


Risk of being convicted of theft and other crimes in anorexia nervosa and bulimia nervosa: A prospective cohort study in a Swedish female population

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Abstract

Objective: We examined epidemiological associations between anorexia nervosa (AN) and bulimia nervosa (BN) and risks of committing theft and other crimes in a nationwide female population.

Method: Females born in Sweden during 1979–1998 ($N = 957,106$) were followed from age 15 for up to 20 years using information on clinically diagnosed AN and BN (exposures), convictions of theft and other crimes (outcomes), psychiatric comorbidities, and familial relatedness from Swedish national registers. We estimated hazard ratios (HRs) of criminality in exposed versus unexposed females using Cox proportional hazards regressions and explored how comorbidities and unmeasured familial factors explained the associations.

Results: The cumulative incidence of convictions of theft (primarily petty theft) and other crimes was higher in exposed females (AN: 11.60% theft, 7.39% other convictions; BN: 17.97% theft, 13.17% other convictions) than in unexposed females (~5% theft, ~6% other convictions). The significantly increased risk of being convicted of theft in exposed females (AN: HR = 2.51, 95% confidence interval = [2.29, 2.74], BN: 4.31 [3.68, 5.05]) was partially explained by comorbidities; unmeasured familial factors partially explained the association with convictions of theft in BN but not in AN. Females with BN had a doubled risk of convictions of other crimes, which was partially explained by comorbidities.

Discussion: Individuals with eating disorders had increased risk for convictions of theft and potentially other crimes. Results underscore the importance of regular forensic screening and encourage research on mechanisms underlying the relation between crime and eating disorder psychopathology and efforts to determine how best to address such relation in treatment.

KEYWORDS

anorexia nervosa, bulimia nervosa, cohort study, crime, eating disorders, epidemiology, register-based, stealing, theft behavior

1 | INTRODUCTION

Eating disorders, including anorexia nervosa (AN) and bulimia nervosa (BN), are psychiatric disorders that have broad impact on health and quality of life in afflicted individuals (Fichter & Quadflieg, 2016; Smink, van Hoeken, & Hoek, 2013; Whiteford et al., 2013). Criminality, among other adversities (Herzog, Nussbaum, & Marmor, 1996; Smink et al., 2013; Yao et al., 2016), has been associated with eating disorders (Coker, Smith, Westphal, Zonana, & McKee, 2014). As it might add to the psychosocial burden on individuals who are already suffering from the direct effects of eating disorders (Grant & Kim, 2002, 2005), it is important to verify the association between eating disorders and criminal behaviors and to understand mechanisms of the associations. However, research on criminality in eating disorders is quite limited. The majority of previous reports came from clinical observations focusing on theft behavior in eating disorders (Baum & Goldner, 1995; Crisp, Hsu, & Harding, 1980; McElroy, Hudson, Pope, & Keck, 1991; Mitchell, Fletcher, Gibeau, Pyle, & Eckert, 1992). Rates of committing theft were typically greater in individuals with BN and the binge-purge subtype of AN than in individuals with the restrictive subtype of AN (McElroy, Pope, Hudson, Keck, & White, 1991; Vandereycken & van Houdenhove, 1996), and have been related to increased levels of impulsivity, comorbidities, and severity of eating disorders (Herzog et al., 1996; Krahn, Nairn, Gosnell, & Drownowski, 1991). Only one study was population-based (Coker et al., 2014), but relied on self-ratings from a US adolescent sample, and reported increased risks of committing theft and other crimes in individuals with binge eating behaviors compared with individuals without (Coker et al., 2014).

Psychiatric morbidity such as personality disorders and attention-deficit hyperactivity disorder (ADHD)—both associated with increased risk of crime (Coker et al., 2014; Vinkers, de Beurs, Barendregt, Rinne, & Hoek, 2011)—co-occur with eating disorders and could account for any observed associations between eating disorders and crime (Hudson, Hiripi, Pope, & Kessler, 2007). Twin and family studies have established underlying genetic and familial environmental liabilities for both eating disorders and criminality (Rhee & Waldman, 2002; Yilmaz, Hardaway, & Bulik, 2015), such liabilities might overlap and confound the associations. Alternative explanations to the associations between eating disorders and criminality have not been previously explored.

