

Parental Mental Illness and Eating Disorders in Offspring

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ABSTRACT

Objective: To investigate which parental mental illnesses are associated with eating disorders in their offspring.

Method: We used data from a record-linkage cohort study of 158,679 children aged 12–24 years at the end of follow-up, resident in Stockholm County from 2001 to 2007, to investigate whether different parental mental illnesses are risk factors for eating disorders in their offspring. The outcome measure was diagnosis of any eating disorder, either from an ICD or DSM-IV code, or inferred from an appointment at a specialist eating disorder clinic.

Results: Mental illness in parents is a risk factor for eating disorders in female offspring (Adjusted Hazard Ratio (AHR) 1.57 (95% CI 1.42, 1.92), $p < 0.0001$). Risk of eating disorders is increased if there is a parental diagnosis of bipolar affective disorder (AHR 2.28 (95% CI 1.39, 3.72), $p = 0.004$), personality disorder (AHR

1.57 (95% CI 1.01, 2.44), $p = 0.043$) or anxiety/depression (AHR 1.57 (95% CI 1.32, 1.86), $p < 0.0001$). There is a lack of statistical evidence for an association with parental schizophrenia (AHR 1.41 (95% CI 0.96, 2.07), $p = 0.08$), and somatoform disorder (AHR 1.25 (95% CI 0.74, 2.13), $p = 0.40$). There is no support for a relationship between parental substance misuse and eating disorders in children (AHR 1.08 (95% CI 0.82, 1.43), $p = 0.57$).

Discussion: Parental mental illness, specifically parental anxiety, depression, bipolar affective disorder, and personality disorders, are risk factors for eating disorders in their offspring.
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Keywords: eating disorders; anorexia nervosa; bulimia nervosa; EDNOS; parent; child; mental illness

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Introduction

Eating disorders (ED) predominantly occur in adolescent and young adult women,¹ with a prevalence of over 3% in 12- to 23-year-olds.² ED have severe consequences for patients, including increased mortality, with standardized mortality ratios of 1.7

in anorexia nervosa (AN), 1.57 in bulimia nervosa (BN), and 1.81 in patients with an eating disorder not otherwise specified (EDNOS).³ Eight to ten years after initial diagnosis, between 15 and 30% of patients continue to suffer from an ED^{4–6} and consequent disruption of their ability to function in education, employment, and relationships.

Parental mental illness may increase the risk of a child having a hospital admission with AN, with one longitudinal study finding an adjusted risk ratio of 1.8 (95% CI 1.3, 2.3) for children of parents admitted to psychiatric inpatient care.⁷ The same study did not find an association between parental substance misuse and inpatient care for AN in the offspring. To our knowledge, no previous register-based studies have investigated parental mental illness as a risk factor for diagnosis of any ED in in- and outpatients.

We are unaware of any longitudinal studies investigating specific parental mental illnesses and ED in offspring, but there are many case-control studies which investigate the prevalence of mental illnesses in probands and their first degree relatives and find conflicting results. Case-control studies may be subject to selection bias, and the use of retrospective diagnoses leads to problems with recall bias.

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The numbers in each study are small, which may also contribute to a lack of consistency in the findings. Mental illnesses which have been found to have higher prevalence in relatives of probands with AN include anxiety disorders,⁸ OCD,^{8,9} depression,^{10,11} obsessive compulsive personality disorder,¹² and substance misuse.¹³ One study did not find an association between parental anxiety and AN.¹¹ Mental illnesses which have been found at higher prevalence in relatives of probands with BN include OCD,¹⁴ depression,^{14–17} bipolar affective disorder,¹⁵ personality disorder,¹⁷ and substance misuse.¹⁵ However, another study did not find an association between BN and parental personality disorder or substance misuse.¹⁶ Mental illnesses which have been found at higher prevalence in relatives of probands with any ED include anxiety disorders,¹² OCD,^{12,18} and depression.¹² One study did not find an association with anxiety disorders or depression.¹⁸

