An Extended Swedish National Adoption Study of Bipolar Disorder Illness and Cross-Generational Familial Association With Schizophrenia and Major Depression

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IMPORTANT Information about how risk for bipolar disorder is transmitted across generations and how parental risk for bipolar disorder relates to their children's risk for schizophrenia and major depression is limited.

OBJECTIVE To evaluate the sources of parent-offspring transmission of bipolar disorder and its familial cross-generational association with schizophrenia and major depression.

DESIGN, SETTING, AND PARTICIPANTS Parents and offspring (born 1960-1990) from 4 family types were ascertained from Swedish national samples: intact (offspring, n = 2,175,259), not-lived-with biological father (n = 152,436), lived-with stepfather (n = 73,785), and adoptive (n = 15,624). Data analysis was conducted from October 28, 2019, to January 8, 2020.

EXPOSURES Three sources of parent-offspring resemblance: genes plus rearing, genes only, and rearing only.

MAIN OUTCOMES AND MEASURES Diagnosis of bipolar disorder, broad schizophrenia (ie, schizophrenia as a 3-level variable: unaffected, nonaffective psychosis, and schizophrenia) and major depression obtained from Swedish national registries. Parent-offspring resemblance was assessed primarily by tetrachoric correlation (ie, correlation of liability) and for key results, odds ratios (ORs) from logistic regression. Cross-generational associations of bipolar disorder with broad schizophrenia and major depression were assessed by their transmission from bipolar disorder in parents and transmission to bipolar disorder in offspring.

RESULTS The study population included 2,417,104 individuals of 4 family types (51.8% male and 48.2% female; median age, 41 [range, 25-60] years). For bipolar disorder to bipolar disorder transmission, tetrachoric correlations for 3 types of parent-offspring relationships were statistically homogeneous across family type and mothers and fathers for genes plus rearing (0.25; 95% CI, 0.24-0.26), genes only (0.22; 95% CI, 0.18-0.26), and rearing only (0.07; 95% CI, 0.01 to 0.15). Parallel ORs were 5.20 (95% CI, 4.91-5.50), 3.66 (95% CI, 2.97-4.51), and 1.63 (95% CI, 0.96-2.78). Best-estimate, cross-disorder tetrachoric correlations for 3 types of parent-offspring relationships for bipolar disorder and broad schizophrenia were 0.12 (95% CI, 0.11-0.13) for genes plus rearing, 0.12 (95% CI, 0.09-0.14) for genes only, and 0.03 (95% CI, 0.01 to 0.04) for rearing only, with parallel ORs of 1.95 (95% CI, 1.93-1.97), 2.04 (95% CI, 1.75-2.38), and 0.76 (95% CI, 0.43-1.35). For bipolar disorder and major depression, the parallel tetrachoric correlations were 0.09 (95% CI, 0.07-0.10) for genes plus rearing, 0.04 (95% CI, 0.01-0.07) for genes only, and 0.05 (95% CI, 0.01-0.08) for rearing only; parallel ORs were 1.53 (95% CI, 1.50-1.57), 1.23 (95% CI, 1.13-1.34), and 1.25 (95% CI, 1.09-1.42). Heritability for bipolar disorder was estimated at 0.44 (95% CI, 0.36-0.48). Genetic correlations were estimated at 0.572 (95% CI, 0.560-0.589) between bipolar disorder and broad schizophrenia and 0.302 (95% CI, 0.001-0.523) between bipolar disorder and major depression.