We conducted a prospective cohort study using registry data from a nationally representative female cohort in Sweden to thoroughly examine the associations between clinically diagnosed AN and BN and convictions of theft and other crimes. We considerably expand the literature not only by relying on population-based registers for identification of both exposures (clinically diagnosed AN and BN) and outcomes (convictions of theft and other crimes), but also by carefully attending potential confounders that have not been considered in previous research. Interlinked Swedish population-based registers provided diagnostic information on eating disorders and comorbid psychiatric disorders and criminal conviction records, and the large population size allowed us to account for their potential impact on observed associations. In addition to a standard cohort design (i.e., population-level

comparison where we compared the risks of outcomes in differentially exposed unrelated individuals in the study population), we employed a sibling-comparison design in which we compared the risk of crime in differentially exposed full-sisters (i.e., sisters born to the same biological parents) to adjust for unmeasured familial factors; defined as factors shared by full-sisters such as some genetic and familial environmental factors (D'Onofrio, Lahey, Turkheimer, & Lichtenstein, 2013).

2 | METHOD

This study was approved by the Regional Ethics Review Board in Stockholm, Sweden. The requirement for informed consent was waived because the study was based on administrative population-based registers. An independent government agency (Statistics Sweden) merged and de-identified register data and delivered them for research purposes (Ludvigsson et al., 2016).

2.1 | Data sources

With the unique individual identification number in registry we linked several national registers. The Swedish Total Population Register provides information on birth year and month, death date, and migration type and date (Ludvigsson et al., 2016). The National Patient Register (NPR) contains inpatient psychiatric diagnoses since 1973 and outpatient psychiatric diagnoses since 2001; diagnoses in the NPR are based on the Swedish versions of the International Classification of Diseases, Eighth Revision (ICD-8, 1973–1986), ICD-9 (1987–1996), and ICD-10 (since 1997) (Ludvigsson et al., 2011). Two quality registers for eating disorders, that is, Swedish National Quality Register for Eating Disorder Treatment (Riksät, since 1999) and quality assurance system for eating disorders (Stepwise, since 2005), provide eating disorder diagnoses from specialized treatment centers across Sweden (Birgegard, Bjorck, & Clinton, 2010; Emilsson, Lindahl, Koster, Lambe, & Ludvigsson, 2015), with increased coverage over time (Javaras et al., 2015). Quality register diagnoses are based on the Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revision (DSM-IV-TR) (American Psychiatric Association, 2000). The Criminal Conviction Register (held by the National Council for Crime Prevention) provides criminal conviction records from Swedish lower courts since 1973, regardless of the medico-legal disposition of the convicted offender (Frisell, Pawitan, Langstrom, & Lichtenstein, 2012). Since the age of criminal responsibility in Sweden is 15 years (Frisell, Pawitan, et al., 2012), law-breaking behaviors before age 15 years are not registered. The Multi-Generation Register (Statistics Sweden) links biological parents to individuals who were born after 1932 and lived in Sweden any time after 1961. Information from these registers was updated through December 31, 2013.

2.2 | Study population

The study population included females born in Sweden between January 1, 1979 and December 31, 1998. Adoptees, those whose biological parents were not identifiable in the Multi-Generation Register, and those

who died or emigrated before age 15 years were excluded, yielding a total study population of 957,106 females. They were followed from age 15 years to the time when they left the cohort, that is, when the participant (a) experienced the outcome, (b) emigrated, (c) died, or (d) reached December 31, 2013, whichever came first. We further linked females with the same biological parents using parents' personal identification numbers in the Multi-Generation Register, to identify full-sisters in the study population.