Explanations for an association between parental mental illness and offspring eating disorder could be either genetic, environmental, or a combination of the two. The genetic explanation would be that some genes predispose to more than one mental illness. There is some evidence from twin studies in support of this idea, some shared genetic variance for depression and AN,^{19,20} and for depression and BN²¹ have been found. Personality traits, such as harm avoidance, have also been shown to predispose to anxiety, depression, and ED.²² A variation of this idea is that a parental mental illness may be inherited by the child and then predispose them to developing an ED: for example it has been proposed that early onset anxiety disorders might represent a risk factor for developing an ED, with anxiety being heritable.²³

Alternatively, the increase in risk of ED in the children of parents with mental illnesses may be due to the effect on the child of having a parent with a mental illness, and the stress that this causes. A range of adversities in childhood, including physical neglect and sexual abuse, and also low paternal communication with the child, and poverty, are risk factors for developing ED or problems with eating and weight.²⁴ Patients with AN also report greater numbers of negative life events than controls, and greater numbers of negative life events involving parents than those with other mental illnesses.²⁵

The aim of this large population based study is to investigate whether different parental mental illnesses are associated with an increased risk of ED in their offspring. This will resolve previous conflicting results and may also suggest mechanisms whereby different parental mental illnesses may alter the risk of eating disorders in offspring. We

hypothesize that any parental mental illness, and specifically anxiety and depression, will increase the risk of ED in children. We will also investigate whether other parental mental illnesses increase this risk. The study will adjust for important confounders [year of birth of the child, mother's age at the birth of the child, occupational class of the parents, maternal education, family type (single mother/two parent family) and number of siblings] which may potentially confound any association.

