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We are family – parents, siblings and eating disorders in a prospective total-population study of 250,000 Swedish males and females

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Abstract

Objectives: Eating disorders (ED) are an important cause of morbidity in adolescents. This study aimed to explore role of parental characteristics and other aspects of family background in the development of ED in adolescent males and females.

Methods: The study builds on a prospective total-population cohort, using register data and record linkage. *The Stockholm Youth Cohort* comprises all children and adolescents resident in Stockholm County during 2001-2007, plus their parents and siblings. Individuals aged 12+ (N=249,884), were followed up to identify cases of ED from specialist care. We investigated associations of family socio-economic position, parental age and family composition with the incidence of ED.

Results: A total of 3251 cases of ED (2971 females and 280 males) were recorded. Cox regression modelling indicated that there was an increased risk of ED in female offspring of parents who had higher education (e.g. adjusted HR 1.69, 95% CI 1.42, 2.02, for degree-level vs. elementary-level maternal education) but not in males (HR 0.73, 95% CI 0.42, 1.28, for degree-level vs. elementary maternal education, $p < 0.001$ for sex interaction). In females, increasing number of full siblings was negatively associated with ED (e.g. fully adjusted HR 0.92, 95% CI 0.87-0.97, per sibling) while increasing number of half-siblings appeared to increase risk of ED (HR 1.05, 95%CI 1.01, 1.09).

Conclusions: The effect of parental education on ED risk varies between sex, while the effect of number of siblings varies according to whether they are full or half siblings. A deeper understanding of these differences and their underlying mechanisms may provide aetiological insight and inform the design of preventive interventions.

Key Words: Eating disorders, adolescents, females, males, family characteristics, socio-economic position

Introduction

Psychiatric ill-health is increasing among Swedish adolescents (1.), and eating disorders (ED) are among the more severe psychiatric conditions that affect this age group (2.). Although traditionally most ED research has focused on females, recent years have also seen increasing attention to ED in males (3-5.). Much of this research has focused on examining gender differences in clinical presentation and co-morbidity (6.). There has also been growing interest in comparing ED risk factors between males and females, with some commonalities reported in relation to the negative effect of factors such as family discord, abuse, depression and childhood overweight/obesity (4; 7-10.). Yet because most research studies contained at most only few males, they generally lacked the statistical power to examine gender differences in ED risk factors. This has applied particularly to risk factors related to family socio-economic position (SEP) or family composition, which are likely to have weaker individual-level effects than severe or specific risk factors such as sexual abuse or irregular eating, but which may have greater population-level health impacts because they are more common.

As for what is known about the effects of such factors in females, previous studies have reported conflicting results with respect to SEP. Several large Swedish studies have indicated that higher parental education and/or social class predict a higher rate of hospitalization for anorexia and/or ED (11-13.). Similar results have been reported in a large British study (14.). This has not been supported in all research, however, with some studies reporting no association between ED/ED symptoms and SEP (15; 16.). Moreover, most studies were based on females only: to our knowledge only one previous study has included reasonably large samples of both males and females and compared SEP associations between the two. This study reported that among Portuguese 13-year old adolescents, ED symptoms were associated with high SEP in females but with low SEP in males (10.).

With regard to parental age, this has been suggested as a risk factor in the development of different psychiatric disorders in offspring, including bipolar disorder (17.), autism (18; 19.) and schizophrenia (20.). Few studies have examined parental age in relation to ED, however, and these have generated inconsistent results. Early ED research indicated an association between higher parental age (especially in mothers) and risk for ED (21; 22.), while later studies reported no such associations (23; 24.). Similarly with respect to family and sibling composition, previous studies have reported conflicting findings (25.). Of two British studies investigating the sex and birth order of the siblings of ED patients, one did not find any associations (26.), while the other suggested that females with anorexia nervosa had fewer brothers and were born later in the birth order (27.).