2.3 | Exposures: AN and BN

The two exposures (not mutually exclusive) were AN and BN diagnosed before or during follow-up. The exposure AN included clinically diagnosed AN or atypical AN, identified with ICD-9 code 307B or ICD-10 codes F50.0 or F50.1 or meeting DSM-IV-TR criteria for AN or Eating Disorder Not Otherwise Specified (EDNOS) examples 1 and 2. The exposure BN included clinically diagnosed BN or atypical BN, identified with ICD-10 codes F50.2 or F50.3 or meeting DSM-IV-TR criteria for BN or EDNOS example 3. We obtained diagnoses from the NPR and the quality registers for eating disorders. The period for diagnosing BN was shorter than that for AN, as BN was not an independent eating disorder category in the Swedish version of ICD-9 ("Klassifikation av Sjukdomar 1987," 1987) but was identifiable in ICD-10 (since 1997), potentially leading to more under-detection of BN cases than of AN cases.

The exposures are time-varying. Females whose AN or BN was first diagnosed before or at age 15 years were treated as exposed during the entire follow-up. Females whose AN or BN was first diagnosed after age 15 years but before leaving the cohort were treated as unexposed from age 15 years to the date of the diagnosis and as exposed afterwards.

2.4 | Outcomes: Convictions of theft and other crimes

We defined the primary outcome as convictions of theft, identified in the Criminal Conviction Register and defined it as the first conviction according to the Swedish Penal Code Chapter 8 Sections 1, 2, 4, 7–11, and 13 (i.e., theft, petty theft, gross theft, vehicle theft, unlawful dispossession, self-repossession, unlawful diversion of energy, unlawful takes from a forest or field if not considered as trespassing, and theft committed against a person living with or closely related to the convicted person). We also explored being convicted of "other crimes"; defined as the first nontheft conviction. The outcome date was defined as the date of committing the crime, if registered; otherwise, we used the date of conviction as the outcome date.

2.5 | Comorbidities

We included other eating disorders, personality disorders, and ADHD diagnosed at any time as covariates, as they have been associated with both eating disorders and criminality in a way that we believe could confound the associations of interest (Bulik et al., 2010; Coker et al., 2014; Hudson et al., 2007; Vinkers et al., 2011). Other eating disorders were defined as having any eating disorder diagnosis other than the

exposure (identified by ICD-9 codes 307B or 307 F or ICD-10 codes F50.0, F50.1, F50.2, F50.3, or F50.9 or meeting DSM-IV-TR criteria for an eating disorder, except the exposure). Personality disorders were identified with ICD-9 code 301 or ICD-10 codes F60–F61. ADHD was defined as ICD-9 code 314 or ICD-10 code F90 or being prescribed with specific ADHD medication(s) as defined in previous studies (Chen et al., 2017).

2.6 | Statistical analysis

To illustrate the unadjusted associations between the exposures and outcomes, we plotted the estimated cumulative incidence of being convicted of theft and other crimes in exposed and unexposed groups using the Kaplan–Meier method (software: R 3.2.2). Note that given the time-varying feature of the exposures, the estimated cumulative incidence curve could be interpreted as a proportion of individuals with outcome event among individuals who did not change their exposure status during follow-up, if the association between current exposure status and outcomes were independent of previous exposure status (Lichtenstein et al., 2012). We also estimated the overall incidence rates in exposed and unexposed females and unadjusted incidence rate ratios during follow-up.

Next, we performed population-level comparisons to estimate the relative risks of being convicted of theft and other crimes in females exposed to AN and BN compared to *unexposed, unrelated females* in the study population. Cox proportional hazards regressions were applied (Model 1) to estimate hazard ratio (HR) as a measure of relative risk. To account for the potential effect of comorbidities on the associations, we further adjusted for other eating disorders, personality disorders, and ADHD (Model 2). To explore the effect of each of the comorbidities, we adjusted for them separately in sensitivity analyses. To account for the potential effect of unmeasured familial factors, we conducted sibling-comparisons, where stratified Cox proportional hazards regressions were applied to estimate HRs of being convicted of theft and other crimes in females exposed to AN and BN compared to *their unexposed full-sisters* in the study population (Model 3). By comparing the risks of outcomes in differentially exposed full-sisters, sibling-comparisons automatically controlled for unmeasured familial factors shared by full-sisters in a family. Contrasting estimates from population-level comparisons versus sibling-comparisons could inform whether unmeasured familial factors influenced tested associations. Simplistically, if unmeasured familial factors mainly accounted for the associations, we would expect decreased effect size of association estimates in sibling-comparisons compared with population-level comparisons. Conversely, if the associations were independent of unmeasured familial factors, we would expect similar estimates in both population-level and sibling-comparisons. Note that only females with differently exposed full-sisters were included in Model 3.