METHOD

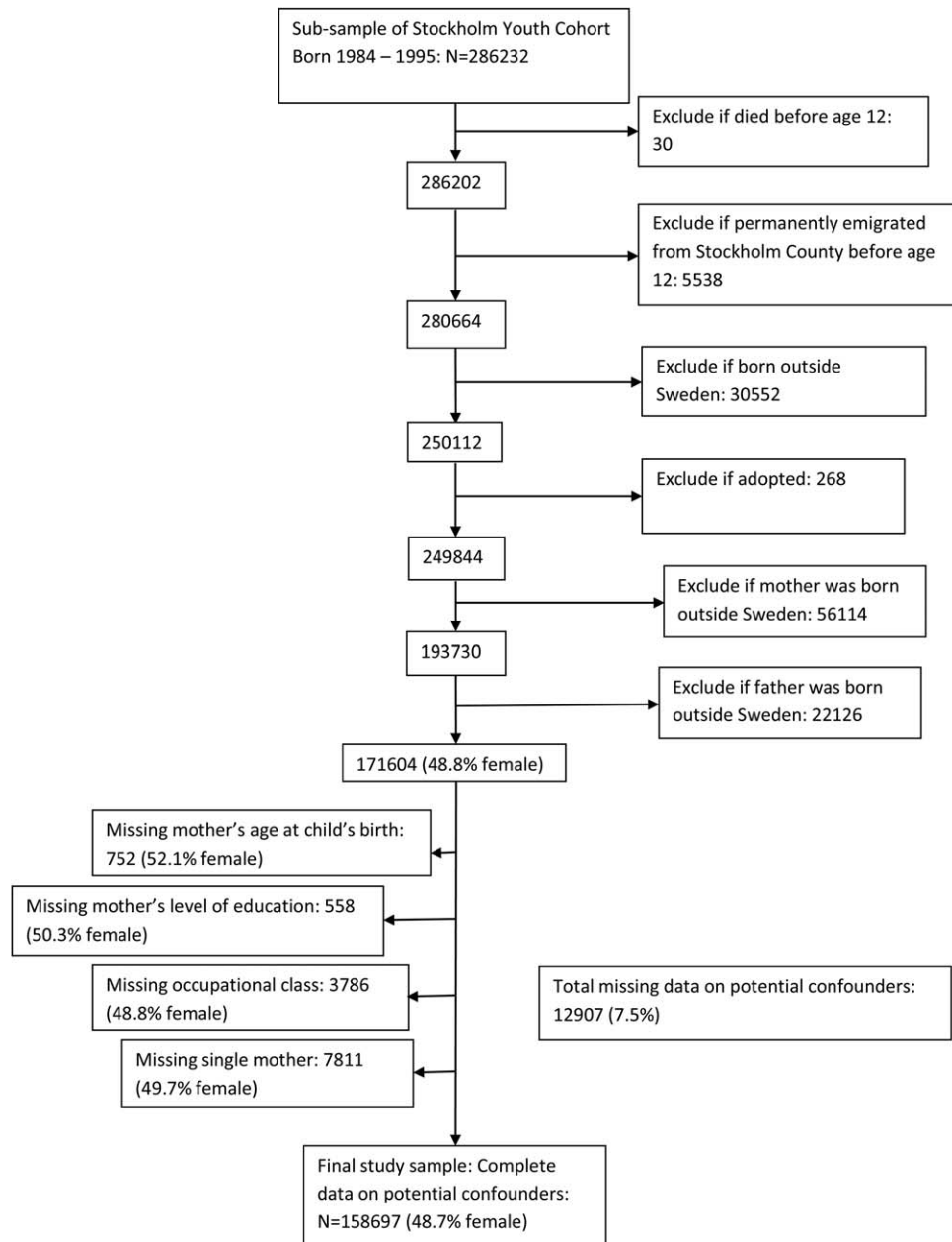
Study Population

The Stockholm Youth Cohort^{26,27} is a register-based study which comprises all children aged 0 to 17 years who were resident in Stockholm County from 2001 to 2007, identified through the *Register of Total Population* (provided by Statistics Sweden). Information on ED, other mental illnesses and covariates was obtained for study participants, and their parents, ascertained via the *The Multi-Generation Register*,²⁸ through record-linkage to national and regional health data and administrative registers using the national registration number assigned to all residents of Sweden.²⁹ Our paper concerning parental ED in this cohort is currently under review. For the purpose of this study, we applied the following inclusion restrictions: year of birth between 1984 and 1995, in order to guarantee that they had reached age 12 before the end of the follow up period; born in Sweden to two parents born in Sweden; and not adopted. This was in order to ensure that we knew who the parents were and would have data on parental mental illnesses. The final analytical sample consisted of 158,679 children (Fig. 1).

Definition of ED Cases

The sources for identifying ED cases were the Clinical Database for Child and Adolescent Psychiatry in Stockholm, which includes diagnostic information according to DSM-IV groupings from child and adolescent psychiatric services since 2001, Stockholm County Council Health Service Use Register (VAL), including information on the clinic attended, available from 1997 onwards, the Stockholm Adult Psychiatric Care Register, which holds information on all publically run adult psychiatric care within Stockholm County, including diagnosis coded according to DSM-IV groupings until 2004, and according to ICD-10 since 2005, and the National Patient Register, with complete information on psychiatric inpatient diagnoses according to the ICD since 1973.³⁰ Participants were identified as having a diagnosis of an eating disorder either if they attended a specialist ED clinic, or if they were given an ICD code indicating diagnosis of any ED

FIGURE 1. Derivation of the analytical sample.



(F50 in ICD-10, 307.B/307.F in ICD-9, 306.5 in ICD-8), or DSM-IV code for any ED by a specialist clinician as an inpatient or outpatient during the period of follow up. The date of first attendance at a specialist ED clinic, or the date of first diagnosis with an ED, whichever was first, was used as the date of diagnosis with an ED. Only diagnoses made after the age of 12 were considered, as feeding difficulties in infants and toddlers are also coded as ED, and the aetiology is likely to be somewhat different between the groups.

Definition of Mental Illnesses in Parents

The registers described above were used to identify mental illness in parents. Diagnosis of any mental illness in each parent throughout their life until the subject child turned 10 years old were included. This was in order to ensure that parental diagnoses precede offspring eating disorders. If a diagnosis was made within this time frame for more than one child within a family, each child would be regarded as having been exposed to parental mental illness.

