ORIGINAL ARTICLE

Associations between parental socioeconomic-, family-, and sibling status and risk of eating disorders in offspring in a Danish national female cohort

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Abstract

Objective: Studies on parental socioeconomic status (SES) and family risk factors for eating disorders (EDs) have yielded inconsistent results; however, several studies have identified high parental educational attainment as a risk factor. The aim was to evaluate associations of parental SES and family composition with anorexia nervosa (AN), bulimia nervosa (BN), and eating disorders not otherwise specified (EDNOS) in the offspring, adjusting for parental age and parental mental health.

Methods: The cohort included women born in Denmark between January 1, 1989 and December 31, 2010, derived from Danish national registers. Each person was followed from their sixth birthday until onset of the disorder of interest or to December 31, 2016. Exposure variables were: childhood SES, defined as individually evaluated parental level of income, occupation, and education; sibling status; and family composition. Outcomes were: AN, BN, EDNOS, and major depressive disorder (MDD), included as a psychiatric comparison disorder. Risks were estimated using Cox proportional hazards.

Results: High parental SES was associated with increased risk of especially AN, and less so BN and EDNOS, in offspring. In comparison, low SES was associated with a higher risk of MDD. No differences between maternal or paternal socioeconomic risk

factors were found. Family composition and sibling status showed limited influence on ED risk.

Discussion: SES shows opposite associations with AN than MDD, whereas associations with BN and EDNOS are intermediate. The socioeconomic backdrop of AN differs markedly from that reported in other psychiatric disorders. Whether that is due to genetic and/or environmental factors remains unknown.

Public Significance statement: Parental socioeconomic background (SES) may influence eating disorders risk in offspring somewhat differently than other psychiatric disorders. In Denmark, higher parental SES was associated with increased risk of, particularly, anorexia nervosa (AN). Importantly AN does strike across the SES spectrum. We must ensure that individuals of all backgrounds have equal access to care and are equally likely to be detected and treated appropriately for eating disorders.

1 | INTRODUCTION

The etiology of eating disorders (EDs) is complex and not fully understood. Genetic, biological, and psychosocial risk and resilience factors appear to act in the development of the illnesses (Jacobi et al., 2004; Woerwag-Mehta & Treasure, 2008). Putative risk or resilience factors for anorexia nervosa (AN) (Woerwag-Mehta & Treasure, 2008), for bulimia nervosa (BN), and eating disorders not otherwise specified (EDNOS) (Jacobi et al., 2004) have been explored to better understand the mechanisms underlying the development of the disorders and thereby improve prediction, prevention, and treatment. In previous studies of the Danish general population we have focused on perinatal risk factors (Larsen et al., 2021), adverse events in childhood (Larsen et al., 2017), and autoimmune and autoinflammatory diseases (Zerwas et al., 2017) as risk factors for AN, BN, and EDNOS. The aim of this study is to evaluate the associations between parental socioeconomic factors (defined as individually evaluated parental levels of income, occupation, and education), family composition and sibling status and the risk of AN, BN, and EDNOS in the offspring.

1.1 | Socioeconomic factors

Longitudinal nationwide register studies (Goodman, Heshmati, & Koupil, 2014; Goodman, Heshmati, Malki, et al., 2014; Kendler et al., 2018; Razaz & Cnattingius, 2018; Sundquist et al., 2017), population-based birth cohort studies, predominantly from Sweden, and cross-sectional studies have identified high levels of parental education (Ahren-Moonga et al., 2009; Goodman, Heshmati, & Koupil, 2014; Goodman, Heshmati, Malki, et al., 2014; Kendler et al., 2018; O'Brien et al., 2017; Sundquist et al., 2017), maternal education (Ahrén et al., 2012; Ahren-Moonga et al., 2009; Razaz &

Cnattingius, 2018), income, and socioeconomic status (SES) as risk factors for the development of AN (Lindberg & Hjern, 2003; Razaz & Cnattingius, 2018), BN (Goodman, Heshmati, Malki, et al., 2014), EDNOS (Goodman, Heshmati, Malki, et al., 2014), and EDs in general (Ahrén et al., 2013; Ahren-Moonga et al., 2009; Björkenstam et al., 2017; O'Brien et al., 2017; Sundquist et al., 2017) in offspring. Conversely, associations between lower parental income level (Ahrén et al., 2013) and adverse sociodemographic conditions (including moving, not living with parents, food insecurity) with AN (Fairburn et al., 1999; Lindberg & Hjern, 2003; Ottosen & Skov, 2010), BN (Fairburn et al., 1997), EDNOS (Fairburn et al., 1998), and EDs in general (Jacobi et al., 2004; O'Brien et al., 2017; Ottosen & Skov, 2010) have also been reported. A recent systematic review of SES and associations with AN, BN, and binge-eating disorder concluded that no consistent evidence supports that certain socioeconomic backgrounds determine the risk of any ED (Huryk et al., 2021). However, studies were predominantly cross-sectional, reporting on own and not on parental status, many studies included small sample-sizes, leaving a risk of selection bias and with no possibility of determining causality. Furthermore, different definitions of SES hamper comparison across studies. Adequately powered, population-based studies with longitudinal designs are needed to further explore the influence of parental socioeconomic factors as risk factors for the development of specific EDs (Huryk et al., 2021; Weissman, 2019).

1.2 | Family composition and sibling status

The impact of family composition and number of siblings as possible risk factors for development of EDs have also yielded divergent results. One study reported that the risk of AN was increased in families with step-parents (Fairburn et al., 1999), whereas another study found no effect of family composition (Razaz &

Cnattingius, 2018). Other studies have indicated that having full siblings is associated with lower risk of AN, EDNOS (Goodman, Heshmati, Malki, et al., 2014), and EDs (Ahrén et al., 2013) as does having brothers in particular (Eagles et al., 2005; Steinhausen et al., 2015). In contrast, having half siblings has been associated with increased risk of BN, EDNOS (Goodman, Heshmati, Malki, et al., 2014), and EDs (Ahrén et al., 2013). Finally, elevated risk of AN has been associated with being born as the second or later child in the birth order (Eagles et al., 2005).