All the Cox models used attained age as underlying time scale and were adjusted for birth year as a categorical variable. We addressed nonindependency of data using a cluster-robust estimator of standard error and visually examined Schoenfeld residuals to verify the validity

TABLE 1 Model-related description of the study population

	Exposed to AN	Unexposed to AN	Exposed to BN	Unexposed to BN
<i>Theft as outcome</i>				
Population in each group, N ^a	11,114	954,581	5,197	957,007
Outcome events, N	496	39,940	159	40,278
Follow-up time, in person-year	64,629.27	8,767,634.87	26,278.95	8,805,985.19
Incidence rate, cases per 1000 person-years (95% CI)	7.67 (7.03, 8.38)	4.56 (4.51, 4.60)	6.05 (5.18, 7.07)	4.57 (4.53, 4.62)
Incidence rate ratio (95% CI)	1.59 (1.46, 1.74)	Ref	1.40 (1.20, 1.64)	Ref
<i>Other crimes as outcome</i>				
Population in each group, N ^a	11,245	954,581	5,394	957,007
Outcome events, N	316	41,376	168	41,524
Follow-up time, in person-year	66,992.91	8,810,309.54	27,607.49	8,849,694.97
Incidence rate, cases per 1000 person-years (95% CI)	4.72 (4.22, 5.27)	4.70 (4.65, 4.74)	6.09 (5.23, 7.08)	4.69 (4.65, 4.74)
Incidence rate ratio (95% CI)	0.95 (0.85, 1.06)	Ref	1.33 (1.14, 1.55)	Ref

Note. AN = anorexia nervosa; BN = bulimia nervosa; N = number of observations; 95% CI = 95% confidence interval; Ref = reference group for calculating incidence rate ratio.

^aParticipants could first be unexposed and then became exposed; therefore, the sum of exposed and unexposed populations was greater than the total population.

of the proportional hazards assumption. We performed data management in SAS 9.4 and analyses in Stata 13.0.

3 | RESULTS

3.1 | Descriptive statistics

The 957,106 females in the study population were from 736,538 families. We identified 410,026 biological full-sisters from 189,458 families. Descriptive statistics are presented in Table 1. Convictions of both theft and other crimes were more prevalent in females exposed to eating disorders than in unexposed females (except other crimes in AN) and more prevalent in females exposed to BN than in females exposed to AN (Figure 1). After up-to-20-year follow-up since age 15 years, the estimated cumulative incidence for conviction of theft (i.e., the estimated probability of being convicted of theft before age 35) was 11.60% (95% confidence interval [95% CI], 10.53%–12.77%) in females exposed to AN and 17.97% (14.19%–22.62%) in females exposed to BN, compared to 5.04% (4.98%–5.09%) and 5.07% (5.01%–5.12%) in females unexposed to AN and BN, respectively. The distribution of subtypes of theft did not differ significantly between exposed and unexposed females for both AN and BN, with “petty theft” being the most common subtype (Supporting Information Table S1). The corresponding numbers for being convicted of other crimes were 7.39% (6.47%–8.43%) in females exposed to AN and 13.17% (10.79%–16.04%) in females exposed to BN, compared to 6.24% (6.16%–6.32%) and 6.23% (6.16%–6.31%) in females unexposed to AN and BN, respectively.