TABLE 1. Baseline characteristics by parental mental illness (excluding drug/alcohol use)

	All Participants (N = 158,697)	Parental Mental Illness (N = 12,388)	No Parental Mental Illness (N = 146,309)
Maternal age (mean, SD)	29.2(5.0)	29.0 (5.3)	29.2 (5.0)
Parental occupational class (N, % unskilled manual worker)	14,635 (9.2)	2,020 (16.3)	12,615 (8.6)
Maternal education (N, % with >12 years' education)	58,324 (36.8)	3,507 (28.3)	54,817 (37.4)
Single mother (N, %)	5,444 (3.4)	944 (7.6)	4,500 (3.1)
Number of siblings (N, % with no siblings)	23,442 (14.8)	3,104 (25.1)	20,338 (13.9)
Gender (N, % girls)	77,323 (48.7)	5,979 (48.3)	71,344 (48.8)

Other Variables

Record linkage with Statistics Sweden enabled us to obtain data on parental age at child birth (from the *Multi-generation Register*), occupational class at time of child birth according to the Swedish socio-economic classification (unskilled manual workers, skilled manual workers, lower nonmanual workers, intermediate nonmanual workers, higher nonmanual workers, self-employed), and level of education (9 years or less; 10–12 years; more than 12 years (university)), both derived from the *Integrated Database for Labour Market Research* kept by Statistics Sweden,³¹ family type at birth of child (single or two parent family)³² and number of siblings at birth of child. Data on premature birth, birth weight and smoking during pregnancy were ascertained from The Medical Birth Register at the National Board of Health and Welfare.³²

Statistical Analysis

Children entered the study on their twelfth birthday, and were followed up until the first of: diagnosis of ED (the “event”), emigration from Stockholm, death, or 31 December 2007 (the end of the follow-up period). For descriptive purposes, incidence rates of ED diagnosis in the different groups were calculated per 100,000 person years.

We used Cox proportional hazards regression to estimate hazard ratios (HR) and corresponding 95% confidence intervals (CI) of ED diagnosis in those with a parental history of different mental illnesses compared to those without. Multivariable analysis was used in order to adjust for factors which may confound these relationships. The analyses were stratified by year of birth of the participants in order to allow for nonproportional effects of birth cohort. Adjustments were made for maternal age at child's birth, occupational class of the parents, as higher SES has been found to be associated with AN in children,⁷ maternal level of education, as higher parental education has been found to be associated with ED in offspring,^{19,33,34} family type (single or two parent family) and number of siblings, as there is conflicting evidence from case-control studies regarding whether this may alter risk of ED.^{35–37} We checked for evidence of interaction between parental mental disorders and any of the potential confounders. Robust variance estimation using mater-

nal identification number as a cluster variable was performed to account for having multiple siblings from the same family in the analysis sample. Proportionality of hazards assumption was examined graphically using Nelson-Aalen plots, and formally using a statistical test based on Schoenfeld residuals.³⁸ This confirmed the appropriateness of using this method of analysis.

Analysis of any parental mental illness excluding drug or alcohol misuse (to enable comparison with previous studies⁷) was performed separately for fathers and mothers. Sensitivity analysis was performed removing all parents with a diagnosis of an ED, and also including drug and alcohol misuse in the definition of parental mental illness. Analyses were repeated with a hierarchy of parental mental illnesses (available from the corresponding author on request), such that each parent was only counted as having one mental illness (the one highest in the hierarchy). All analyses were performed using STATA version 12. The study was approved by the Stockholm Regional Ethical Review Board.

