Risks for Nonaffective Psychotic Disorder and Bipolar Disorder in Young People With Autism Spectrum Disorder: A Population-Based Study

Jean-Paul Selten, MD, PhD; Michael Lundberg, MPH; Dheeraj Rai, MRCPsych, PhD; Cecilia Magnusson, MD, PhD

IMPORTANCE Whether individuals with autism spectrum disorder (ASD) are at increased risk for nonaffective psychotic disorder (NAPD) or bipolar disorder (BD) is unknown.

OBJECTIVE To test whether the risks for NAPD and BD in individuals with ASD are increased and whether these risks are higher than those of their siblings not diagnosed as having ASD.

DESIGN, SETTING, AND PARTICIPANTS We performed a nested case-control study of all individuals 17 years or younger who ever resided in Stockholm County, Sweden, from January 1, 2001, through December 31, 2011 (Stockholm Youth Cohort). We included cohort members ever diagnosed as having ASD (n = 9062) and their full siblings never diagnosed as having ASD. Each case was matched with 10 control individuals of the same sex born during the same month and year. Using Swedish registers, cases, siblings, and controls were followed up until December 31, 2011. By then, the oldest individuals had reached the age of 27 years.

EXPOSURES Autism spectrum disorder, registered before age 16 or 28 years. We distinguished between ASD with and without intellectual disability (ID).

MAIN OUTCOMES AND MEASURES We calculated odds ratios (ORs) for NAPD and BD adjusted for age, sex, population density of place of birth, personal or parental history of migration, hearing impairment, parental age, parental income, parental educational level, and parental history of psychiatric disorder.

RESULTS The adjusted ORs for NAPD and BD for cases with non-ID ASD registered before age 16 years were 5.6 (95% CI, 3.3-8.5) and 5.8 (95% CI, 3.9-8.7), respectively; the adjusted ORs for cases with ID ASD were 3.5 (95% CI, 2.0-6.0) and 1.8 (95% CI, 0.8-4.1). The adjusted ORs for NAPD and BD in cases with non-ID ASD registered before age 28 years were 12.3 (95% CI, 9.5-15.9) and 8.5 (95% CI, 6.5-11.2), respectively; for cases with ID ASD, these ORs were 6.4 (95% CI, 4.2-9.8) and 2.0 (95% CI, 1.0-3.9), respectively. The ORs for NAPD and BD for the nonautistic full siblings of cases for whom ASD was registered before age 16 years, adjusted for hearing loss, were 1.8 (95% CI, 1.1-2.7) and 1.7 (95% CI, 1.1-2.6), respectively.

CONCLUSIONS AND RELEVANCE A diagnosis of ASD is associated with a substantially increased risk for NAPD and BD. This finding contributes to our understanding of these disorders and has implications for the management of ASD.
Autism spectrum disorders (ASDs) are neurodevelopmental disorders characterized by impairments in social interaction and communication and by restricted and repetitive behaviors and interests. Little is known about the adult outcome of ASDs, especially regarding the risk for developing psychotic disorders. To our knowledge, the largest study of the risk for nonaffective psychotic disorder (NAPD) included only 163 individuals, whereas the largest investigation of the risk for bipolar disorder (BD) included 129 individuals. A review concluded that the evidence of an increased risk is conflicting. Better information about prognosis is particularly topical considering the marked increase in the proportion of children being diagnosed as having ASD in recent years.

Several lines of evidence suggest that individuals with ASD may be at increased risk for NAPD and BD. First, some evidence suggests that the disorders co-occur in families. With a single exception, the larger family studies found that relatives of children with ASD were more likely to have a schizophrenia diagnosis. One of these studies reported that the first-degree relatives were also at an increased risk for BD. Second, some evidence of genetic overlap between these disorders exists. For example, studies of copy number variants in the genome have identified some rare mutations that are associated with ASD and schizophrenia. However, a study of common single-nucleotide polymorphisms found only a low genetic correlation between ASD and schizophrenia and a nonsignificant correlation between ASD and BD (see also Vorstman et al). Third, ASD and psychoses may share certain risk factors, such as high paternal age, obstetric complications, and maternal infections. Finally, the stress resulting from the impairments associated with ASD may increase the risk for NAPD and BD. For example, social exclusion, which is common in ASD, has been hypothesized to be an important risk factor for NAPD.