3.2 | Hazard ratios of being convicted of theft and other crimes in eating disorders

HRs of being convicted of theft in eating disorders are presented in Table 2. Population-level comparisons (Model 1) revealed that the risk of being convicted of theft in females exposed to AN was more than twice the risk of unexposed females (HR [95% CI]: 2.51 [2.29, 2.74]). The increased risk of being convicted of theft in AN was partially explained by other eating disorders, personality disorders, and ADHD (1.84 [1.67, 2.02], Model 2), but remained similar in sibling-comparison (3.31 [2.57, 4.28], Model 3), suggesting unmeasured familial factors were not likely to explain the association. We observed a stronger association with convictions of theft in BN than in AN; the risk of being convicted of theft in females exposed to BN was more than four times the risk in unexposed females (4.31 [3.68, 5.05], Model 1). HRs of being convicted of theft in BN decreased when adjusting for psychiatric comorbidities (2.62 [2.23, 3.09], Model 2) and in sibling-comparison (2.78 [1.69, 4.60], Model 3), suggesting that comorbidities and unmeasured familial factors partially explained the association between BN and the convictions of theft.

Table 3 presents the HRs of being convicted of other crimes in eating disorders. Population-level comparisons (Model 1) revealed a weak association between AN and being convicted of other crimes (1.16 [1.04, 1.29]), which disappeared when adjusting for psychiatric comorbidities (Model 2) or unmeasured familial factors (Model 3). A stronger association with convictions of other crimes was found in BN (2.15 [1.85, 2.51], Model 1), which attenuated but remained statistically significant after adjusting for psychiatric comorbidities (1.47 [1.26, 1.73], Model 2) and remained similar when adjusting for unmeasured familial