RESULTS

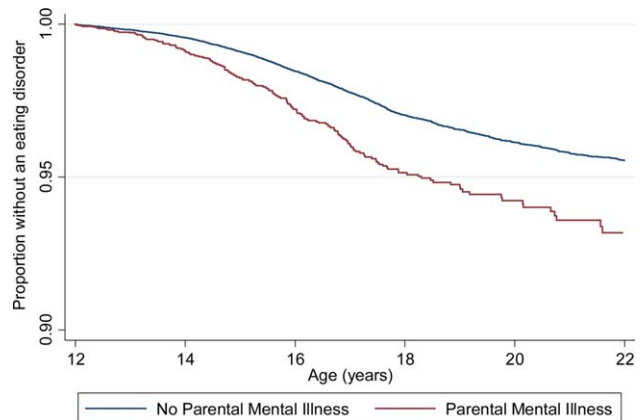
Incidence Rates of ED

Baseline characteristics of subjects with and without parental mental illness (excluding drug/alcohol use) are described in **Table 1**. Study participants (born in 1984–1995) were followed from the age of 12 for an average period of 5.4 years, contributing 886,241 person-years under follow-up, during which 2,131 incident events of ED were identified. This corresponds to an incidence rate of 240 new ED per 100,000 person years (95% CI 230, 251). The incidence was 456 per 100,000 person-years (95% CI 436, 477) in females and 37 per 100,000 person-years (95% CI 32, 34) in males. The incidence peaked between 15 and 17 years at 368 per 100,000 person years in all participants, and 698 per 100,000 person years in girls. It is important to note that the incidence rates quoted are the average over this time period and may have varied over the time period.

TABLE 2. Incidence rates of Any Eating Disorder (ED) in Offspring by Parental Mental Illnesses, overall and by gender (incidence rates per 100,000 person years)

Parental Mental Illness	All Offspring			Female Offspring			Male Offspring		
	Total N (%)	N with ED	Rate of ED (95% CI)*	Total N (%)	N with ED	Rate of ED (95% CI)*	Total N (%)	N with ED	Rate of ED (95% CI)*
Total	158,679 (100)	2,131	240 (230, 251)	77,323 (100)	1,960	456 (436, 477)	81,374 (100)	171	37 (32, 34)
Any parental mental illness including alcohol/drug misuse	15,255 (9.6)	245	341 (301, 387)	7,377 (9.5)	216	623 (545, 712)	78,788 (9.7)	29	78 (54, 112)
Any parental mental illness excluding alcohol/drug misuse	143,442	1,886	232 (221, 242)	69,946	1,744	441 (421, 462)	73,496	142	34 (29, 40)
Schizophrenia	12,388 (7.8)	208	378 (330, 433)	5,979 (7.7)	185	697 (604, 805)	64,097 (7.9)	23	81 (54, 121)
Bipolar	146,309	1,923	231 (221, 242)	71,344	1,775	440 (420, 461)	74,965	148	35 (29, 41)
Personality disorder	1,463 (0.9)	28	342 (236, 495)	714 (0.9)	26	645 (439, 947)	749 (0.9)	2	48 (12, 192)
Anxiety/Depression	157,234	2,103	240 (229, 250)	76,609	1,934	454 (434, 474)	80,625	169	37 (32, 43)
Somatiform Disorder	628 (0.4)	19	647 (413, 1014)	302 (0.4)	16	1,110 (680, 1813)	326 (0.4)	3	201 (65, 622)
Alcohol/Drug misuse	158,069	2,112	239 (229, 250)	77,021	1,944	454 (434, 474)	81,048	168	37 (32, 43)
	1,262 (0.8)	20	338 (218, 523)	603 (0.8)	20	707 (456, 1096)	659 (0.8)	0	-
	157,435	2,111	240 (230, 250)	76,720	1,940	454 (434, 475)	80,715	171	38 (32, 44)
	9,837 (6.2)	160	388 (333, 453)	4,743 (6.1)	141	712 (604, 840)	5,094 (6.3)	19	89 (57, 139)
	148,860	1,971	233 (223, 244)	72,580	1,819	444 (424, 464)	76,280	152	35 (29, 40)
	1,153 (0.7)	16	305 (187, 497)	533 (0.7)	14	565 (335, 954)	620 (0.8)	2	72 (18, 288)
	157,544	2,115	240 (230, 251)	76,790	1,946	455 (435, 476)	80,754	169	37 (32, 43)
	4,425 (2.8)	64	266 (208, 340)	2,151 (2.8)	55	471 (362, 614)	2,274 (2.8)	9	73 (38, 140)
	154,272	2,067	240 (230, 250)	75,172	1,905	455 (435, 476)	79,100	162	36 (31, 43)

* per 100,000 person years

GRAPH 1. Kaplan-Meier Survival Estimates for Eating Disorders in Female Offspring. [Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.]

Incidence rates of ED are reported by parental mental illnesses in **Table 2**, in all participants and also stratified by gender. The percentage of parents with a diagnosis of any mental illness (excluding drug or alcohol misuse) was 7.8%, and the percentage with a diagnosis of drug and alcohol misuse was 2.0%.