In this study we had access to an unusually large number of ED cases, identified via exhaustive and multisource case ascertainment. We therefore investigated how family SEP, family composition, parental age, parental income and parental education were associated with the development of adolescent ED in both males and females.

Methods

Study population

The *Stockholm Youth Cohort* (N=589,114) is a database created by record-linkage for all children and adolescents aged 0-17 years who were ever resident in Stockholm County during the period 2001-2007, plus their parents and siblings. The linkage involved a range of national and regional health data and administrative registers. In total 286,232 members of the Stockholm Youth Cohort were born 1984-1995, and therefore could have turned 12 before the end of follow-up (31 December 2007). Of these, we excluded those who died (N=30) or permanently emigrated from Stockholm county (N=5,538) before their 12th birthday. We further excluded those born outside Sweden (N=30,552) or adopted (N=268). Our study population therefore consisted of 249,884 individuals (49% female, age range 12-23 over the period of follow-up).

Case definition and case ascertainment

Our definition of ED included all specific ED diagnoses identified in the International Classification of Diseases (F50.0-F50.9 in ICD-10, 307.1/307.5 in ICD-9, 306.5 in ICD-8) plus the equivalent group-level DSM-IV codes for 'any ED' (specific diagnostic codes not available for DSM-IV). Our definition of cases also included all those who received treatment at a specialist ED clinic and/or who were recorded by clinicians as having attended psychiatric services because of an ED. We included these last groups because diagnosis data was incomplete in our sources for case ascertainment (see below), but we judged these individuals to be at very high risk of having an ED. As a sensitivity analysis we repeated our analyses using a more restricted definition, which only included those who had a recorded diagnosis for ED or who attended a specialist ED clinic at least three times.

We ascertained ED cases using inpatient and outpatient information from all publically-financed child-, adolescent- and adult psychiatric facilities in Stockholm County. This included all publically- and privately-run facilities, both of which can be used free of charge until age 18 and at a relatively low cost thereafter. We used four sources to ascertain cases:

- 1) The *Stockholm Child and Adolescent Psychiatric Care Register* covers all publically-run child and adolescent psychiatric services in Stockholm County since 2001. It includes diagnostic information according to DSM-IV groupings (i.e. coded at the group level of 'any ED') and clinicians also record the psychiatric disorder which led patients to seek care.
- 2) The *Stockholm Adult Psychiatric Care Register* covers all publically-run adult psychiatric care within Stockholm County since 1997. It includes diagnostic information according to DSM-IV groupings until 2004, and according to ICD-10 since 2005.
- 3) The *Stockholm County Council Health Service Use Register (VAL) database* is an administrative register of all visits to publically-financed health services (all ages) in Stockholm County since 1997. It therefore also covers privately-run facilities. Diagnostic information is recorded according to ICD 9/10 but with incomplete data. In addition to providing this diagnostic information, this database also allowed us to identify individuals who were treated in any of the three specialist ED clinics in Stockholm County (two privately-run, one publically-run).
- 4) The *National Patient Register* (28.) covers all psychiatric inpatient care in publically- and privately-run services, with high coverage since 1973 and complete

coverage from 1987. It includes information on discharge diagnoses coded according to ICD-7 to ICD-10.

Of 3251 children identified as an ED case from any source, 2524 were identified in the *Stockholm Child and Adolescent Psychiatric Care Register* (1507 with a recorded ED diagnosis, and 1017 with ED as the reason for seeking care); 938 in the *Stockholm Adult Psychiatric Care Register*; 2232 in the *VAL database* (288 with a diagnosis, 1944 in a specialist ED clinic); and 431 in the *National Patient Register*. Numbers sum up to more than 3251 because many children were identified from multiple sources.