CONCLUSIONS AND RELEVANCE The findings of this study suggest that genes are largely responsible for bipolar disorder transmission across generations, although modest rearing effects are also likely present. Cross-generational transmission between bipolar disorder and broad schizophrenia appears to be entirely genetic with a moderate genetic correlation; for bipolar disorder and major depression, transmission appears to result equally from genes and rearing with a modest genetic correlation.
A s discussed by Burton in Anatomy of Melancholy in his section, “Parents a Cause by Propagation,” human heredity initially developed as a form of inheritance that included the transmission of wealth, land, and disease propensity from parent to child:

That other inward inbred cause of Melancholy is our temperature [temperament], in whole or part, which we receive from our parents... such as the temperature of the father is, such is the son’s, and look what disease the father had... his son will have after him and [he] is as well inheritor of his infirmities, as of his lands... (p80)

Attempts to quantify the role of genetic and environmental factors in the familial transmission of mood disorders—herein, specifically, bipolar disorder—have relied almost exclusively on within-generation resemblance, largely between twins. In the vast amount of literature on familial transmission of bipolar disorder, one small Belgian adoption study, including 29 individuals with bipolar disorder who had been adopted, focused on cross-generational transmission. However, disease transmission within and across generations does not reflect entirely the same processes. Although twin studies of major depression suggest that genetic factors are associated with nearly all familial resemblance, an expanded adoption study suggested that cross-generational transmission results nearly equally from genetic and rearing effects.

Herein, we report an expanded National Swedish adoption study of clinically diagnosed bipolar disorder. We examine intact nuclear families, biological and adoptive parents from an adoption sample, and families with not-lived-with biological fathers and lived-with stepfathers to examine 2 questions: What is the magnitude of the transmission of bipolar disorder from parents to children and to what degree does the transmission result from genetic vs rearing effects? What is the familial cross-generational association between bipolar disorder and 2 key related psychiatric disorders: schizophrenia and major depression?

Methods

We collected information on individuals from Swedish population-based registers with national coverage linked using each person’s unique identification number which, to preserve confidentiality, was replaced with a serial number by Statistics Sweden, a government-owned authority. We secured ethical approval for this study from the regional ethical review board of Lund University, Malmö, Sweden. As approved by Swedish ethical authorities, informed consent was not obtained from individual participants included in this study.

We used the following sources: the Multi-Generation Register, the Population and Housing Censuses, Swedish Hospital Discharge Register (complete national coverage, 1987-2015, and partial coverage, 1969-1986), and Outpatient Care Register (nearly complete national coverage 2001-2015). Furthermore, we used information from a new primary care registry, a research data set including individual-level information, such as clinical diagnoses based on visits to primary health care centers from 15 of 21 counties, which in 2016 contained 87% of the Swedish population (eMethods in the Supplement).

Nonaffective psychosis, bipolar disorder, major depression, and schizophrenia were identified in the Hospital Discharge, Outpatient (Specialist) Care, and Primary Care Registers. Nonaffective psychosis was identified by International Classification of Diseases, 8th Revision (ICD-8), codes 295.4, 295.7, 297, 298.3, and 298.9; International Classification of Diseases, 9th Revision (ICD-9), codes 295E, 295H, 295W, 297, 298E, 298W, and 298X; and International Statistical Classification of Diseases and Related Health Problems (ICD-10) codes F20, F22, F23, F24, F25, F26, F27, F28, and F29. Bipolar disorder was classified by ICD-8 codes 296.1, 296.3, 296.8, 296.9, and 298.1; ICD-9 codes 296A, 296C, 296D, 296E, 296W, and 298B; and ICD-10 codes F30 and F31. Major depression was classified by ICD-8 codes 296.0, 296.2, 298.0, and 300.4; ICD-9 codes 296B, 298A, and 300E; and ICD-10 codes F32 and F33. Schizophrenia was classified by ICD-8 codes 295.1, 295.2, 295.3, 295.9, and 295.6; ICD-9 codes 295B, 295C, 295D, 295G, and 295X; and ICD-10 codes F200, F201, F202, F203, F205, and F209. In the analyses, we used a hierarchy, outlined in the eMethods in the Supplement, based on the number of diagnoses in the registers. If these were similar, we gave greater weight to the latest diagnoses because they would be based on more information on the course of illness. Our hierarchy required each individual to be assigned only 1 of our 4 possible disorders: bipolar disorder, nonaffective psychosis, schizophrenia, or major depression. The ICD codes for major depression and bipolar disorder included both nonpsychotic and psychotic forms of illness.