1.3 | Aims

Our aim was to replicate and extend findings from the longitudinal Swedish register-based study (Goodman, Heshmati, Malki, et al., 2014) in a Danish register-based sample with adequate statistical power to test the consistency of their findings of high parental education being associated with increased risk of AN, BN, and EDNOS and by comparing them with offspring with major depressive disorder (MDD) as a psychiatric control (Plana-Ripoll et al., 2019). An indirect comparison with our previous study on OCD using a similar study design will also be possible (Yilmaz et al., 2022).

We hypothezied that (1) high childhood parental SES (defined as maternal and paternal income-, occupational-, and educational level) would increase the risk of any ED but not MDD in the offspring; and (2) that living in a nuclear family with full siblings would decrease the risk of developing any ED and MDD in the offspring, beyond the influence of the correlated factors of maternal and paternal age and parental mental health in a longitudinal design enabling the determination of timing of exposures in relation to outcomes.

2 | METHODS

2.1 | Data sources

In Denmark, each resident is assigned a unique personal identification number allowing accurate linkage of the many national population-based registers. Data used in this study were obtained from the following registers:

Information on index persons, their parents and siblings came from the Civil Registration System (Pedersen, 2011), which was established in 1968 and contains information on sex, place and date of birth, vital status, date of emigration or death, and addresses. The Civil Registration System also contains personal identifiers of parents, allowing determination of parental ages, full and half siblings, and family composition.

Information on hospital admissions and outcome diagnoses came from the National Patient Register (Lynge et al., 2011) and the Psychiatric Central Research Register (Mors et al., 2011), which record information on diagnoses, type of admission (inpatient or outpatient), and admission and discharge dates for all patients in Danish hospitals. The National Patient Register and the Psychiatric Central Research Register have recorded information on inpatient contacts since their inceptions in 1977 and 1969, respectively, and on outpatient contacts since 1995.

Information on parental socioeconomic factors came from registers in Statistics Denmark; *income level* was obtained from the Income Statistics Register (Baadsgaard & Quitzau, 2011), which includes a wide range of variables regarding individual wages, transfer payments, pensions, and capital income for each year. *Occupational information* came from the Integrated Database on Labor Market Research (Petersson et al., 2011), which includes various variables regarding employment for each individual and each year, including primary occupation. Finally, the Population's Education Register (Jensen & Rasmussen, 2011) contains *information on ongoing and highest completed education* for each individual.

2.2 | Study design and population

In this population-based register-based cohort study, the cohort comprised all women born in Denmark from January 1, 1989 to December 31, 2010 to parents who were also born in Denmark, based on information from the Civil Registration System. Males were not included because the incidence of EDs is lower in males (Zerwas et al., 2015) and therefore analyses with males are often underpowered (male/female ratio is at least 1:12 for EDs in a similar cohort) (Larsen et al., 2021). Each person was followed from their sixth birthday until onset of the disorder of interest, emigration, death, or December 31, 2016, whichever came first. Persons who emigrated or died before age 6 were excluded.

2.3 | Exposures

The evaluated exposures were: order of the child's birth, number of full and half siblings, family composition (single parent/both parents living together), low/medium/high level of maternal and paternal income (separately, in tertiles per calender year), occupational level (outside the labor market; manual and nonmanual employee; director or leader), and basic (compulsory school), short (vocational training/high school), medium (up to and including bachelor), or higher education (graduate/postgraduate degree) (for detailed definitions see Table S1). All exposures, when possible, were defined in childhood at start of follow-up, that is, the year the index person turned 6, ensuring exposure came before outcome. Only numbers of half and full siblings were considered time-dependent variables, to include all of the index person's siblings (not all of them were born the year the index person turned 6). Parents' time spent at work (half time/full time employment) was investigated in the initial analyses in a subsample of the cohort (index persons born January 1, 1989 to December 31, 2006) because data were not available from 2007 onwards.

2.4 | Outcomes

The outcomes were defined as ICD-10 diagnoses (World Health Organization, 1993), which is currently in use in Denmark, in the offspring of narrow AN (ICD-10: F50.0), broad AN (ICD-10: F50.0, F50.1),

TABLE 1 Hazard ratio of eating disorders and major depressive disorder in Danish women born in 1989–2010, exposed to family and socioeconomic factors before age 6 (univariable analyses)