The interpretation of any putatively increased psychosis risk would be furthered by a comparison of psychosis risks for individuals with ASD and their siblings without ASD because this strategy accounts for shared confounding factors. A higher risk for psychosis among individuals with ASD than for their siblings without ASD would strengthen the case for a true association between ASD and psychosis.

The present investigation is a follow-up of a large cohort of young people from Stockholm County, Sweden. We hypothesized that the risks for NAPD and BD would be higher in individuals with ASD than in individuals without ASD and higher in individuals with ASD than in their nonaffected siblings.

Methods

We designed a case-control study nested within the Stockholm Youth Cohort, a multiregister-based cohort consisting of all individuals 17 years or younger who resided in Stockholm County at any time from January 1, 2001, through December 31, 2011 (N = 735 096). All individuals and their first-degree relatives were linked to a range of health, social, and other administrative registries using unique personal identification numbers assigned to each Swedish citizen at birth or, for immigrants, on arrival in Sweden. We excluded adoptees (n = 458) and individuals with missing information on date of birth or parent identification numbers (n = 39 051). We also excluded 6254 individuals without information on the covariates described below. The study cohort therefore consisted of 689 333 individuals. The study was reviewed and approved by the regional ethical review board of Stockholm. In accordance with the Swedish Personal Data Act of 1998 (http://www.datainspektionen.se/in-english/legislation/the-personal-data-act/) and the decision of the ethical review board, we did not obtain informed consent from the study participants. All study data were anonymized.

Cases

Autism spectrum disorder case status was ascertained using national and regional registers that cover all of the known pathways of ASD diagnosis and care in Stockholm County. Autism spectrum disorder was diagnosed when individuals met criteria from the DSM-IV, International Classification of Diseases, Ninth Revision, or the International Statistical Classification of Diseases, Tenth Revision (ICD-10) for a pervasive developmental disorder. Given the maximum age at entry into the cohort in 2001, the oldest members were born in 1984 and were 27 years old in 2011. The validity of the ASD diagnoses in members of this cohort is good. Because intellectual disability (ID) is a source of heterogeneity in ASD, we grouped individuals with ASD into those with (ID ASD) and without (non-ID ASD) the disability.

Control individuals were selected from the background Stockholm Youth Cohort population. Each ASD case was matched by sex and age (month and year of birth) to 10 controls. To avoid a substantial difference in the likelihood of contacting psychiatric services, controls were required to be residents in the Stockholm area at the time the case was registered with ASD. Thus, 9062 individuals registered with ASD at younger than 16 years were matched with 90 620 controls, and 10 726 individuals registered with ASD at younger than 28 years were matched to 107 260 controls. Last, 11 046 full siblings of cases registered with ASD at younger than 16 years were matched with 110 460 controls. To avoid a substantial difference in the likelihood of contacting psychiatric services, controls were required to be residents in the Stockholm area at the time the case was registered with ASD. Thus, 9062 individuals registered with ASD at younger than 16 years were matched with 90 620 controls, and 10 726 individuals registered with ASD at younger than 28 years were matched to 107 260 controls. Last, 11 046 full siblings of cases registered with ASD at younger than 16 years were matched with 110 460 controls of the same sex and age from families without ASD, and 12 859 siblings of cases registered with ASD at younger than 28 years were matched with 128 590 controls.

Outcomes

The case-control sample was followed up to determine whether cohort members had ever been diagnosed as having NAPD (including schizophrenia), schizophrenia, or BD as recorded in the National Patient Register or the Stockholm County Adult Outpatient Psychiatric Register. The National Patient Register contains discharge diagnoses for all inpatient (since 1973) and outpatient (since 2001) psychiatric treatment in Sweden, including admissions to any hospital, whereas the Stockholm County Adult Outpatient Psychiatric Register records the dates and diagnoses for any contact with outpatient psychiatric services in Stockholm County since 1997. Both registers are known to capture psychotic disorders with a high validity.
We defined a broader category of NAPD to include the ICD-10 categories of schizophrenia (ICD-10 code F20), delusional disorder (ICD-10 code F22), acute and transient psychotic disorder (ICD-10 code F23), schizotypic disorder (ICD-10 code F25), and other nonorganic (ICD-10 code F28) and unspecified (ICD-10 code F29) psychotic disorders. Schizotypal disorder (ICD-10 code F21) and schizophrenia simplex (ICD-10 code F20.5) were not considered NAPDs because they do not feature severe psychotic symptoms. Induced delusional disorder (ICD-10 code F24) was also excluded from the group of NAPDs. Individuals diagnosed as having any ICD-10 code F20.x disorder (except F20.6) were considered patients with schizophrenia. Bipolar disorder was operationalized as a diagnosis of manic episode (ICD-10 code F30) or bipolar affective disorder (ICD-10 code F31). Because NAPD and BD in childhood are sometimes difficult to distinguish from ASD or attention-deficit/hyperactivity disorder, the minimum age at registration with this diagnosis was set at 12 years.