The database was created by entering all individuals in the Swedish population born in Sweden in 1960-1990 and included the number of years, during ages 0 to 15 years, that individuals resided in the same household and geographic area as their biological mother, biological father, and possible stepfather. From 1960 to 1985 (every fifth year), we used household identification numbers from the Population and Housing Census to define family types. The household identification
includes all individuals living in the same dwelling. From 1986 onward (every year), we defined family type using the family identification from the total population register. The family identification is defined by related or married individuals registered at the same property. Furthermore, adults registered at the same property who have common children, but are not married, are registered in the same family. We created family types by investigating with whom the offspring shared the same household identification/family identification when they were aged 0 to 15 years. In the detection of stepparents from 1986 onward for an offspring living with their mother, we captured the stepfather only if he was married to the mother of the offspring and/or had a child with her. For the years without this information, we approximated the household with the information from the closest year.

We thereby defined 4 kinds of families: (1) intact (offspring resided from ages 0 to 15 years in the same household with their biological mother and biological father), (2) not-lived-with biological father (offspring never resided in the same household or small community as their biological father), (3) stepfather (children did not reside the entire period from ages 0 to 15 years with their biological father and, from ages 0 to 15 years, resided ≥10 years with a nonbiologically related male 18-50 years older than the child), and (4) adoptive (children adopted when aged <5 years, with information available on both adoptive parents and ≥1 biological parent). Individuals adopted by biological relatives or an adoptive parent living with a biological parent were excluded. To maximize the number of adoptees, we included children born between 1955 and 1990. The not-lived-with biological fathers and stepfathers were defined so that their relationship with their offspring resembled that seen between an adoptee and their biological parent (ie, sharing only genes) and adoptive parent (ie, sharing only rearing environment).

Statistical Analysis

Because of low base rates for schizophrenia, we created a 3-level variable: (1) unaffected, (2) mildly affected (nonaffective psychosis), and (3) severely affected (schizophrenia), which we termed broadly defined schizophrenia (herein, broad schizophrenia). We evaluated the hypothesis that nonaffective psychosis and schizophrenia were disorders of differing severity on the same liability distribution in our largest sample of genetic informative parent-offspring correlations: not-lived-with fathers and offspring. The model fit well: $\chi^2 = 1.5963$, $P = .66$. We examined the tetrachoric correlation between bipolar disorder and major depression, and the polychoric correlation between bipolar disorder and broad schizophrenia in our parent-offspring pairs because this measure of association is easy to interpret in a genetic-epidemiologic context and is insensitive to changes in base rates. We present odds ratios (ORs) for key results from standard logistic models and, where broad schizophrenia was an outcome, used cumulative logistic regression.

To combine results from the different samples, we used the Olkin-Pratt meta-analytical approach. We calculated the combined correlations and $P$ values for the heterogeneity tests that evaluate the null hypothesis that associations are similar across samples. We also calculated the genetic correlation between bipolar disorder and broad schizophrenia and major depression (eMethods in the Supplement provides the formula). Using 2-tailed, unpaired testing, findings were considered significant at $P ≤ .05$.

Data analysis was conducted from October 28, 2019, to January 8, 2020. Statistical analyses were performed using SAS, version 9.4 (SAS Institute Inc) and R, version 3.6.1 (R Foundation for Statistical Computing).