		Narrow anorexia nervosa HR (95% CI)	Broad anorexia nervosa HR (95% CI)	Bulimia nervosa HR (95% CI)	EDNOS HR (95% CI)	Major depressive disorder HR (95% CI)
Number of cases		2722	4146	1773	2954	13,881
Age of onset	Mean (SD)	16.7 (3.2)	16.5 (3.3)	19.6 (3.0)	17.2 (3.5)	18.6 (3.5)
Covariates used for adjustment						
Urbanicity	Capital	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
	Capital suburb	0.90 (0.78, 1.03)	0.89 (0.80, 1.00)	0.99 (0.84, 1.17)	**1.23 (1.06, 1.43)**	1.06 (0.99, 1.14)
	Provincial city	**0.80 (0.70, 0.93)**	0.93 (0.83, 1.04)	**0.72 (0.60, 0.86)***	1.14 (0.98, 1.32)	0.95 (0.88, 1.02)
	Provincial town	**0.72 (0.64, 0.82)***	**0.76 (0.69, 0.84)***	**0.70 (0.61, 0.82)***	1.12 (0.99, 1.28)	0.98 (0.93, 1.04)
	Rural area	**0.61 (0.55, 0.69)***	**0.64 (0.58, 0.71)***	**0.57 (0.49, 0.67)***	0.95 (0.84, 1.08)	**0.93 (0.88, 0.98)**
Maternal age at birth	<21	**0.57 (0.42, 0.77)***	**0.65 (0.52, 0.82)***	0.86 (0.64, 1.15)	1.09 (0.89, 1.34)	**1.48 (1.37, 1.60)***
	21–29	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
	30–39	**1.22 (1.13, 1.32)***	**1.16 (1.09, 1.23)***	**1.12 (1.02, 1.23)**	1.04 (0.96, 1.12)	0.96 (0.93, 1.00)
	40+	1.21 (0.90, 1.64)	1.08 (0.84, 1.39)	1.06 (0.71, 1.59)	1.00 (0.74, 1.37)	1.03 (0.89, 1.19)
Paternal age at birth	<21	**0.44 (0.25, 0.78)***	**0.53 (0.35, 0.81)**	0.79 (0.48, 1.30)	**0.62 (0.40, 0.96)**	**1.42 (1.25, 1.61)***
	21–29	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
	30–39	**1.18 (1.09, 1.28)**	**1.11 (1.04, 1.19)***	**1.12 (1.02, 1.24)**	0.96 (0.89, 1.03)	**0.91 (0.88, 0.95)**
	40+	**1.19 (1.03, 1.38)**	1.09 (0.96, 1.22)	1.11 (0.92, 1.33)	1.13 (0.99, 1.29)	1.02 (0.96, 1.09)
Parental psychiatric illness	No	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
	Other	0.96 (0.87–1.06)	1.07 (0.99–1.15)	**1.26 (1.13–1.40)***	**1.51 (1.39–1.63)***	**1.98 (1.91–2.05)***
	ED	**2.33 (1.66–3.27)***	**2.30 (1.74–3.05)***	**1.93 (1.16–3.21)**	**1.96 (1.34–2.86)***	**2.11 (1.76–2.54)***
Exposures						
Family factors						
Birth order	1st	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
	2nd	1.01 (0.93, 1.10)	1.00 (0.94, 1.07)	1.10 (0.99, 1.22)	1.05 (0.97, 1.13)	**1.06 (1.02, 1.10)***
	≥3rd	**0.82 (0.73, 0.92)***	**0.81 (0.74, 0.89)***	0.98 (0.85, 1.13)	0.94 (0.85, 1.05)	**1.17 (1.12, 1.22)***
No. of full siblings	0	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
	1	1.08 (0.97, 1.21)	1.06 (0.97, 1.15)	0.95 (0.84, 1.09)	**0.86 (0.78, 0.95)**	**0.71 (0.68, 0.74)***
	≥2	1.09 (0.97, 1.23)	1.03 (0.94, 1.13)	1.04 (0.90, 1.20)	**0.80 (0.72, 0.89)***	**0.69 (0.66, 0.72)***
No. of half siblings	0	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
	1	**0.84 (0.74, 0.94)**	0.91 (0.83, 1.00)	1.03 (0.90, 1.19)	**1.12 (1.01, 1.25)**	**1.41 (1.34, 1.47)***
	≥2	**0.77 (0.69, 0.86)***	**0.87 (0.80, 0.95)**	1.04 (0.92, 1.18)	**1.20 (1.09, 1.32)***	**1.63 (1.57, 1.70)***

TABLE 1 (Continued)

		Narrow anorexia nervosa HR (95% CI)	Broad anorexia nervosa HR (95% CI)	Bulimia nervosa HR (95% CI)	EDNOS HR (95% CI)	Major depressive disorder HR (95% CI)
Family composition	Single parent	**0.80 (0.72, 0.89)***	**0.91 (0.84, 0.98)**	1.10 (0.98, 1.23)	**1.26 (1.15, 1.37)***	**1.64 (1.58, 1.70)***
	Both parents	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
Parental socioeconomic factors						
Maternal income level	Low	**0.88 (0.79, 0.97)**	0.94 (0.87, 1.02)	0.97 (0.86, 1.09)	1.00 (0.91, 1.09)	**1.14 (1.09, 1.18)***
	Medium	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
	High	**1.19 (1.09, 1.30)***	**1.16 (1.08, 1.25)***	**1.13 (1.01, 1.26)**	0.96 (0.88, 1.05)	**0.82 (0.78,0.85)***
Paternal income level	Low	**0.83 (0.75, 0.92)***	**0.85 (0.78, 0.92)***	0.88 (0.78, 1.00)	1.02 (0.94, 1.12)	**1.28 (1.23, 1.33)***
	Medium	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
	High	**1.30 (1.19, 1.41)***	**1.21 (1.13, 1.30)***	1.10 (0.98, 1.22)	0.98 (0.90,1.07)	**0.84 (0.81, 0.88)***
Parental income level	Both low	**0.75 (0.64, 0.88)***	**0.82 (0.72, 0.93)***	0.87 (0.72, 1.04)	1.04 (0.92, 1.18)	**1.25 (1.19, 1.32)***
	At least one medium	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
	At least one high	**1.38 (1.27, 1.51)***	**1.27 (1.19, 1.37)***	**1.16 (1.04, 1.29)**	0.99 (0.91, 1.07)	**0.76 (0.73, 0.79)***
Maternal occupational level	Outside labor market	0.91 (0.81, 1.01)	0.95 (0.87, 1.03)	1.05 (0.93, 1.18)	**1.18 (1.07, 1.29)***	**1.48 (1.43, 1.54)***
	Low/medium level	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
	High level	**1.45 (1.31, 1.60)***	**1.36 (1.25, 1.48)***	**1.28 (1.12, 1.46)***	1.10 (0.99, 1.23)	**0.91 (0.86, 0.96)***
Paternal occupational level	Outside labor market	**0.75 (0.63, 0.89)***	**0.81 (0.72, 0.93)***	0.95 (0.79,1.15)	**1.25 (1.10, 1.42)***	**1.52 (1.44, 1.60)***
	Low/medium level	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
	High level	**1.38 (1.26, 1.51)***	**1.27 (1.18, 1.36)***	**1.25 (1.12, 1.41)***	1.05 (0.95, 1.15)	**0.83 (0.79, 0.87)***
Parental occupational level	Both outside labor market	**0.60 (0.45, 0.80)***	**0.74 (0.60, 0.91)**	0.78 (0.58, 1.06)	**1.34 (1.12, 1.61)**	**1.74 (1.62, 1.87)***
	Low/medium level	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
	High level	**1.42 (1.31, 1.54)***	**1.30 (1.22, 1.39)***	**1.28 (1.15, 1.42)***	1.02 (0.94, 1.11)	**0.82 (0.79, 0.86)***
Maternal educational level	Basic level	**0.58 (0.52, 0.66)***	**0.67 (0.61, 0.73)***	**0.66 (0.57, 0.76)***	1.04 (0.94, 1.15)	**1.54 (1.47, 1.62)***
	Short	**0.77 (0.70, 0.84)***	**0.82 (0.76, 0.88)***	**0.85 (0.76, 0.96)**	0.93 (0.85, 1.02)	**1.13 (1.08, 1.18)***
	Medium	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
	Long	**1.18 (1.03, 1.35)**	**1.21 (1.08, 1.35)***	**1.23 (1.02, 1.48)**	0.90 (0.77, 1.06)	0.95 (0.88, 1.04)
Paternal educational level	Basic level	**0.58 (0.52, 0.66)***	**0.65 (0.59, 0.72)***	**0.67 (0.57, 0.78)***	0.97 (0.86, 1.09)	**1.42 (1.35, 1.50)***
	Short	**0.75 (0.67, 0.82)***	**0.80 (0.73, 0.87)***	**0.81 (0.71, 0.92)***	0.93 (0.84, 1.03)	**1.07 (1.01, 1.12)**
	Medium	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
	Long	1.13 (0.99, 1.29)	1.11 (1.00, 1.24)	1.08 (0.91, 1.29)	0.90 (0.78, 1.05)	**0.83 (0.77, 0.90)***
	Basic level	**0.50 (0.42, 0.60)***	**0.59 (0.51, 0.67)***			**1.61 (1.52, 1.70)***