**Statistical Analysis**

We used conditional logistic regression analysis to compare the risks for individuals with ASD with those for controls, conditioned on the matching criteria and potential confounders. We also compared risks of the group of full siblings without ASD and those of the controls.

Because impairments in social interaction are common in NAPD, the validity of an additional NAPD diagnosis in individuals with ASD is a potential concern. The difficulty people with ASD have in understanding the subtleties of social behavior could be mistaken as a sign of psychosis. Conversely, clinicians might assume erroneously that social impairments related to a psychotic disorder have been present since childhood and wrongly make an additional ASD diagnosis. To a lesser extent, these issues apply to an additional diagnosis of BD. We attempted to circumvent this problem by investigating the risk for NAPD or BD in individuals who had been registered with ASD at younger than 16 years (psychosis is rare before age 16 years) and then investigated all individuals diagnosed as having ASD regardless of the age at registration.

Associations could be confounded if individuals with ASD were differentially exposed to certain risk factors for psychosis. For example, parental history of psychosis is a major risk factor for any psychotic disorder. However, a parental history of any mental disorder may increase the risk for a psychotic disorder in a child with ASD. Consequently, we investigated whether cases had a parental history of NAPD, BD, or any other mental disorder (except dementia and delirium) using International Classification of Diseases criteria (eMethods in the Supplement).

Other important risk factors for NAPD include an urban upbringing, a personal or parental history of migration, low intelligence level, drug abuse, childhood trauma, childhood social disadvantage, paternal age, hearing impairment, and obstetric complications. Risk factors for BD include drug abuse, childhood trauma, migration, and paternal age. Although most of these risk factors may act as confounders, some, such as low intelligence level or childhood trauma, may act as mediators or confounders. We adjusted for all these variables except for drug use and childhood trauma, for which we had insufficient information.

Population density of the area of birth in Sweden or, in the case of migrants, the area of first registration in Sweden was categorized in quintiles and used as a proxy for urban upbringing. Individuals were considered migrants if they had been born abroad (first generation) or if at least 1 parent had been born abroad (second generation). Migrants were divided into those from high- and those from low- or medium-developed countries based on the Human Development Index (http://hdr.undp.org/en/content/human-development-index-hdi). People born in Sweden to 2 Swedish-born parents were considered native Swedes.

Socioeconomic measures included the disposable income of the parents adjusted for family size and parental educational level. Hearing impairment was defined as a diagnosis of hearing loss (ICD-10 code H90 or H91). Information on parental ages was retrieved from the Swedish Multigeneration Register. We computed odds ratios (ORs) and 95% CIs, adjusted for parental age, parental psychiatric history, population density of birthplace, personal or parental history of migration, family income, parental educational level, and hearing impairment. In a separate analysis, we also used information from the Medical Birth Register to adjust for prematurity (gestational age <37 weeks) and fetal growth restriction (birth weight for gestational age >2 SD below the mean). This information was not available for 137 009 individuals, most of whom had been born abroad. Last, using the compulsory examination for school departure at ages 15 or 16 years derived from the National School Register, we investigated the impact of cognitive ability by stratifying the non-ID ASD group into a subgroup with low (first, second, and third deciles) and average to high (other deciles) abilities.

**Results**

The parents of individuals with ASD more often had a history of a psychiatric disorder and less favorable socioeconomic characteristics than the parents of controls (Table 1). Remarkably, many mothers of individuals with ASD had a history of a psychiatric disorder, and the age of the fathers was not significantly increased. Individuals with ASD had a less favorable obstetric history, more often had hearing impairment, and were less likely to be born in the most densely populated areas. Fourteen cases with ASD and 6 controls had been registered with a diagnosis of NAPD or BD before age 12 years and were excluded from the analysis of psychosis risk.