Results

The total sample size of the offspring examined in our 4 family types was 2 417 104 (51.8% male and 48.2% female; median age, 41 [range, 25-60] years) (intact, 2 175 259; not-lived-with biological father, 152 436; lived-with stepfather, 73 785; and adoptive, 15 624) (Table 1). The prevalence of the disorders of interest—bipolar disorder, major depression, nonaffective psychosis, and schizophrenia—is reported in Table 1. Across both parents and offspring, prevalence rates for all disorders were generally lowest in intact families, intermediate in stepfather families, and highest in not-lived-with biological father and adoptive families. However, in the adoptive families, rates were consistently lower in the adoptive than biological parents. Bipolar disorder and major depression were consistently more common in females than males; nonaffective psychosis and schizophrenia rates were relatively similar in the 2 sexes. Given the very low prevalence of schizophrenia in parents from the intact and stepparent families and evidence that nonaffective psychosis and schizophrenia reflect disorders of varying severity on the same liability distribution, we used the 3-category variable—broad schizophrenia—for further analyses.

Table 2 depicts the tetrachoric correlations and 95% CIs between bipolar disorder in parents and bipolar disorder, broad schizophrenia, and major depression in their offspring in each parental type across our 4 kinds of families. When we have multiple estimates of the same correlations (ie, genes plus rearing, genes-only, and rearing-only parent-offspring relationships), the final 2 rows present the weighted estimate and $P$ value of the heterogeneity test. None of the findings of the 9 heterogeneity tests in Table 2 were significant, so we focus herein on weighted estimates.

Our best estimates of the magnitude of bipolar disorder to bipolar disorder transmission to offspring were 0.25 (95% CI, 0.24-0.27) from fathers who provide genes plus rearing, 0.21 (95% CI, 0.17-0.25) from fathers who provide genes only, and 0.06 (95% CI, −0.03 to 0.15) from fathers who provide rearing only. Parallel estimates for mothers and offspring were 0.25 (95% CI, 0.24-0.26) for genes plus rearing, 0.28 (95% CI, 0.17-0.39) for genes only, and 0.10 (95% CI, −0.08 to 0.29) for rearing only. We obtained sparser data for genes-only and rearing-only relationships for mothers, so these estimates are less precise. The weighted estimates across fathers and mothers, for which we found no evidence of statistical heterogeneity, are presented in Table 3 and the Figure. For bipolar disorder
to bipolar disorder, transmission equaled 0.25 (95% CI, 0.24-0.26) for genes plus rearing, 0.22 (95% CI, 0.18-0.26) for genes-only, and 0.07 (95% CI, −0.01 to 0.15) for rearing-only relationships. Parallel ORs were 5.20 (95% CI, 4.91-5.50) for genes plus rearing, 3.66 (95% CI, 2.97-4.51) for genes only, and 1.63 (95% CI, 0.96-2.78) for rearing only.

**Bipolar Disorder**

Patterns of transmission of bipolar disorder in the parental generation to broad schizophrenia and major depression in the offspring generation are reported in Table 2; patterns from broad schizophrenia and major depression in the parental generation to bipolar disorder in the offspring generation are reported in Table 4. For both analyses, results for mothers and fathers were statistically homogeneous (Table 3), as were results for genes-only and rearing-only analyses across both the bipolar disorder in parent and bipolar disorder in offspring designs (eTable 1 in the Supplement). However, in the genes plus rearing analyses, for which very large samples were available, results were mod-
estly but significantly different. The results were in opposite directions with higher correlations for major depression transmission to bipolar disorder than for transmission from bipolar disorder to major depression and for bipolar disorder transmission to broad schizophrenia than broad schizophrenia transmission to bipolar disorder.

Our best estimates for parent-offspring transmission of bipolar disorder and major depression were 0.09 (95% CI, 0.07-
Using all available data on parental genes-only relationships, the cross-generational genetic correlations were 0.302 (95% CI, 0.001-0.523) between bipolar disorder and major depression and 0.572 (95% CI, 0.560-0.589) between bipolar disorder and broad schizophrenia. Genetic correlations are often interpreted to equal the proportion of shared genetic variance between 2 disorders. This correlation, however, is likely better estimated by $r^2$, meaning that the proportion of genetic variance shared is 10% between bipolar disorder and major depression and 33% between bipolar disorder and broad schizophrenia.