TABLE 1 (Continued)

		Narrow anorexia nervosa HR (95% CI)	Broad anorexia nervosa HR (95% CI)	Bulimia nervosa HR (95% CI)	EDNOS HR (95% CI)	Major depressive disorder HR (95% CI)
Parental educational level				**0.53 (0.43, 0.64)***	1.06 (0.93, 1.21)	
	Short	**0.72 (0.66, 0.78)***	**0.78 (0.73, 0.84)***	**0.76 (0.68, 0.84)***	0.95 (0.87, 1.03)	**1.15 (1.10, 1.20)***
	Medium	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
	Long	**1.22 (1.09, 1.36)***	**1.19 (1.08, 1.30)***	1.12 (0.97, 1.29)	0.91 (0.80, 1.03)	**0.86 (0.81, 0.92)***

Note: All analyses were adjusted for age, time period, urbanicity (but were not otherwise mutually adjusted). Bold: Results that are statistically significant at $\alpha = 0.05$. Associations still significant after adjusting for multiple testing are marked with *.

Abbreviations: CI, confidence interval; EDNOS, eating disorder not otherwise specified; HR, hazard ratio.

BN (ICD-10: F50.2, F50.3), EDNOS (ICD-10: F50.8, F50.9), and MDD (ICD-10: F32, F33) in either the National Patient Register or the Psychiatric Central Research Register. Only the EDNOS diagnosis is available in ICD-10 and not specific ED diagnoses such as binge-eating disorder. A person could be in more than one of the diagnostic groups (Figure S1). MDD in the offspring was included as a psychiatric comparison, representing a prevalent major mental illness. The date of onset for each outcome was defined as the registration date for the first inpatient or outpatient contact leading to a discharge diagnosis of the respective disorder. To minimize misdiagnoses of other childhood feeding and eating disorders, any ED diagnosis before age 6 was not included. Individuals diagnosed with MDD before age 6 were excluded from that particular analysis.

2.5 | Statistical analysis

For each exposure, we performed survival analyses using Cox proportional hazards regression with age as the underlying time variable. We estimated hazard ratios (HRs) with 95% confidence intervals (CIs) for each type of disorder. Variables for the analyses were chosen on the basis of findings from the literature and from our earlier study from a similar birth cohort (Larsen et al., 2017). The included variables are moderately correlated, all with Pearson correlation coefficient below 0.6. Given the power and large number of exposed cases, reducing the number of parameters for the multivariabte analyses was not necessary and automatic tools such as Lasso did not exclude any variables (Chasseloup et al., 2020). To account for multiple testing, we derived false discovery rate corrected p values using the Benjamini–Hochberg method, accounting for the number of test in each set of analyses separately. All analyses were adjusted for calendar time, categorized as 1995–2005, 2006–2011, and 2012–2016 and urbanicity defined as living in the capital, capital suburb, provincial city, provincial town, or rural area. All multivariable analyses were additionally adjusted for maternal/paternal age (in 10-year intervals) at the child's birth and parental psychiatric illness (no, ED, other). Test for trend was performed for the dosage variables assuming the same effect from one level to the level above, and p values were reported.

Finally, we performed interaction analyses, to ensure that parental ages did not explain the distribution of the socioeconomic exposures, testing whether the linear effect of income and educational level differed across categories of maternal and paternal age.

All analyses were performed in STATA 15 (StataCorp., 2017). The study was approved by the Danish Data Protection Agency. By Danish law, informed consent and approval by the ethical committee is not required for register-based studies. The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

3 | RESULTS

3.1 | Incidence

From a total population of 561,816 women who were followed-up for more than 6.2 million person years until a maximum age of 28, we identified 2722 cases with narrow AN, 4146 with broad AN, 1773 with BN, 2954 with EDNOS, and 13,979 with MDD (Table S2 and Figure S1). The incidence rates for both narrow and broad AN and EDNOS were highest in the 13–22-year olds; the incidence rates for BN and MDD were highest in the 18–28-year olds (Table S2).