Incidence rates of ED were higher when parents had suffered from any mental illness, depression/anxiety, bipolar disorder, or personality disorder. The limited numbers of male cases precluded any meaningful inference; therefore no further analyses were performed in males.

Multivariable Analysis and Hazard Ratios of ED

Graph 1 shows the survival curves for female offspring of parents with and without a mental illness, for those with complete data on confounding variables. The graph does not take account of the effect of confounding variables.

Table 3 shows results from the multivariable analyses of the association between any parental mental illness, excluding drug or alcohol misuse, and offspring incidence of ED, also distinguishing between paternal and maternal mental illness. We found evidence in support of the hypothesis that mental illness in parents is a risk factor for ED in female offspring, with an Adjusted Hazard Ratio (AHR) of 1.57 (1.34, 1.83), $p < 0.0001$. Analyses of both male and female offspring gave similar results. Compared with no parental diagnosis of mental illness, the presence of 2 or more parental diagnoses of mental illnesses gave an AHR of 2.08 (1.59, 2.72), $p < 0.0001$, and the presence of one parental mental illness gave an AHR of 1.42 (1.18, 1.70), $p < 0.0001$.

TABLE 3. Hazard ratios of incidence rates of ED in female offspring by history of parental mental illness (excluding drugs/alcohol misuse)

Parental Mental Illness	Stratified Analysis ^a (HR, 95% CI, <i>p</i> Value) (Including Those Without Data on Confounders: <i>N</i> = 83,694)	Stratified Analysis ^a (HR, 95% CI, <i>p</i> Value) (<i>N</i> = 77,323)	Stratified, Adjusted Analysis ^b (HR, 95% CI, <i>p</i> Value) (<i>N</i> = 77,323)
Any parental mental illness	1.54 (1.32, 1.78), <i>p</i> < 0.0001	1.53 (1.31, 1.78), <i>p</i> < 0.0001	1.57 (1.34, 1.83), <i>p</i> < 0.0001
Any maternal mental illness	1.56 (1.30, 1.88), <i>p</i> < 0.0001	1.51 (1.25, 1.82), <i>p</i> < 0.0001	1.55 (1.28, 1.87), <i>p</i> < 0.0001
Any paternal mental illness	1.60 (1.28, 1.98), <i>p</i> < 0.0001	1.65 (1.33, 2.06), <i>p</i> < 0.0001	1.69 (1.36, 2.11), <i>p</i> < 0.0001

^aStratified by year of birth of child, clustered by mother.

^bStratified by year of birth of child, adjusted for maternal age, occupational class, maternal education (three levels), family type, and number of siblings, clustered by mother.

Table 4 reports estimated hazard ratios for ED in females with different parental mental illnesses. There is an increased risk of ED where one or both parents have a diagnosis of bipolar affective disorder (AHR 2.28 (95% CI 1.39, 3.72), *p* = 0.004) personality disorder (AHR 1.57 (95% CI 1.01, 2.44), *p* = 0.043) or anxiety/depression (AHR 1.57 (95% CI 1.32, 1.86), *p* < 0.0001). Although the point estimates for the size of the effects are different, the confidence intervals overlap and those where the number of cases is smaller (bipolar, personality disorder) have wider confidence intervals. There is a lack of statistical evidence for an association with parental schizophrenia (AHR 1.41 (95% CI 0.96, 2.07), *p* = 0.08), and with somatoform disorder (AHR 1.25 (95% CI 0.74, 2.13), *p* = 0.40). Results for parental history of drug or alcohol misuse (AHR 1.08 (95% CI 0.82, 1.43), *p* = 0.57), do not support any association between substance misuse in parents and ED in children. Hazard ratios for all children are very similar to those for females only and are available on request from the corresponding author

All the models presented satisfied the assumption of proportional hazards.

We found no evidence of interaction between parental mental disorders and any of the potential confounders.

Sensitivity analysis were performed removing all parents with a diagnosis of an ED from the analysis and, separately, including drug and alcohol misuse in the definition of parental mental illness. The analyses were also repeated using a hierarchy of parental mental illness such that each parent would only be counted as having the diagnosis highest in the hierarchy. None of these amendments substantially altered the results.