Narrow-sense heritability for bipolar disorder can be easily calculated from our results, equalling twice the correlation of genes-only parent-offspring relationships: 0.44 (95% CI, 0.36-0.48). We repeated the analyses conducted for broad schizophrenia using only classic schizophrenia (eMethods, eTable 2, eTable 3, and eTable 4 in the Supplement). Some of the correlations could not be estimated and others have had wide CIs, but the final weighted estimates for bipolar disorder-schizophrenia parent-offspring correlations were nearly identical to those found for broad schizophrenia for parents who provide genes plus rearing (0.12; 95% CI, 0.07-0.16), and rearing only (0.02; 95% CI, −0.13 to 0.16) to their offspring. The genetic correlation calculated between bipolar disorder and schizophrenia equaled 0.44 (95% CI, 0.324-0.510).

### Discussion

We sought to clarify, in an extended adoption design in Swedish national samples, the sources of parent-offspring transmission for bipolar disorder and for the cross-generational transmission between bipolar disorder and broad schizophrenia and major depression. We review our main results in turn.
The historical connection between major depression and bipolar disorder is strong, having been incorporated into Kraepelin’s category of manic-depressive illness and categorized in the same section in DSM-III and DSM-IV. Although the findings were rarely quantified, prior family and twin studies have generally suggested a close familial relationship, with one twin study estimating a genetic correlation of 0.65. Genetic correlations calculated from common single-nucleotide variants have been somewhat smaller, with a mean (SE) of 0.47 (0.06) estimated in one key study. In our analyses, the best weighted estimates for the parent-offspring correlations between bipolar disorder and major depression were 0.09 (OR, 1.53; 95% CI, 1.50-1.57) for parents who provide genes plus rearing, 0.04 (OR, 1.23; 95% CI, 1.13-1.34) for genes only, and 0.05 (OR, 1.25; 95% CI, 1.09-1.42) for rearing only, with all correlations reaching statistical significance. The cross-generational association between these 2 disorders was relatively modest and approximately equally the result of genetic and rearing associations, with their genetic correlation estimated at 0.302. The magnitude and sources of the cross-generational transmission of bipolar disorder to major depression are similar to those found previously for major depression to major depression transmission.9

Using weighted estimates across multiple family types and across mothers and fathers, the tetrachoric correlations for bipolar disorder between parents and offspring were estimated at 0.25 (OR, 5.20) for parents who provide genes plus rearing, 0.22 (OR, 3.66) for those who provide genes only, and 0.07 (OR, 1.63) for those who provide rearing only to their offspring. Our finding for the genes-only association is consistent with what is, to our knowledge, the sole prior adoption study of bipolar disorder. In this study, bipolar disorder was found in 4 of 57 biological parents of children with bipolar disorder who were adopted vs 0 of 42 biological parents of control offspring.6 Our results suggest that a large proportion of the cross-generational transmission of bipolar disorder results from genetic factors. However, results from both the direct examination of rearing-only parents (P = .07) and lower correlations in the genes-only vs genes plus rearing parent-offspring relationships (P = .13) together provide consistent, albeit not definitive, evidence for a contribution of rearing effects to bipolar disorder parent-offspring transmission.

Our estimate of the heritability of bipolar disorder (0.44) is lower than estimates from prior twin samples and the estimate from a large, Swedish twin-sibling study: 0.59. However, most adoption studies produce lower heritability estimates than found in twin analyses for the same phenotype.
Studying schizophrenia on its own in the Swedish registry was challenging because of its rarity, especially in parents, owing to their substantially reduced fecundity. Having shown that nonaffective psychosis and schizophrenia appear to reflect different severities on the same genetic liability distribution, our analyses primarily used a broader 3-category variable: broad schizophrenia.