3.2 | Univariable analyses

The risk of EDNOS was lower when having two or more full siblings (HR: 0.80; 95% CI: 0.72–0.89) and of MDD (one sibling, 0.71; 0.68–0.74; two or more siblings, 0.69; 0.66–0.72) compared to having no siblings. Having two or more half siblings had a protective effect on the risk of narrow AN (0.77; 0.69–0.86) compared to having no half siblings (Table 1). In contrast, the risk of EDNOS and MDD was higher with a greater number of half siblings with hazards ranging from 1.20 (1.09–1.32) to 1.63 (1.57–1.70) compared to having no half siblings. No other significant effects regarding number of siblings, or having a full or half sibling were found for any of the outcome illnesses.

TABLE 2 Hazard ratio for eating disorders and major depressive disorder in Danish women born in 1989–2010, exposed to family end socioeconomic factors before age 6 (multivariable analyses)

		Narrow anorexia nervosa HR (95% CI)	Broad anorexia nervosa HR (95% CI)	Bulimia nervosa HR (95% CI)	EDNOS HR (95% CI)	Major depressive disorder HR (95% CI)
Covariates used for adjustment						
Urbanicity	Capital	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
	Capital suburb	0.97 (0.84, 1.11)	0.97 (0.86, 1.09)	1.03 (0.87, 1.23)	**1.23 (1.06, 1.44)**	1.07 (0.99, 1.14)
	Provincial city	0.86 (0.74, 1.00)	0.99 (0.88, 1.12)	**0.76 (0.63, 0.91)**	1.17 (1.00, 1.36)	0.93 (0.87, 1.00)
	Provincial town	**0.83 (0.73, 0.95)**	**0.86 (0.78, 0.96)**	**0.78 (0.67, 0.92)**	1.15 (1.00, 1.32)	**0.93 (0.87, 0.99)**
	Rural area	**0.77 (0.68, 0.88)***	**0.78 (0.71, 0.87)***	**0.67 (0.57, 0.79)***	0.98 (0.86,1.13)	**0.88 (0.83, 0.94)***
Maternal age at birth	<21	0.78 (0.56, 1.09)	0.78 (0.61, 1.00)	0.94 (0.68, 1.31)	1.02 (0.81, 1.28)	1.02 (0.93, 1.12)
	21–29	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
	30–39	**1.16 (1.06, 1.28)**	**1.14 (1.05, 1.23)***	1.05 (0.93, 1.19)	1.05 (0.96, 1.16)	1.01 (0.97, 1.06)
	40+	1.22 (0.88, 1.69)	1.11 (0.84, 1.47)	0.98 (0.63, 1.52)	0.93 (0.67, 1.31)	0.96 (0.82, 1.12)
Paternal age at birth	<21	0.62 (0.33, 1.16)	0.69 (0.44, 1.08)	0.89 (0.51, 1.53)	**0.55 (0.35, 0.87)**	0.99 (0.86, 1.15)
	21–29	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
	30–39	1.06 (0.96, 1.16)	1.03 (0.96, 1.12)	1.06 (0.94, 1.19)	0.97 (0.88, 1.06)	0.98 (0.94, 1.02)
	40+	1.09 (0.92, 1.29)	1.00 (0.87, 1.15)	1.01 (0.82, 1.24)	1.09 (0.93, 1.28)	0.95 (0.88, 1.02)
Parental psychiatric illness	No	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
	Other	**1.17 (1.06–1.30)**	**1.24 (1.15–1.35)***	**1.40 (1.25–1.57)***	**1.46 (1.33–1.59)***	**1.66 (1.60–1.73)***
	ED	**2.72 (1.93–3.84)***	**2.62 (1.97–3.48)***	**2.00 (1.18–3.40)**	**1.95 (1.33–2.85)***	**1.84 (1.52–2.22)***
Exposures						
Family factors						
Birth order	1st	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
	2nd	0.94 (0.86, 1.03)	0.95 (0.88, 1.02)	1.08 (0.97, 1.21)	1.05 (0.97, 1.15)	**1.11 (1.07, 1.16)***
	≥3rd	**0.71 (0.62, 0.82)***	**0.73 (0.65, 0.82)***	0.91 (0.76, 1.08)	0.92 (0.80, 1.05)	**1.16 (1.10, 1.24)***
No. of full siblings	0	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
	1	0.92 (0.80, 1.04)	0.98 (0.88, 1.08)	0.95 (0.81, 1.11)	0.94 (0.83, 1.05)	**0.93 (0.88, 0.98)**
	≥2	0.98 (0.85, 1.14)	1.02 (0.90, 1.15)	1.08 (0.90, 1.29)	0.92 (0.80, 1.06)	**0.90 (0.86, 0.97)**
No. of half siblings	0	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
	1	0.92 (0.80, 1.04)	0.98 (0.89, 1.09)	1.06 (0.91, 1.23)	1.00 (0.89, 1.12)	**1.14 (1.09, 1.20)***
	≥2	0.92 (0.80, 1.06)	1.00 (0.90, 1.12)	1.11 (0.94, 1.30)	1.01 (0.89, 1.15)	**1.17 (1.11, 1.24)***
Family composition	Single parent	0.96 (0.84, 1.09)	1.01 (0.91, 1.11)	1.16 (1.00, 1.34)	**1.14 (1.02, 1.27)**	**1.16 (1.10, 1.22)***
	Both parents	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)

TABLE 2 (Continued)