Analyses were repeated with further adjustment for smoking during pregnancy, birth weight, and being small for gestational age. These adjustments reduced the numbers of participants with data available and the statistical power of our analysis, but did not change the direction or size of the main effects.

Missing Data

There was little missing data, and crude incidence rates and hazard ratios were both very similar for all records compared with records with complete data on all confounding variables. Crude incidence rates are available on request from the corresponding author. Crude hazard ratios are presented in **Tables 3** and **4**.

Discussion

Principle Findings

We have found good evidence in support of our hypothesis that parental mental illness is a risk factor for subsequent diagnosis of ED in their offspring, between the ages of 12 and 24, after controlling for potential confounders. This association was most evident for parental diagnoses of anxiety and depression and bipolar affective disorder and personality disorder. We found no evidence that parental schizophrenia, somatoform disorders and drug and alcohol misuse were associated with increased incidence rates of ED in the offspring. The confidence intervals for the association with parental schizophrenia and somatoform disorder include the possibility that they are associated with up to a doubling of the incidence rate of ED in their offspring, as well as the possibility that there is no association. The absence of a relationship in this dataset may be due to the relatively small numbers of exposed children. Although the absolute risk of ED is fairly small, as it is a relatively uncommon illness, the doubling of risk with a parental history of mental illness is an important contribution to our understanding of the risk factors contributing to ED.

Strengths and Limitations

Our study was large and, to our knowledge, is the only published study to date which uses total population data to study the effects of different mental illnesses, as opposed to mental illness in general, in parents, and all ED in their children,

TABLE 4. Hazard ratios of incidence rates of ED in female offspring by history of parental mental illness

Parental Mental Illness	Stratified Analysis ^a (HR, 95% CI, <i>p</i> Value) (Including Those Without Data on Confounders: <i>N</i> = 83,694)	Stratified Analysis ^a (HR, 95% CI, <i>p</i> Value) (<i>N</i> = 77,323)	Stratified, Adjusted Analysis ^b (HR, 95% CI, <i>p</i> value) (<i>N</i> = 77,323)
Schizophrenia	1.39 (0.96, 2.01), <i>p</i> = 0.09	1.43 (0.97, 2.10), <i>p</i> = 0.07	1.41 (0.96, 2.07), <i>p</i> = 0.08
Bipolar	2.21 (1.35, 3.62), <i>p</i> < 0.002	2.40 (1.47, 3.92), <i>p</i> < 0.0001	2.28 (1.39, 3.72), <i>p</i> = 0.004
Personality disorder	1.46 (0.96, 2.22), <i>p</i> = 0.078	1.50 (0.97, 2.33), <i>p</i> = 0.069	1.57 (1.01, 2.44), <i>p</i> = 0.043
Depression/ Anxiety	1.60 (1.35, 1.89), <i>p</i> < 0.0001	1.53 (1.29, 1.82), <i>p</i> < 0.0001	1.57 (1.32, 1.86), <i>p</i> < 0.0001
Somatoform Disorder	1.17 (0.69, 1.99), <i>p</i> = .56	1.21 (0.72, 2.05), <i>p</i> = 0.48	1.25 (0.74, 2.13), <i>p</i> = 0.40
Alcohol/Drug misuse	1.08 (0.85, 1.38), <i>p</i> = 0.52	1.03 (0.79, 1.36), <i>p</i> = 0.81	1.08 (0.82, 1.43), <i>p</i> = 0.57

^aStratified by year of birth of child, clustered by mother.

^bStratified by year of birth of child, adjusted for maternal age, occupational class, maternal education (three levels), family type, and number of siblings, clustered by mother.

thus avoiding the problem of recall bias. It was population rather than clinic based, reducing the problem of selection bias. The study participants were followed up between the ages of 12–24 years, the age range with the highest incidence of ED. Furthermore, information on when participants presented to mental health services was used to define the outcome, so these results are likely to reflect incidence of ED as seen in clinical practice.

