM-IV (American Psychiatric Association, 1994) criteria. All evaluations were conducted by either a child and adolescent psychiatrist or a master’s level research assistant, with 3 years experience in psychiatric interviewing. Inter-rater reliability was established by rating videotaped interviews, observing trained rater, observing trained rater interviews, and performing interviews with observation by a trained rater, as outlined by Geller and colleagues (Geller et al., 1998). Both the parents and the children were interviewed. Diagnostic decisions were ultimately made by a board-certified child and adolescent psychiatrist based on personal interview, discussion with the research assistant and written notes of parental and offspring responses to interview questions.

Inclusion criteria for the parents and the group of offspring with BD required a diagnosis of bipolar I or bipolar II disorder. The offspring with ADHD required a diagnosis of ADHD (combined type) and mild to moderate affective symptoms, as determined by a score of at least 10 on the Young Mania Rating Scale (YMRS) (Young, 1978) or 30 on the Children’s Depression Rating Scale-Revised (CDRS-R) (Poznanski and Mokros, 1995). The offspring with ADHD did not meet criteria for bipolar I or II disorder. Subjects with comorbid pervasive developmental disorders, neurologic diseases such as seizure disorders, or mental retardation were excluded. Healthy controls had no family history of psychiatric illness in first-degree relatives and no history of BD in first or second-degree relatives. The required age range for children was 9–18 years of age.

Demographic data collected included age, gender, parental occupation and level of education, household income, ethnic status, birth order and number of siblings, and parental and child age of illness onset. The socioeconomic status of the families was measured using the Four Factor Index of Social Status (Hollingshead and Redlich, 1958).
2.3. Barron–Welsh Art Scale (BWAS)

Parents and children completed the Barron–Welsh Art Scale (BWAS) (Barron and Welsh, 1952; Barron, 1963). This is a measure of creativity consisting of 86 3–5 in. figures of varying complexity, which were selected from 400 figures comprising the Welsh Figure Preference Test (WFPT) (Welsh, 1949, 1980). All together the BWAS consists of 62 WFPT figures that best discriminate artists from control subjects and 24 figures that are not scored. Thirty-eight simple and/or symmetrical figures disliked by artists, and 24 more complex and/or asymmetrical figures liked by artists comprise the BWAS Dislike and Like subscales, respectively, the sum of which yields the BWAS Total score. Higher scores on these scales, resulting from disliking simple and symmetrical and liking complex and asymmetrical designs, are considered to reflect a higher degree of creativity. The responses to the pictures are consistent within non-clinical samples with reliability at or above 0.90 (Gough et al., 1996).

BWAS scores of creative individuals have been demonstrated to be correlated with other indicators of creativity (Gough et al., 1996). Although the BWAS became used to measure creativity with appreciation of the more complex pictures indicating greater creativity, there is controversy surrounding its use, especially with regard to validity and the specific dimension of creativity as- 

\[ \text{BWAS scores} \]

2.4. Statistical methods

Statistical analyses were performed using SPSS 11.5. Analyses of variance (ANOVA) were used to compare the two groups of adults (bipolar adults and controls) and the three groups of children (bipolar offspring with BD, bipolar offspring with ADHD, and controls) on the BWAS Dislike and BWAS Like subscales, and the BWAS Total score. Also, analyses of covariation (ANCOVA) were performed when appropriate. The BWAS data of the parents and the children were not normally distributed and were therefore normalized using log transformation. Spearman correlation coefficients were calculated to assess the relationship between duration of bipolar illness and creativity among bipolar offspring with BD. The alpha was set at \( \alpha = 0.05 \) for all analyses.

3. Results

3.1. Cohort

Of the 40 parents with BD, the 33% who had bipolar I disorder were 38% female and the 67% who had bipolar II disorder were 96% female. The control group consisted of 18 parents, 89% were female.

The BD offspring group included 40 children (28 males, 12 females), 20 with BD (18 subjects with BD I, 2 subjects with BD II) (15 males), and 20 with ADHD (but without BD) (13 males). There were 18 healthy control children (10 males).

Bipolar adults ranged from age 26 to 55 years (mean age = 42.5). Bipolar offspring ranged from age 9 to 18 years (mean age = 13.2). Control parents and their control children ranged from age 31 to 55 years (mean age = 45.1) and from age 10 to 18 years (mean age = 14.5), respectively.