		Narrow anorexia nervosa HR (95% CI)	Broad anorexia nervosa HR (95% CI)	Bulimia nervosa HR (95% CI)	EDNOS HR (95% CI)	Major depressive disorder HR (95% CI)
Parental socioeconomic factors						
Maternal income level	Low	**0.89 (0.79, 0.99)**	0.96 (0.87, 1.05)	0.96 (0.84, 1.10)	0.95 (0.86, 1.05)	0.99 (0.95, 1.04)
	Medium	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
	High	1.00 (0.91, 1.11)	1.01 (0.93, 1.09)	1.00 (0.89, 1.13)	0.95 (0.87, 1.05)	**0.89 (0.85, 0.93)***
Paternal income level	Low	0.93 (0.83,1.04)	0.91 (0.83,1.00)	0.90 (0.78,1.03)	0.94 (0.85,1.04)	**1.06 (1.02,1.11)**
	Medium	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
	High	**1.17 (1.06, 1.28)***	**1.12 (1.04, 1.21)**	1.00 (0.89, 1.13)	1.00 (0.91, 1.09)	**0.92 (0.88,0.96)***
Maternal occupational level	Outside labor market	1.12 (0.99, 1.27)	1.08 (0.98, 1.19)	1.12 (0.97, 1.30)	**1.13 (1.01, 1.26)**	**1.16 (1.11, 1.22)***
	Low/medium level	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
	High level	**1.15 (1.02, 1.30)**	**1.13 (1.02, 1.25)**	1.03 (0.88, 1.21)	1.13 (1.00, 1.29)	1.07 (1.00, 1.15)
Paternal occupational level	Outside labor market	0.86 (0.72, 1.05)	0.92 (0.80, 1.07)	0.99 (0.81, 1.22)	1.11 (0.96, 1.28)	1.02 (0.96, 1.09)
	Low/medium level	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
	High level	1.02 (0.92, 1.14)	0.99 (0.90, 1.08)	1.03 (0.89, 1.18)	1.06 (0.95, 1.19)	1.01 (0.96, 1.07)
Maternal educational level	Basic level	**0.79 (0.67, 0.90)***	**0.82 (0.73, 0.92)***	**0.70 (0.59, 0.82)***	0.97 (0.85, 1.10)	**1.09 (1.02, 1.15)**
	Short	**0.88 (0.79, 0.98)**	**0.91 (0.84, 0.99)**	0.91 (0.80, 1.04)	0.94 (0.85, 1.04)	1.04 (0.99, 1.09)
	Medium	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
	Long	0.99 (0.85, 1.15)	1.05 (0.93, 1.19)	1.16 (0.95, 1.43)	0.89 (0.75, 1.07)	1.06 (0.96, 1.16)
Paternal educational level	Basic level	**0.78 (0.67, 0.90)***	**0.79 (0.71, 0.89)***	**0.76 (0.64, 0.91)**	0.92 (0.80, 1.06)	1.04 (0.97, 1.10)
	Short	**0.88 (0.79, 0.98)**	**0.90 (0.82, 0.98)**	0.89 (0.77, 1.03)	0.95 (0.85, 1.06)	0.95 (0.90, 1.00)
	Medium	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
	Long	1.02 (0.89, 1.17)	1.03 (0.91, 1.15)	1.01 (0.84, 1.22)	0.90 (0.77, 1.06)	**0.87 (0.80, 0.95)***

Note: All analyses were additionally adjusted for age, time period and were mutually adjusted for all other covariates in the table. Bold: Results that are statistically significant at $\alpha = 0.05$. Associations still significant after adjusting for multiple testing are marked with *.
Abbreviations: CI, confidence interval; EDNOS, eating disorder not otherwise specified; HR, hazard ratio.

Low paternal income level (0.85; 0.78–0.92) compared to medium income level decreased the risk of broad AN, but was not significant for the maternal income level. Fathers being outside the labor market decreased the risk of narrow (0.75; 0.63–0.89) and broad AN (0.81; 0.72–0.93) compared to low/medium level occupation, but results were not significant for mothers. High maternal, but not paternal, educational level was associated with increased risk of broad AN (1.21; 1.08–1.35) compared to medium level, and high paternal educational level compared to medium

level reduced the risk of MDD (0.83; 077–0.90), but was not significant for mothers. No other significant differences emerged between the effect of maternal, paternal, or parental income-, occupational-, or educational levels on the risk for all outcomes. Altogether, significant effects of SES as risk factors for EDs or MDD could not exclusively be linked to maternal nor paternal SES.

The influence of the parents' time spent at work, investigated in a subsample, showed that only mothers working full time combined

with fathers working part-time yielded a significant result for the risk for MDD (1.57; 1.34–1.84), whereas results for all combinations of parents working full- or part-time were not significantly associated with risk for all ED diagnoses evaluated.

3.3 | Multivariable analyses

Covariates used for adjusting the multivariable analyses showed that parents with an "other" psychiatric disorder compared to parents without a disorder had effects for all outcome illnesses, except narrow AN, with risk estimates ranging from 1.24 (1.15–1.35) for broad AN to 1.66 (1.60–1.73) for MDD. When parents had an ED the risk estimates were even higher and showed the opposite pattern, 1.84 (1.52–2.22) for MDD and 2.71 (1.93–3.84) for narrow AN (Table 2). High maternal age at the child's birth was associated with increased risk of both narrow (test for linear trend, $p = .001$) and broad AN (test for linear trend, $p = .0001$), and older mothers (age 30–39 years at child birth) significantly increased the risk of broad AN (1.14; 1.05–1.23) compared to 21–29 year old mothers. However, maternal age at the child's birth had no influence on BN, EDNOS, or MDD. Paternal age had no independent influence on either outcome.

Results of family factors showed that the risk of both narrow (0.71; 0.62–0.82) and broad AN (0.73; 0.65–0.82) decreased by being the third or later child in the birth order compared to being the first born, whereas the risk of MDD increased by being the second (1.11; 1.07–1.16) or third or later in the birth order (1.16; 1.10–1.24). Living in a single parent family compared to living in families with two parents was a risk factor for MDD (1.16; 1.10–1.22). Likewise, having one (1.14; 1.09–1.20) or two (1.17; 1.11–1.24) compared to having no half siblings increased the risk of MDD. No other of the family factors kept significance in the multivariable analyses.