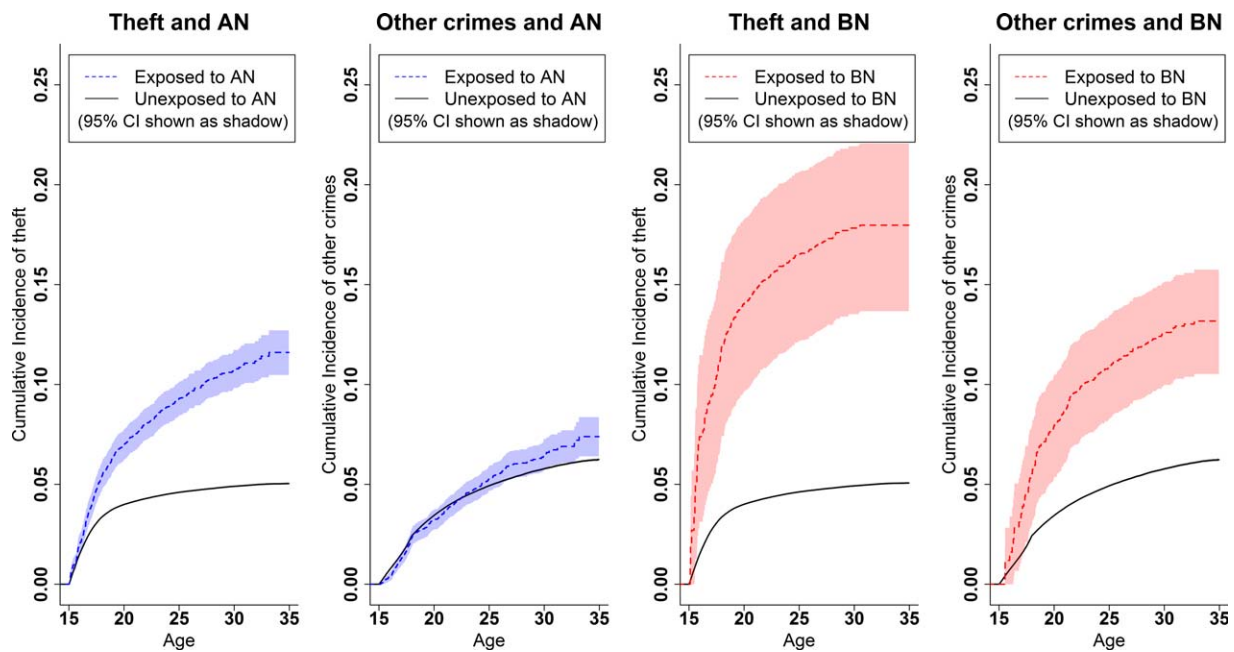


FIGURE 1 Cumulative incidence of being convicted of theft and other crimes in females exposed and unexposed to AN and BN. *Note.* Both theft and other crimes occurred more frequently in females exposed to eating disorders than in unexposed females (except other crimes in AN), and more frequently in females exposed to BN than in females exposed to AN. After up-to-20-year follow-up since age 15 years, the estimated cumulative incidence of being convicted of theft (i.e., the estimated probability of being convicted of theft before age 35) was 11.60% in females exposed to AN and 17.97% in females exposed to BN, compared to 5.04% and 5.07% in females unexposed to AN and BN, respectively. The corresponding numbers for other crimes were 7.39% in females exposed to AN and 13.17% in females exposed to BN, compared to 6.24% and 6.23% in females unexposed to AN and BN, respectively. [Color figure can be viewed at wileyonlinelibrary.com]

factors in sibling-comparison (1.91 [1.23, 2.95], Model 3). The effect of each comorbid psychiatric disorder on the examined associations is presented in Supporting Information (Supporting Information Table S2).

4 | DISCUSSION

In this large cohort study, we thoroughly explored the associations between AN and BN and the risk of being convicted of theft and other crimes in a Swedish female population, while attending to the potential influences of comorbidities and unmeasured familial factors (i.e., factors shared by full-sisters including genetic factors and various shared environmental factors such as familial lifestyle factors). We found increased

risk of being convicted of theft in females exposed to AN or BN compared to unexposed females. BN was also associated with increased risk of being convicted of other types of crime. Both absolute and relative risks were higher in BN than in AN. The increased risk of criminality in eating disorders is worthy of clinical attention as convictions could increase stress and anxiety, interrupt treatment, and adversely influence the course of treatment and recovery (Goldschmidt et al., 2014).

4.1 | Theft

The large size of the cohort provided sufficient statistical power to control for several important psychiatric comorbidities: other eating

TABLE 2 The relative risks of being convicted of theft in eating disorders in population-level and sibling-comparisons

Theft	AN			BN		
	HR (95% CI)	<i>p</i> -value	No. informative individuals (families)	HR (95% CI)	<i>p</i> -value	No. informative individuals (families)
Model 1	2.51 (2.29, 2.74)	<.001	957,105 (736,537)	4.31 (3.68, 5.05)	<.001	957,106 (736,538)
Model 2	1.84 (1.67, 2.02)	<.001	957,105 (736,537)	2.62 (2.23, 3.09)	<.001	957,106 (736,538)
Model 3	3.31 (2.57, 4.28)	<.001	975 (419) ^a	2.78 (1.69, 4.60)	<.001	439 (191) ^a

Note. AN = anorexia nervosa; BN = bulimia nervosa; HR = hazard ratio; 95% CI = 95% confidence interval. Model 1: Population-level comparison; Cox proportional hazards regression was applied, adjusted for birth year. Model 2: Population-level comparison; based on Model 1, Model 2 additionally adjusted for other eating disorders, personality disorders, and ADHD. Model 3: Sibling-comparison; stratified Cox proportional hazards regression was applied, adjusted for birth year. Compared with Model 1, Model 3 additionally adjusted for unmeasured familial factors shared by full-sisters. Estimates with *p*-value < .05 are presented as bold characters.

^aThe presented numbers are the numbers of individuals (numbers of families in parenthesis) that contributed directly to the estimation of associations, other individuals in the population might contribute indirectly to the estimation.

TABLE 3 The relative risks of being convicted of other crimes in eating disorders in population-level and sibling-comparisons

Other crimes	AN			BN		
	HR (95% CI)	p-value	No. informative individuals (families)	HR (95% CI)	p-value	No. informative individuals (families)
Model 1	1.16 (1.04, 1.29)	.01	957,105 (736,538)	2.15 (1.85, 2.51)	<.001	957,105 (736,538)
Model 2	0.93 (0.83, 1.04)	.20	957,105 (736,538)	1.47 (1.26, 1.73)	<.001	957,105 (736,538)
Model 3	1.14 (0.86, 1.52)	.36	639 (274) ^a	1.91 (1.23, 2.95)	.004	379 (166) ^a

Note. AN = anorexia nervosa; BN = bulimia nervosa; HR = hazard ratio; 95% CI = 95% confidence interval. Model 1: Population-level comparison; Cox proportional hazards regression was applied, adjusted for birth year. Model 2: Population-level comparison; based on Model 1, Model 2 additionally adjusted for other eating disorders, personality disorders, and ADHD. Model 3: Sibling-comparison; stratified Cox proportional hazards regression was applied, adjusted for birth year. Compared with Model 1, Model 3 additionally adjusted for unmeasured familial factors shared by full-sisters. Estimates with *p*-value < .05 are presented as bold characters.