3.2. Comorbidity

Of the bipolar offspring with BD, 100% had comorbid ADHD, 85% had oppositional defiant disorder (ODD), 5% had conduct disorder (CD), and 40% had one or more anxiety disorders (separation anxiety disorder, generalized anxiety disorder, social phobia, obsessive-compulsive disorder, or post-traumatic stress disorder). Of the bipolar offspring with ADHD but without BD, 50% had comorbid major depressive disorder or dysthymia, 50% had ODD, 5% had CD, and 45% had anxiety disorders. These were lifetime diagnoses, with the majority of diagnoses being current at the time of evaluation.

3.3. Demographic information

Descriptive statistics including mean age, gender distribution, socioeconomic status (SES), and ethnic distribution of each diagnostic group, and current mood state of subjects appear in Table 1. There were no significant differences between the two parental groups in age (\( F = 1.78, \text{df} = 1, p = 0.19 \)), gender (\( \chi^2 = 0.31, \text{df} = 1, \)).
Table 1
Demographics and comorbid diagnoses of offspring

<table>
<thead>
<tr>
<th></th>
<th>Bipolar adults</th>
<th>Control adults</th>
<th>Bipolar offspring with BD</th>
<th>Bipolar offspring with ADHD</th>
<th>Control children</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>N</strong></td>
<td>40</td>
<td>18</td>
<td>20</td>
<td>20</td>
<td>18</td>
</tr>
<tr>
<td>Mean age, years (SD)</td>
<td>42.5 (6.5)</td>
<td>45.1 (7.5)</td>
<td>13.9 (2.8)</td>
<td>12.4 (2.2)</td>
<td>14.5 (2.7)</td>
</tr>
<tr>
<td>Gender, % male</td>
<td>9 (23)</td>
<td>2 (11)</td>
<td>15 (75)</td>
<td>13 (65)</td>
<td>10 (56)</td>
</tr>
<tr>
<td>SES (SD)</td>
<td>4.1 (0.8)</td>
<td>4.5 (0.8)</td>
<td>4.1 (0.9)</td>
<td>4.1 (0.8)</td>
<td>4.5 (0.8)</td>
</tr>
<tr>
<td><strong>Race (%)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>African-American</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>1 (5)</td>
<td>1 (5)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Hispanic</td>
<td>1 (3)</td>
<td>1 (6)</td>
<td>0 (0)</td>
<td>2 (10)</td>
<td>1 (6)</td>
</tr>
<tr>
<td>Asian</td>
<td>2 (5)</td>
<td>4 (22)</td>
<td>0 (0)</td>
<td>3 (15)</td>
<td>3 (16)</td>
</tr>
<tr>
<td>Caucasian</td>
<td>37 (92)</td>
<td>13 (72)</td>
<td>19 (95)</td>
<td>14 (70)</td>
<td>14 (78)</td>
</tr>
<tr>
<td><strong>Comorbid diagnoses of offspring (%)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anxiety disorder</td>
<td>–</td>
<td>–</td>
<td>9 (40)</td>
<td>9 (45)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Depressive disorder</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>10 (50)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>ADHD</td>
<td>–</td>
<td>–</td>
<td>20 (100)</td>
<td>20 (100)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>ODD</td>
<td>–</td>
<td>–</td>
<td>17 (85)</td>
<td>10 (50)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>CD</td>
<td>–</td>
<td>–</td>
<td>1 (5)</td>
<td>1 (5)</td>
<td>0 (0)</td>
</tr>
<tr>
<td><strong>Mood state (SD)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>YMRS</td>
<td>–</td>
<td>–</td>
<td>16.4 (9.4)</td>
<td>14.5 (4.8)</td>
<td>–</td>
</tr>
<tr>
<td>CDRS-R</td>
<td>–</td>
<td>–</td>
<td>41 (13.6)</td>
<td>33.9 (8)</td>
<td>–</td>
</tr>
</tbody>
</table>

Anxiety disorder = separation anxiety disorder, generalized anxiety disorder, social phobia, obsessive-compulsive disorder, or post-traumatic stress disorder; Depressive disorder = major depressive disorder or dysthymia; ADHD = attention-deficit/hyperactivity disorder; ODD = oppositional defiant disorder; CD = conduct disorder; YMRS = Young Mania Rating Scale; CDRS-R = Children’s Depression Rating Scale-Revised.

$p = 0.31$), or SES (Mann–Whitney: $X^2 = -1.69$, $p = 0.09$).

Omnibus ANOVA ($F = 3.3$, $df = 2$, $p = 0.04$) indicated age differences among the three diagnostic groups of children; bipolar offspring with ADHD were about two years younger than healthy control children ($p = 0.02$). There were no gender differences ($X^2 = 1.6$, $df = 2$, $p = 0.45$) or SES differences by group (Kruskal–Wallis: $X^2 = 2.8$, $df = 2$, $p = 0.24$).