Among parental socioeconomic factors, elevated risks on narrow AN (1.17; 1.06–1.28) was associated with high paternal income, whereas both high maternal (0.89; 0.85–0.93) and paternal income (0.92; 0.88–0.96) had protective effects on risk of MDD. Linear test for trend confirmed a dose–response pattern with a positive trend for both maternal income for narrow AN ($p < .03$) and paternal income for narrow and broad AN (both $p < .0001$), but a negative trend for both maternal and paternal income for MDD (both $p < .0001$). Occupational level of neither parent kept significance regarding the risk of any ED, but having a mother who was outside the labor market was associated with increased risk for MDD (1.16; 1.11–1.22). Basic level of maternal education compared to medium level was protective of narrow (0.79; 0.67–0.90) and broad AN (0.82; 0.73–0.92) and BN (0.70; 0.59–0.82), and similar results were seen for paternal basic education for narrow (0.78; 0.67–0.90) and broad AN (0.79; 0.71–0.89). This pattern was not significant for EDNOS and was opposite for MDD where we observed a protective effect of high level paternal education (0.87; 0.80–0.95). Linear test for trend confirmed the positive association for maternal and paternal

education for narrow AN (both $p < .0001$), broad AN (both $p < .0001$), and BN (maternal, $p < .0001$; paternal, $p < .003$), the trends for EDNOS were not significant, whereas a significant negative association existed between paternal (but not maternal) education and MDD ($p < .004$).

Interaction analyses between maternal and paternal ages and their income and educational levels showed that the effect of parental income differed significantly across levels of paternal age: income had a greater effect for paternal age at birth <21 years compared with the other age categories. No other interactions were significant; thus, this is an expected chance finding when testing 20 interactions.

4 | DISCUSSION

In this longitudinal study based on the total Danish female population born 1989–2010 and followed-up until 2016, we investigated whether childhood parental SES (i.e., maternal and paternal income, occupational and educational level) was a risk factor for AN, BN, or EDNOS in the offspring, when the influence of other factors such as parental age, parental mental health, and family composition were adjusted for.

Even though EDs emerge in individuals from all socioeconomic backgrounds (Ahrén et al., 2012; Huryk et al., 2021), results suggest an elevated risk of developing narrow and broad AN with increasing parental SES. This pattern is less pronounced for BN, and there is an association in the opposite direction for MDD, that is, higher risk with lower parental occupation or lower risk with high income or education. These results corroborate earlier findings on AN and BN in Swedish longitudinal studies (Ahrén et al., 2012, 2013; Ahren-Moonga et al., 2009; Björkenstam et al., 2017; Goodman, Heshmati, & Koupil, 2014; Goodman, Heshmati, Malki, et al., 2014; Kendler et al., 2018; Lindberg & Hjern, 2003; Sundquist et al., 2017), increasing confidence in results. However, we found that risk was well-explained by the SES of both parents, not just by maternal educational level as shown in some previous studies (Ahrén et al., 2012, 2013; Ahren-Moonga et al., 2009; Razaz & Cnattingius, 2018).

The similar contribution of maternal and paternal educational level may reflect the fact that men and women in Denmark are very similar socioeconomically; exemplified by the labor market participation in 2018 for adults (aged 16–64 years) being 76.5% for men and 72.6% for women (Statistics Denmark, 2022). Furthermore, all types of families are supported by the Danish social network: nearly all preschool children are in daycare while their parents are at work (Ministry for Children and Social Affairs, 2018). This social context may minimize differential parental contribution to risk. Finally, the time parents spent at work also did not significantly influence the risk of neither EDs nor MDD.

The patterns observed for the development of narrow and broad AN, and to some extent BN, in this study differ from the observed patterns for the development of major psychiatric disorders like MDD and psychotic disorder (Björkenstam et al., 2016, 2017), which are

more frequently associated with low SES and adverse events, such as childhood adversities, divorced parents, low personal or parental educational level, low income, and unemployment. Associations of SES for OCD, however, broadly resemple those observed in AN (Yilmaz et al., 2022). Our results align with a previous study on a similar cohort, where we found that childhood adversities (family disruption, residential instability, placement in out-of-home care, familial death, parental somatic illness, parental psychiatric illness, parental disability, severe parental criminality, and parental substance use disorder) had either no effect or a protective effect on the risk of AN, but increased the risk of BN and EDNOS and the comparison disorders anxiety, OCD, and MDD (Larsen et al., 2017). This protective effect of some childhood adversities (living in a single parent family, household public assistance) on the risk of AN has been described previously (Björkenstam et al., 2016; Lindberg & Hjern, 2003), but not universally (Fairburn et al., 1999). Reasons for these differences are not entirely clear.

Further exploration of possible mechanisms of why and how higher parental SES increases risk for AN and BN is needed. Genetic factors may play a role. Reported twin-based heritabilities for EDs are on average 50%–60% (Bulik et al., 2015). Heritable traits in the parents, which may be linked to both higher SES and higher vulnerability of developing an ED in the offspring, could be part of a developmental pathway. A genome-wide association study has shown significant positive genetic correlations between AN and various measures of educational attainment (years of education, college completion, but not IQ), suggesting that an association previously thought to be environmental may actually reflect shared genetic factors (Watson et al., 2019).

High familial educational level has been linked to high school performance and a subsequent higher risk of AN and BN in offspring, and thereby shows a different educational trajectory than for other psychiatric disorders (Kendler et al., 2018). Whether this association is based on genetic or environmental factors, such as higher expectations or sociocultural values of educational attainment in families with higher SES, which may act as stress factors predisposing for EDs (Ahrén et al., 2012; Fairburn et al., 1999; Goodman, Heshmati, & Koupil, 2014), is unclear.

Perfectionism is another unmeasured trait that has been identified in individuals with EDs (Holland et al., 2013), AN (Fairburn et al., 1999; Wade et al., 2008), and to a lesser extent, in individuals with BN and EDNOS (Fairburn et al., 1998, 1997). Perfectionism includes having high personal standards, good organizational skills, and high reward dependence and may partly be determined by genetic liabilities (Jacobs et al., 2009; Noble et al., 2013; Wade et al., 2008). It is associated with a greater drive for good school performance (Ahrén et al., 2012; Ahren-Moonga et al., 2009; Dura & Bornstein, 1989). Furthermore, a recent Norwegian population-based study reported that perfectionism was overrepresented in girls from perceived affluent backgrounds; however, in this study the finding was independent of parental education (Sand et al., 2021).