^aThe presented numbers are the numbers of individuals (numbers of families in parenthesis) that contributed directly to the estimation of associations, other individuals in the population might contribute indirectly to the estimation.

disorders, personality disorders, and ADHD (Hudson et al., 2007; Vinkers et al., 2011). Adjusting for the comorbidities decreased the associations with being convicted of theft in AN and BN, suggesting that the associations were partially explained by the comorbidities. Comparing the results from population-level comparisons and sibling-comparisons informed whether unmeasured familial factors explained the associations. The association between AN and being convicted of theft remained similar in population-level comparison and in sibling-comparison, suggesting that the association may reflect direct effects between AN and being convicted of theft rather than confounding effects of unmeasured familial factors shared by full-sisters. It is nevertheless important to note that the association between AN and being convicted of theft could still be confounded by factors that were not shared by full-sisters, which were not captured or specified by our measurements and study design. Future studies with different designs could further explore specific factors that explain the association. In contrast, the association between BN and the risk of being convicted of theft was greater in population-level comparison than in sibling-comparison, suggesting that the increased risk of being convicted of theft in BN could be partially explained by unmeasured familial factors, such as genetic and shared familial environmental factors. We were unable to answer more detailed questions about the nature of the unmeasured familial factors for BN and committing theft, but suggest that impulsivity might be one important shared factor to consider in future research, as it is a heritable trait that has been associated with both BN and committing theft (Bezdjian, Baker, & Tuvblad, 2011; Grant & Kim, 2002; Wildes & Marcus, 2013). Interestingly, AN and BN differed in the extent to which unmeasured familial factors explained their associations with theft, which may reflect differences in their etiologies.

4.2 | Other crimes

To our knowledge, only one prior study has explored associations between eating disorders and the risk of committing nontheft crimes and reported increased risk of committing other crimes in individuals with any binge eating behaviors based on self-report measures (Coker

et al., 2014). Similarly, we observed increased risk of being convicted of other crimes in females with clinically diagnosed BN and further found that the association was partially explained by psychiatric comorbidities. However, the overall estimate of risk of being convicted of “other crimes,” a broadly defined group, does not necessarily reflect how each type of crime is associated with eating disorders. More detailed investigations are needed to elucidate the precise nature of the associations between eating disorders and the risk of committing various types of nontheft crimes.

4.3 | Clinical implications

The clinical implications of the results are three-fold. First, the associations should inform clinicians of the increased risk of criminality in individuals with eating disorders. Related legal issues may introduce stress that hampers recovery and increases the risk of relapse in individuals with eating disorders (Goldschmidt et al., 2014; Grilo et al., 2012). Second, criminal behavior might suggest the presence of multi-impulsive or emotionally dysregulated forms of eating disorders (Wildes & Marcus, 2013). Individuals with this form of eating disorder may have other comorbid impulsive behaviors and poorer treatment response (Wildes & Marcus, 2013). Finally, the results may suggest a more direct effect of eating disorders (especially AN) on the risk of being convicted of theft, although this study was unable to explore specific mechanisms. Unfortunately, we were unable to obtain information on the motivation for theft or specifics on the items being stolen; studies that provide greater detail on such information may aid in understanding the nature of theft behaviors in individuals with eating disorders and how it differs from individuals without eating-related psychopathology who also engage in theft behaviors.