Table 2 represents the BWAS mean scores and standard deviations.

4. BWAS

4.1. Parents

Parents with BD compared to controls had nonsignificantly (32%) higher BWAS Total scores (ANOVA: $F = 2.7$, $df = 1$, $p = 0.10$), and significantly (120%) higher BWAS Dislike subscale scores ($F = 5.3$, $df = 1$, $p = 0.02$).

Fig. 1 represents the BWAS percentage difference from healthy controls and Fig. 2 represents the mean differences found between the two groups of parents on the BWAS Total score and BWAS Dislike subscale.

4.2. Children

ANOVA indicated mean differences between groups on the BWAS Dislike scale ($F = 4.2$, $df = 2$, $p = 0.02$), but not on the BWAS Like scale ($F = 0.5$, $df = 2$, $p = 0.63$). Planned follow-up comparisons (Bonferroni) showed that offspring with BD scored 107% higher than the control group on the BWAS Dislike scale ($p = 0.04$). Likewise, offspring with ADHD scored 91% higher than the control group on the BWAS Dislike scale ($p = 0.04$) (Fig. 1). There were group mean differences on the BWAS Dislike scale also after covarying for age ($F = 3.1$, $df = 3$, $p = 0.04$) (Fig. 3). ANOVA indicated statistically significant mean differences between groups on the BWAS Total scale ($F = 3.4$, $df = 2$, $p = 0.04$) with offspring with BD scoring 67% and offspring with ADHD scoring 40% higher than healthy control.

Table 2
BWAS mean scores and standard deviations

<table>
<thead>
<tr>
<th></th>
<th>Bipolar adults</th>
<th>Control adults</th>
<th>Bipolar offspring with BD</th>
<th>Bipolar offspring with ADHD</th>
<th>Control children</th>
</tr>
</thead>
<tbody>
<tr>
<td>(n=40) mean (SD)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BWAS Total score</td>
<td>20.2 (12.4)</td>
<td>15.3 (9.5)</td>
<td>27.1 (12.9)</td>
<td>22.6 (10)</td>
<td>16.2 (10.4)</td>
</tr>
<tr>
<td>BWAS Dislike score</td>
<td>11.9 (10.4)</td>
<td>5.4 (6.3)</td>
<td>15.7 (10.4)</td>
<td>14.5 (9.6)</td>
<td>7.6 (7.6)</td>
</tr>
<tr>
<td>BWAS Like score</td>
<td>8.2 (6.1)</td>
<td>9.9 (5.4)</td>
<td>11.4 (8.1)</td>
<td>8.1 (6)</td>
<td>8.6 (6.4)</td>
</tr>
</tbody>
</table>
Fig. 1. Percentage difference in BWAS Total score and BWAS Dislike score between parents with BD, bipolar offspring, and healthy control adults/children.

Fig. 2. BWAS Total and Dislike scores of bipolar adults and controls. *p < 0.05.

Fig. 3. BWAS Total and Dislike scores of bipolar offspring and controls. *p < 0.05.

Fig. 4. Correlation between BWAS Total score and duration of illness of bipolar offspring with BD.

Fig. 5. Percentage difference in BWAS Total score and BWAS Dislike score between parents with BD, bipolar offspring, and healthy control adults/children.

Fig. 6. BWAS Total and Dislike scores of bipolar adults and controls. *p < 0.05.

Fig. 7. BWAS Total and Dislike scores of bipolar offspring and controls. *p < 0.05.

5. Discussion

We found that parents with BD and their offspring with BD or ADHD had robustly (91–120%) increased BWAS Dislike subscale scores compared to healthy controls, supporting an association between BD and creativity, and consistent with co-transmission of creativity and affective disturbance. We also found more modest (32–67%) nonsignificant increases in BWAS Total scores in parents with BD and their offspring with BD or ADHD compared to healthy controls. These results are consistent with findings of previous studies indicating increased BWAS Total scores in adults with BD, driven by robustly increased BWAS Dislike subscale scores (Santosa et al., 1999; Strong et al., 2003).

This is the first study to show that bipolar offspring with psychopathology may have higher creativity than healthy control children as assessed by the BWAS. This finding is consistent with results from previous studies reporting higher levels of creativity in the first-degree relatives of writers diagnosed with affective disorder as well as in the first-degree relatives of bipolar and cyclothymic adults (Andreasen, 1987; Richards et al., 1988).