We did not, as hypothesized, find a protective effect of having full siblings on the risk of EDs, as reported earlier for AN, EDNOS (Eagles et al., 2005; Goodman, Heshmati, Malki, et al., 2014; Steinhausen

et al., 2015), and ED (Ahrén et al., 2013). Even though the effect of family factors in this study were limited, we observed an opposite socioeconomic risk-pattern between AN and MDD, with a protective or no effect on AN risk and increased risk for MDD associated with being born later (i.e., being exposed to older siblings), living with a single parent, and having more full or half siblings. These are well-known indicators of blended families and of less privileged socioeconomic situations.

Increasing maternal age influenced the risk of broad AN independent of maternal income and educational level supporting the opposite risk-pattern to what was seen for MDD, and which is also reported for other psychiatric illnesses; that is, that early motherhood may pose a risk for development of psychiatric illness in the offspring (McGrath et al., 2014). Paternal age, in contrast, had no independent effect in this study, corroborating with findings of little effect of paternal age in association with EDs in general (McGrath et al., 2014), but differing from a previous Swedish study finding higher risk of AN and EDs with higher parental age (Javaras et al., 2017). Whether these descrepancies are due to methodological differences is unclear. Effects of parental ages may have both genetic and environmental (e.g., selection into late parenthood) background (Javaras et al., 2017; McGrath et al., 2014). Finally, it was evident that psychiatric disorders in parents influence the risk of all EDs and MDD in offspring, which has been extensively documented (Fairburn et al., 1999; Goodman, Heshmati, Malki, et al., 2014; Larsen et al., 2017; Lindberg & Hjern, 2003; Steinhausen et al., 2015). As expected parental EDs predispose more to EDs in the offspring, while other psychiatric illnesses predispose more to MDD.

Even after adjusting for maternal and paternal age and parental psychiatric disorders, together with mutual adjustment for the rest of the variables in the mutivariable analyses, there were still effects of maternal and paternal SES on the outcomes as described. Altogether, these findings point in the direction of influence of different heritable genetic liabilities in the development of AN, EDs, and MDD.

4.1 | Strengths and limitations

The strengths of this study include the population-based cohort design, large number of cases, and opportunity for long-term follow-up without attrition. Since the Danish registers record information on all citizens regarding admissions to public hospitals, where assessment and treatment is free of charge, social selection is minimal. Furthermore, we have adjusted for urbanicity to correct for eventual selection that might be caused by different referral patterns in different parts of the country due to treatment availability or educational levels of the population in certain geographical areas. Nevertheless, we cannot be certain to have avoided residual selection, because treatment seeking behavior among individuals with EDs is less frequent among individuals with other ED diagnosis than AN and BN, as well as individuals with low SES background (Silén et al., 2021; Sonneville & Lipson, 2018). And, we cannot rule out that the referral-pattern in countries in which socioeconomically disadvantaged individuals have less access to health care would yield different results.

The number of AN cases referred to hospital treatment (both inpatient and outpatient) in Denmark is higher than the number of referred cases with BN and EDNOS, which is evident in this study, and is also seen in other European countries (Galmiche et al., 2019). It may be due to more AN cases being detected because of the severe somatic implications of the illness (especially underweight), whereas many normal weight BN and EDNOS cases less often seek help in the public hospitals (only cases from public hospitals are in the patient registries in Denmark). Diagnoses in the Danish patient registries are given by medical doctors in the specialties of internal medicine (somatic referrals) and psychiatry (psychiatric referrals) and are in general of good quality (Bock et al., 2009; Mohr-Jensen et al., 2016; Nissen et al., 2017); however, the EDs diagnoses remain to be validated. A recent study in Sweden has documented the reliability of their register-based ED diagnoses, and, given similarities of the population and healthcare systems, we anticipate that reliability would be similarly high in Denmark (Birgegård et al., 2022).

Although BN and MDD typically have older ages of onset than AN (Pedersen et al., 2014; Zerwas et al., 2015) differences in age of onset were minimized by using initial diagnoses. Furthermore, because maximum age of follow-up was 28 years, only MDD with early debut were included. Even though MDD is frequently comorbid with EDs (Plana-Ripoll et al., 2019), we demonstrated a different parental socioeconomic pattern in EDs and MDD. If MDD and EDs were not comorbid, we would have expected an even greater difference.

Another limitation is that we only have access to information that is recorded in the registers and are unable to assess other potentially important variables such as family environment and psychological characteristics of family members.

Our results on EDNOS fall between those of the other EDs and MDD. In ICD-10, this diagnostic category is heterogeneous, covering both restrictive and bulimic EDs not fulfilling all criteria for typical or atypical disorders and includes binge-eating disorder and other EDs (e.g., avoidant/restrictive food intake disorder) (American Psychiatric Association, 2013), which may influence the results.

4.2 | Conclusion

We conclude that higher parental SES is associated with increased risk of developing narrow and broad AN, and to a lesser extent BN and EDNOS in the offspring, beyond the effect of parental age, parental mental health, and family composition. Family composition and sibling status showed limited influence on the risks of EDs. The socioeconomic risk-pattern of AN was opposite to that of MDD, a representative of a major psychiatric comparison disorder. Our results further describe a somewhat different association pattern in AN (e.g., fewer adverse events and higher SES) than MDD and other psychiatric disorders (Larsen et al., 2017). Clinically, it is important to be aware of possible social selection that may prevent individuals of lower SES to seek treatment. Our results do not suggest that AN is confined to higher socioeconomic strata; the illness occurs across the socioeconomic spectrum but distributes differently than many other major psychiatric conditions.

CONFLICT OF INTEREST

Cynthia M. Bulik: Shire (grant recipient, Scientific Advisory Board); Pearson (author, royalty recipient); Equip Health Inc. (Clinical Advisory Board). Other authors declare no conflict of interest.

DATA AVAILABILITY STATEMENT

Access to data requires application to the Danish Health Data Authority and the Danish Data Protection Agency.

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