4.4 | Limitations

This study has several limitations. First, as register data captured only treatment-seeking individuals and since the coverage of registers has increased over time (Javaras et al., 2015), individuals with eating disorders who remained outside of the health care system would not be

identifiable from registers, resulting in exposure misclassification. The misclassification of BN is likely to be greater than that of AN since BN was not a separate diagnosis in the Swedish version of ICD-9 while AN was ("Klassifikation av Sjukdomar 1987," 1987). This exposure misclassification is probably nondifferential in terms of the criminal outcomes that happened subsequent to diagnosis, and would most likely result in underestimations of the true effects (Flegal, Brownie, & Haas, 1986). In addition, the fact that eating disorder exposures in this study reflected cases that were identified by the health care system, and knowing that a significant proportion of individuals with eating disorders do not seek medical treatment (Ali et al., 2017; Regan, Cachelin, & Minnick, 2017), may limit the generalizability of the results within the more severe end of the diagnostic spectrum. Second, many criminal behaviors in Sweden are not reported, meaning that law-breaking individuals may not be charged or convicted (*Victims' tendency to report crime*, 2008), resulting in misclassification of criminal outcomes in our study. Such misclassification would bias the estimates of associations if it differed between exposed and unexposed individuals; otherwise, if independent of the eating disorder, the misclassification is not likely to bias the estimations of relative risks (such as HRs). The results were based on information on convictions (i.e., did not include crimes that were not reported or crimes that were committed but did not lead to convictions) and may have limited generalizability to any criminal behaviors. Third, sibling-comparisons account for unmeasured familial factors that make siblings similar, such as genetic factors, shared diet, familial socioeconomic conditions, and so forth. However, these factors may also have nonshared effects (e.g., full-siblings may cope differently with poor family socioeconomic conditions) and the sibling-comparisons are unable to adjust for such nonshared effects of common risk factors. Such residual confounders could even bias the estimates from sibling-comparisons if full-sisters are less similar in terms of these confounders than of exposures (Frisell, Oberg, Kuja-Halkola, & Sjolander, 2012), but we were unable to quantify such influences or specify such factors. Fourth, prior knowledge could not inform whether comorbid psychiatric disorders were confounders, mediators, and/or colliders (i.e., common results of eating disorders and criminality) (Greenland, 2003) in terms of the associations of interest. Adjusting for comorbidities could introduce bias, if, for instance, the common causes of exposure and comorbidities are independent from the common causes of outcome and comorbidities (D'Onofrio et al., 2016; Greenland, 2003; Yao et al., 2016). Therefore, we did not adjust for more comorbidities and encourage caution when interpreting the adjusted results. Fifth, due to the low prevalence of AN and BN in males in our data, we were unable to conduct parallel analyses for boys and men. Therefore, our results may not generalize to males with eating disorders. Finally, given that the ICD codes could not distinguish between the restrictive and binge/purge subtypes of AN, we were unable to determine whether risk of criminality differed by AN subtype. Similarly, as binge-eating disorder is not specified as an independent diagnostic category in ICD 8, 9, or 10, we could not examine the association of the criminal outcomes with this eating disorder presentation.

5 | CONCLUSION

In this large population-based study, we observed increased risk of being convicted of theft and other crimes in AN and BN. The association with convictions of theft and other crimes was stronger in BN than in AN and was partially explained by comorbidities. BN, but not AN, might share common familial factors with theft behaviors. With reference to clinical practice, although taking a criminal/forensic history should indeed be a component of all psychiatric evaluations, in practice, this may not always be the case with eating disorders. The failure to assess forensic history may in part be due to (erroneous) preconceived notions related to the demographics of the majority of patients with eating disorders (i.e., adolescent to young adult females) as being a low-risk population for criminal activity. Our results provide an important reminder to clinicians to routinely screen for criminal/forensic history in patients with eating disorders.

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CONFLICT OF INTEREST

Dr. Norring is a consultant on a research grant from Shire. Dr. Lichtenstein has served as a speaker for Medice. Dr. Bulik is a consultant for and grant recipient from Shire. Dr. Larsson has served as a speaker for Eli-Lilly and Shire and has received research grants from Shire; all outside this study.

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SUPPORTING INFORMATION

Additional Supporting Information may be found online in the supporting information tab for this article.

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