Of the individuals registered with ASD at younger than 16 years, 0.6% had NAPD and 0.6% had BD compared with 0.1% each of the matched controls (Table 1). The increased risks for NAPD and BD remained after adjustment for possible confounders (Table 2). Further adjustment for prematurity and fetal growth restriction did not substantially affect our findings (eTable 1 in the Supplement). The most commonly diagnosed subtype of NAPD among individuals with ASD was unspecified nonorganic psychosis (45.6%). For the subtypes of BD...
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among individuals with ASD, the proportion of International Classification of Diseases codes referring to psychotic features was 9.8%. The risk for schizophrenia was also increased (eTable 2 in the Supplement). Information on the age at first registration for NAPD or BD is provided in eTable 3 in the Supplement.

The relationships were even stronger for the larger group of individuals registered with ASD at younger than 28 years (Table 2). Again, further adjustment for prematurity and fetal growth restriction had a minimal effect.

The cognitive ability of individuals with ASD seemed to influence their risk for NAPD because the risk for this disorder was higher among individuals with non-ID ASD than among those with ID ASD (Table 2). Stratification of individuals with non-ID ASD according to the examination grade attained when they left school at 15 or 16 years of age revealed that the risk for NAPD varied according to age at registration. As for individuals registered with ASD at younger than 28 years, the risk for NAPD was higher among those with average to high levels of performance than among those with low levels (Table 3). Too few individuals were registered with ASD at younger than 16 years to analyze this potential association. The influence of cognitive ability on the risk for BD was even more pronounced (Tables 2 and 3).

The full siblings of the cases registered with ASD at younger than 16 years showed greater risks for NAPD (25 siblings; OR, 1.8 [95% CI, 1.1-2.7]) and BD (27 siblings; OR, 1.7 [95% CI, 1.1-2.6]) than controls (142 and 157 siblings, respectively) but much less so than the individuals with ASD (eTable 4 in the Supplement gives the results for full siblings of cases registered with ASD at younger than 28 years). The pattern of results was similar when individuals were divided into those with and without ID.

Discussion

We found that individuals with ASD were at higher risk for developing NAPD, schizophrenia, and BD than age- and sex-matched individuals without ASD from the general population. This risk was greater among individuals with non-ID ASD. The risk for these disorders was also significantly higher among individuals with ASD than among their siblings without ASD.

This study had a number of strengths, such as the high quality of the Swedish registers, the large sample, and the ability to adjust for a range of confounders and to examine the risks for siblings of individuals with ASD. The fact that the cohort has not gone through the whole period of risk for developing NAPD or BD (the period of maximum risk is 18-30 years) is a limitation and precludes estimation of a lifetime morbidity risk. The association between ASD and the risk for NAPD and BD might have been overestimated if individuals with an unrecognized ASD come to the attention of services only after they have developed psychosis. Another limitation is the inability to estimate the influence of drug abuse and childhood trauma. Although no good evidence of a higher rate of drug abuse in ASD exists,29 children with ASD are often bullied, which may increase their risk for psychosis.33 Another limitation is that our insight into the structure underlying major mental disorders is limited and that our results are based on categorical diagnoses with a disputed validity. However, studies using dimensional measures of autism and psychosis reported similar findings.12,34

The correctness of a diagnosis of NAPD in individuals with ASD could be questioned, and confirmation by additional studies using a diagnostic interview is warranted. This issue is even more relevant for individuals with ID ASD because the possibility of misdiagnosing atypical symptoms is high,25,35 and clinicians may attribute symptoms of comorbid mental illness to the individual’s ID, a phenomenon described as diagnostic overshadowing.36 However, the observation that individuals

Table 1. Characteristics of Members of the Stockholm Youth Cohort Registered With ASD Before Age 16 Years and Their Matched General Population Controls