The main limitations of our study relate to the correct and complete identification of ED in children, and mental illnesses in their parents. There are limitations to collecting data from administrative health care registers, as not everyone with an ED presents for treatment. A household survey in the United States found that only 50–63.2% of respondents with AN, BN, and binge eating disorder received treatment at some point in their lives,³⁹ although such numbers might be expected to be higher in Sweden, where there is a comprehensive, government-provided health service. There are no studies on rates of presentation for care for people with eating disorders in Sweden, but register-based studies of eating disorders including both in and outpatient diagnoses^{26,40} are contributing to our understanding of the aetiology of eating disorders as they enable us to study much larger numbers of people with diagnosed eating disorders. The fact that not everyone with an eating disorder presents to services should not in itself affect the relationship between mental illnesses in parents and ED in their children, although it may mean that we underestimate the association between parental mental illness and child eating disorders.

The diagnosis of an ED is based on attendance at a specialist ED clinic, or on clinician coding of an ED. Specialist ED clinics in Sweden exist to treat AN, BN, and EDNOS, and it seems likely that the vast majority of referrals will be for patients with these conditions. Similarly, we would expect that specialist clinicians are able to accurately identify and code for ED in their patients. It would have been interesting to have

been able to analyze the diagnoses of AN and BN separately. However, the numbers of offspring with each of these diagnoses were too small to perform any adequately powered analyses. It is possible that an inability to separate out these diagnoses may have lead to the study not finding an association between drug and alcohol use and ED, as there may be an association with BN and drug and alcohol use but not AN and drug and alcohol use.

Complete identification of all parental mental illnesses is also limited as not all parents will have presented for assessment and treatment. However it is unlikely that rates of presentation for treatment will have varied between parents of children with and without ED, as parental mental illnesses occurred before the offspring's diagnosis. Another limitation with the data on parental mental illness is that although we know that this occurred before the child's ED, we don't know how long the child was living with a parent with a mental illness, or at what age. There may be a dose-response relationship between length of parental mental illness and likelihood of child ED which we cannot investigate.

Finally, the routine reporting of diagnoses to the central registers in Sweden has changed over the years, with diagnoses being coded in adult outpatient psychiatric and ED clinics from 1997 onwards. This means that we may have underestimated the incidence of ED in those who were older at the time of entry into the study. It is also likely to mean that we have underestimated the number of parents with a history of a mental illness, particularly in their earlier years. This should not have affected our results, as this underestimation is likely to be the same for parents of children with and without ED.

Results in Relation to Other Studies and Unanswered Questions

Our results with regard to the association between any parental mental illness (excluding

alcohol and drug misuse) and ED in children are similar in magnitude to the results found by Lindberg⁷ in their record linkage study of AN, in which they used records of admission to psychiatric hospital as their exposure and outcome measures. Our results were also similar with respect to not finding an association between parental drug or alcohol misuse and offspring ED,⁷ and contrast with a study which found a genetic correlation between drug misuse and BN.⁴¹

The temporal relationship between parental mental illness and child ED rules out the possibility that parental diagnoses are consequences of ED in offspring. There are several possible explanations for the association, which could be either genetic or environmental, or most likely a combination of the two.

Our finding that ED are more common in children of parents with many different mental illnesses gives weight to the suggestion that the association may be environmental; however, we found no association between parental drug and alcohol misuse and ED in children, and one might expect parental drug or alcohol misuse to be as stressful and disruptive as parental mental illness for a child. One possible explanation is that there may be common psychological risk factors underlying different mental illnesses and ED, which are not shared by people who are dependent on drugs and alcohol. For example, a parent with depression may hold beliefs such as "people will only love me if I'm perfect", and their offspring may then share these beliefs, which may be a risk factor for developing ED. In contrast, a parent with a substance misuse problem may not hold beliefs which would predispose to the development of ED in their children. Future studies of the relationship between parental mental illness, parenting styles and offspring eating disorders would be interesting to help inform treatment of ED in the context of parental mental illness.

In summary, we found that evidence in support of our hypothesis that parental mental illness, and specifically parental anxiety, depression, bipolar affective disorder, and personality disorders are associated with the subsequent diagnosis of ED in their offspring.

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