Bipolar disorder and schizophrenia have traditionally been viewed as largely separate disorders since their conceptualization by Kraepelin, and most family studies have indicated minimal to modest levels of coaggregation. However, our results, consistent with findings from a large-scale twin and sibling study in Sweden, noted more substantial cross-transmission, this time across generations. The best weighted estimates for the parent-offspring correlations between bipolar disorder and broad schizophrenia were 0.12 (OR, 1.95; 95% CI, 1.93-1.97) for parents who provide genes plus rearing, 0.12 (OR, 2.04; 95% CI, 1.75-2.38) for genes only, and –0.03 (OR, 0.76; 95% CI, 0.43 to 1.35) for rearing only. These results are qualitatively different from those found for bipolar disorder and major depression in showing that all of the cross-generational transmission of the liability shared between bipolar disorder and broad schizophrenia appears to be due to genetic factors. The correlations and ORs for bipolar disorder and broad schizophrenia are substantially lower than those seen for bipolar disorder transmission to bipolar disorder, but the genetic correlation between bipolar disorder and broad schizophrenia was estimated at 0.572. These findings are similar to those in a large Swedish twin-sibling study (0.60) and lower than those reported from common single-nucleotide variant analyses: 0.6822 and 0.70. Our results obtained for narrowly defined schizophrenia appear to support our use of broad schizophrenia as a broader definition of schizophrenia.

The statistical homogeneity of our estimates across family types supports the validity of our methodologic assumptions, as does the similarity of father-offspring and mother-offspring correlations. The latter also argue against substantial intraterine effects in the cross-generational transmission of these disorders.

Limitations

These results should be interpreted in the context of 2 methodologic limitations. First, we relied largely on hospital diagnoses for broad schizophrenia and bipolar disorder and a mixture of inpatient and outpatient diagnoses for major depression. Hospital diagnoses of schizophrenia frequently met DSM-IV criteria for schizophrenia in Swedish studies using record reviews (96%) and diagnostic interviews (94%). Hospital diagnoses of bipolar disorder in Sweden have also been validated by record review. The validity of the major depression diagnosis in the Swedish primary care registry was supported by its prevalence, sex ratio, sibling and twin correlations, and associations with well-documented psychosocial risk factors.

Second, longitudinal studies suggest that a modest proportion of cases first diagnosed as bipolar disorder are re-diagnosed at follow-up as schizophrenia, with a smaller proportion shifting from schizophrenia to bipolar disorder. Correcting for this misclassification moderately reduced the bipolar disorder-schizophrenia genetic correlation from the molecular genetic data. In our sample, 17% of individuals with a broad schizophrenia diagnosis also had a bipolar disorder diagnosis and the reverse figure was 18%. To assess misclassification in our findings, we eliminated 4.0% of bipolar disorder, 5.2% of nonaffective psychosis, and 1.9% of schizophrenia cases where we were least certain about the correct diagnostic assignment. Consistent with prior findings, genes-only cross-generational transmission between bipolar disorder and broad schizophrenia decreased from 0.12 to 0.09 and the genetic correlation declined from 0.57 to 0.46 (95% CI, 0.44-0.49).

Conclusions

This expanded national Swedish adoption study has yielded 4 major findings. First, bipolar disorder appears to be transmitted relatively strongly across generations largely through genetic factors, but modest rearing effects are also evident. Second, our findings suggest that the cross-generational transmission of risk from bipolar disorder to major depression seems modest and due almost equally to genetic and rearing effects. Third, parent-offspring transmission between bipolar disorder and broad schizophrenia, by contrast, appears to result entirely from genetic factors, with the 2 disorders having a moderate genetic correlation. Fourth, these results are based on clinical diagnoses and, to our knowledge, a novel extended adoption study design. However, we believe the apparent validity of our findings is supported by their consistency across family types and, with 2 modest exceptions, estimations from transmission from bipolar disorder in parents to bipolar disorder in offspring.

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