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>ASD Cases (n = 9062)</th>
<th>Controls (n = 90 620)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male sex</td>
<td>73.0</td>
<td>73.0</td>
</tr>
<tr>
<td>Age at end of follow-up, mean (SD), y</td>
<td>15.3 (6.2)</td>
<td>15.3 (6.2)</td>
</tr>
<tr>
<td>Parental age at birth, mean (SD), y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal</td>
<td>29.9 (5.4)</td>
<td>29.9 (5.2)</td>
</tr>
<tr>
<td>Paternal</td>
<td>33.1 (6.7)</td>
<td>32.9 (6.3)</td>
</tr>
<tr>
<td>In highest quintile of disposable family income at birth</td>
<td>17.0</td>
<td>21.4</td>
</tr>
<tr>
<td>Elementary maternal educational level</td>
<td>17.5</td>
<td>15.7</td>
</tr>
<tr>
<td>Index person and both parents born in Sweden</td>
<td>64.2</td>
<td>64.2</td>
</tr>
<tr>
<td>Parental history of NAPD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal</td>
<td>1.4</td>
<td>0.8</td>
</tr>
<tr>
<td>Paternal</td>
<td>1.4</td>
<td>0.8</td>
</tr>
<tr>
<td>Parental history of BD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal</td>
<td>2.0</td>
<td>1.0</td>
</tr>
<tr>
<td>Paternal</td>
<td>1.0</td>
<td>0.6</td>
</tr>
<tr>
<td>Parental history of other psychiatric disorder</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal</td>
<td>45.0</td>
<td>31.1</td>
</tr>
<tr>
<td>Paternal</td>
<td>26.0</td>
<td>19.0</td>
</tr>
<tr>
<td>Fetal growth restriction</td>
<td>4.2</td>
<td>2.3</td>
</tr>
<tr>
<td>Premature birth</td>
<td>9.0</td>
<td>5.8</td>
</tr>
<tr>
<td>Place of birth in highest quintile of population density</td>
<td>16.9</td>
<td>19.8</td>
</tr>
<tr>
<td>Diagnosis, No. (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hearing impairment</td>
<td>374 (4.1)</td>
<td>1593 (1.8)</td>
</tr>
<tr>
<td>NAPD (including schizophrenia)</td>
<td>57 (0.6)</td>
<td>110 (0.1)</td>
</tr>
<tr>
<td>Schizophrenia</td>
<td>10 (0.1)</td>
<td>17 (0.0)</td>
</tr>
<tr>
<td>BD</td>
<td>51 (0.6)</td>
<td>96 (0.1)</td>
</tr>
</tbody>
</table>

Abbreviations: ASD, autism spectrum disorder; BD, bipolar disorder; NAPD, nonaffective psychotic disorder.

* Unless otherwise indicated, data are expressed as percentage of participants.

† Indicates pervasive developmental disorder according to criteria from the DSM-IV, International Classification of Diseases, Ninth Revision, or the International Statistical Classification of Diseases, 10th Revision (ICD-10).

‡ Includes schizophrenia, schizoaffective disorder, delusional disorder, or other nonaffective or nonorganic psychotic disorder (ICD-10 criteria), with age at registration for any of these diagnoses at least 12 years.

§ Indicates bipolar affective disorder or manic episode (ICD-10 criteria), with age at registration for any of these diagnoses at least 12 years.

∥ Indicates gestational age of less than 37 weeks.

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with ASD were also at an increased risk for BD, a disorder unlikely to be confused with ASD, argues strongly against misdiagnosis as the sole explanation for our findings.

The observation of greater risks for individuals without ID and for those who performed well at school was intriguing. As described above, ruling out diagnostic bias is difficult. However, the greater risk for individuals with ASD with greater cognitive resources might be attributable to a greater awareness of their impairments. If true, this interpretation would run contrary to the established association between low IQ and the risk for schizophrenia. However, because the genetic correlation between IQ and schizophrenia is low, the association may reflect environmental mediation of a genetic effect. Clearly, this topic needs further research.

The results of our study confirm and extend those reported in studies from Denmark. Mouridsen et al. 190 reported that, after a mean observation time of 36.9 years, 31 of 89 individuals diagnosed as having atypical autism during childhood had been diagnosed as having a schizophrenia spectrum disorder. In a study with a mean follow-up of 32.5 years, 40 of 118 individuals with autistic disorder had been diagnosed as having a schizophrenia spectrum disorder. Both studies 190, 40 reported that the risk for affective disorder not further specified was 2 to 3 times higher than that for controls. The results of our study are also consistent with the findings of two retrospective investigations that applied ASD diagnostic interviews to patients with schizophrenia and their parents. As for BD, a systematic evaluation found that 14 of 66