We found significant mean differences in the BWAS Dislike score in the three groups of children, with bipolar offspring with BD and bipolar offspring with ADHD scoring higher than healthy controls. This finding implies a greater dislike of simple and symmetrical figures.
in children with and at high risk for BD, an indication of higher creativity in the two clinical groups. This could reflect increased access to negative affect, which could yield both benefits with respect to providing affective energy for creative achievement, but also yield liabilities with respect to quality of interpersonal relationships or susceptibility to depression. The BWAS Dislike scores were distributed normally and there was no evidence for subgroups of individuals who were particularly creative. It could be that the lack of significant differences between the three pediatric and two adult groups on the BWAS Total score was due to limited statistical power given our small sample size.

Contrary to our hypotheses, the BWAS scores of bipolar offspring with ADHD did not differ significantly from the scores of bipolar offspring with BD, although the latter were modestly higher. This similarity might be attributed to the bipolar offspring with ADHD being a high-risk group for developing BD and thus possibly prodromal for full BD development (Chang et al., 2003a,b). Therefore, they may share similar neurobiological, genetic, temperamental, and psychological characteristics of children with BD contributing to creativity scores similar to offspring with BD and higher than healthy control children. For instance, bipolar offspring with BD and bipolar offspring with ADHD were found to be similar on several scales of the Child Behavior Checklist (CBCL), an instrument measuring social competence and behavior problems in children (Dienes et al., 2002). Further, this outcome suggests that mild to moderate affective disturbance could contribute to the expression of creativity. This is consistent with observations that adult creative controls have substantial temperamental overlap with adults with BD (Nowakowska et al., in press). It is also possible that there is an underlying environmental component to creativity responsible for the similarity in the creativity scores of the two groups of bipolar offspring. As already established, creativity is a complex heterogeneous phenomenon, related to multiple factors. Perhaps genetic aspects, family environment, and putative prodromal symptoms of BD affect creativity in a way similar to the way BD is affected by physiological and environmental stressors, thus resulting in increased creativity. Therefore, in future studies it would be helpful to include comparison groups of children with ADHD only, who are not offspring of bipolar parents, and offspring of unipolar depression patients. Also, studying unaffected bipolar offspring with no psychiatric diagnosis may further clarify the mechanism of transmission of creativity and may provide evidence for a genetic link, or conversely strengthen the extent of environmental contribution. Clearly, further studies are needed to assess the role of genetic and environmental factors in creativity and BD. For example, to our knowledge there have been no published studies investigating whether creativity is genetically transmitted.

As predicted, the results of this study demonstrated a negative correlation between duration of illness and BWAS scores in bipolar offspring with BD. It appears that although bipolar offspring show higher creativity as indicated by BWAS Dislike scores, longer bipolar illness may attenuate this effect. Successive episodes of mania could interfere with creativity in very much the same way these symptoms interfere with school performance and overall psychosocial functioning, and therefore might decrease creativity. However, 95% of bipolar offspring with BD in this study were taking psychotropic medications at the time of assessment. It is possible that prolonged exposure to medications could also contribute to decreased creativity scores.

Limitations of this study include the relatively small sample size. It is possible that this sample size may have accounted for the lack of significant differences in BWAS Total scores. Another limitation is the possible effect of age on the results of the BWAS. Bipolar offspring with ADHD were younger than healthy controls. However, age differences were factored into the analyses, limiting the impact of this potentially confounding factor. Another issue is the use of the BWAS as an assessment of creativity, which measures only one specific aspect of creativity (Ridley, 1977a,b, 1979). The nature of the exact creativity constructs underlying the BWAS remains to be established. Also, despite the demonstrated potential to distinguish between artist and non-artist groups (Welsh, 1975, 1980) some studies have shown only weak to moderate correlations between the BWAS and other measures of creativity (Welsh, 1975; Rump, 1977). Due to the instrument’s nonverbal quality and its perceptual preference, it could be viewed as a measure of enhanced perceptual creativity, thus assessing a unique facet of creativity not reflected in other commonly used creativity tests. More importantly, as noted above recent evidence suggests that the BWAS may have relevance to affective processing, making it of particular interest in assessing relationships between mood and creativity. Although the BWAS has been validated in adults, it remains to be validated in children. Inclusion of a pediatric creative control group in future studies is necessary to begin to address this issue. Another limitation of this study is that bipolar offspring might be more creative largely due to family environment and not genetics, thus being influenced by their parents’ aesthetic views, artistic projects, or direct guidance. Therefore, future research should include assessment of environmental factors in studying the relationship between creativity and BD.

In spite of these limitations, this study is the first to show that children with familial BD have higher creativity than healthy control children. The results of this research provide preliminary insight into the link between BD and creativity and suggest the need for future
clarifications of the mechanisms of intergenerational transmission of creativity.

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