### Table 2. Adjusted ORs for NAPD and BD Among Members of the Stockholm Youth Cohort With ASD

<table>
<thead>
<tr>
<th>ASD Status</th>
<th>NAPD**</th>
<th>BD**</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of Cases</td>
<td>(95% CI)</td>
</tr>
<tr>
<td>Non-ID ASD</td>
<td>37</td>
<td>6.2 (4.1-9.3)</td>
</tr>
<tr>
<td>ID ASD</td>
<td>20</td>
<td>4.1 (2.5-7.0)</td>
</tr>
<tr>
<td>All ASD</td>
<td>57</td>
<td>5.3 (1.8-7.3)</td>
</tr>
</tbody>
</table>

**Includes pervasive developmental disorder according to criteria from the International Classification of Diseases, Ninth Revision, or International Statistical Classification of Diseases, 10th Revision (ICD-10).

### Table 3. Adjusted ORs for NAPD and BD Among Members of the Stockholm Youth Cohort Registered With Non-ID ASD by Age at Registration of ASD and School Performance

<table>
<thead>
<tr>
<th>School Performance Level</th>
<th>NAPD**</th>
<th>BD**</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of Cases</td>
<td>(95% CI)</td>
</tr>
</tbody>
</table>
| Registration of ASD Before Age 16 y
| Low†                    | 28 | 5.2 (2.8-9.6) | 18 | 2.5 (1.2-4.8) |
| Average to high‡        | 5 | 2.5 (0.8-8.0) | 13 | 10.5 (4.2-26.4) |
| Registration of ASD Before Age 28 y
| Low†                    | 94 | 11.1 (7.3-16.9) | 59 | 4.3 (2.7-6.6) |
| Average to high‡        | 52 | 18.3 (10.2-33.0) | 46 | 12.6 (7.4-21.4) |

Abbreviations: ASD, autism spectrum disorder; BD, bipolar disorder; ID, intellectual disability; NAPD, nonaffective psychotic disorder; OR, odds ratio.

† Indicates at 15 or 16 years of age.
‡ Indicates pervasive developmental disorder according to criteria from the International Statistical Classification of Diseases, 10th Revision (ICD-10) criteria, with age at registration for any of these diagnoses at least 12 years.
* Indicates at 15 or 16 years of age.
** Indicates at 15 or 16 years of age.
children and adolescents with ASD (21.2%) met criteria for mania and that 14 of 128 children with mania (10.9%) met criteria for ASD. Stahlberg et al.2 reported that 9 of 129 adult cases of ASD (7.0%) had BD with psychotic features.

How do these findings relate to current knowledge about the etiology of BD or NAPD? The literature makes little mention of an association between ASD and BD. For example, the authoritative textbook, Manic-Depressive Illness, has no entries on ASD or pervasive developmental disorder.23 That ASD and NAPD might be associated is not surprising. Kraepelin44 observed that many children who went on to develop dementia praecox “exhibited a quiet, shy, retiring disposition, made no friendships, lived only for themselves.” In adulthood, these individuals were often diagnosed as having schizoid or schizotypal personalities, which are known risk factors for later schizophrenia. At present, individuals with similar symptoms probably see a physician earlier and might be diagnosed as having ASD. Indeed, the DSM-5 manual states that differentiating individuals with schizoid or schizotypal disorder from those with milder forms of ASD may be difficult.45 Our finding of a higher risk for psychosis among individuals registered with ASD after age 16 years supports this idea because the latter individuals are probably less impaired than those registered at an earlier age. This finding is not intended to mean that individuals with ASD who develop a psychotic disorder are in reality individuals with a schizoid or schizotypal personality disorder but to underline that the diagnostic boundaries between ASD and these personality disorders, if they exist at all, are uncertain.46 Further investigation of these boundaries and of the possibility of distinguishing individuals with ASD who go on to develop NAPD or BD from those who do not is needed.47 One possible explanation for the association between ASD and NAPD or BD is a common underlying genetic vulnerability.9,14 Another explanation implies that ASD increases the exposure or sensitivity to psychosis risk factors.14,20,23 The sibling analyses presented herein suggest that shared familial factors do not explain the association.

Conclusions

Our findings suggest that individuals with ASD have a substantially increased risk for affective and nonaffective psychoses. The results have implications for the provision of mental health care for individuals with ASD, who often fall between services, particularly as they transition into adulthood.

ARTICLE INFORMATION
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Conflict of Interest Disclosures: None reported.

REFERENCES
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Original Investigation Research