Exposures and other covariates

We identified biological parents, siblings and half-siblings, and their dates of birth and personal identity numbers (29.) using the Swedish Multi-Generation Register (30.). From these data we calculated parental age at the child's birth and the number, gender and age (relative to the index child) of siblings and half-siblings. We used the Longitudinal Integration Database for Health Insurance and Labour Market Studies (31.) to collect prospectively recorded information on socio-economic variables. In order to capture various aspects of SEP, we included income (measured 2004) and education (measured 2006: see Table 1). Our measure of family income was the individualised disposable family income, which combines all sources of income (including social benefits), deducts all known outgoing expenses (including taxes) and adjusts for family size. We also used the Medical Birth Registry (32.) to identify whether one or both parents were unemployed at the time of the child's birth (binary variable yes/no), and to establish family type at birth. Because the coding of this variable changed over our study period, we could only consistently classify family types as those containing both biological parents versus 'other' family types. We therefore use this binary variable in our analysis but, for comparison, present an alternative sub-categorisation in Table 1 which distinguishes single-parent households where possible.

Data analysis

Hazard ratios were calculated using Cox regression, adjusting for age and study period and stratifying by gender. Follow-up started when the child turned 12 or immigrated into Stockholm County (whichever was later) and continued until 31 December 2007 or until the child died, emigrated or met our criteria to become an ED case (whichever was earliest). We first adjusted only for year of birth ('minimally-adjusted analyses') and then proceeded additionally to adjust for family SEP and parental age ('adjusted analysis 1') and finally for family SEP, parental age and family composition ('adjusted analysis 2'). We modeled the 'number of siblings' variables continuously (all $p > 0.01$ for non-linearity); all other explanatory variables were entered as categorical variables and we report p-values for heterogeneity. In order to quantify the potential health impacts of reported associations, we calculated population attributable fractions for selected variables.

The frequency of missing data for family and social covariates ranged from 0-11.5%. We used multiple imputation by chained equations (five imputations) to impute missing data under an assumption of missing at random. Robust standard errors were used to allow for potential correlations between children born to the same mother. All analyses were conducted using Stata 11.1.

Table 1: Characteristics of study population and crude rates of eating disorders

			Females (N=121,747)			Males (N=128,097)		
			N	D	Rate per 100,000	N	D	Rate per 100,000
TOTAL			121,747	2971	457	128,097	280	40
Family SEP	Mother's highest educational level, 2006	Pre-secondary, ≤8 yrs	3488	34	172	3625	7	33
		Pre-secondary, 9-10 yrs	11,684	231	353	11,783	30	45
		Secondary, ≤2 yrs	35,547	755	386	37,499	93	44
		Secondary, 3 yrs	18,546	411	451	19,631	39	40
		Post-secondary, ≤2 yrs	20,479	561	513	21,565	43	37
		Post-secondary, 3+ yrs	28,008	908	610	29,714	59	37
	Father's highest educational level, 2006	Pre-secondary, ≤8 yrs	3971	71	298	4141	8	32
		Pre-secondary, 9-10 yrs	15,748	332	382	16,470	33	36
		Secondary, ≤2 yrs	34,690	725	402	36,465	75	39
		Secondary, 3 yrs	16,101	394	453	17,280	40	43
		Post-secondary, ≤2 yrs	16,993	421	478	17,995	47	50
		Post-secondary, 3+ yrs	25,544	816	602	26,786	55	38
	Family disposable income, 2004 (quarters)	Quarter 1 (low)	29,785	788	510	30,639	75	47
		Quarter 2	29,505	659	425	30,908	72	44
		Quarter 3	29,357	656	422	31,035	61	37
Quarter 4 (high)		29,136	793	480	31,228	63	34	
Any parent unemployed at child's birth	No	107,540	2759	457	112,903	249	39	
	Yes	14,207	212	457	15,194	31	62	
Parent's Age	Mother's age at child's birth	<20 yrs	2836	73	471	2992	13	77
		20-29 yrs	63,653	1489	433	66,777	136	37
		30-39 yrs	50,542	1297	487	53,455	112	39
		40+ yrs	3099	85	528	3204	15	87
	Father's age at child's birth	<20 yrs	793	14	315	773	4	92
		20-29 yrs	44,351	1025	427	46,759	104	41
		30-39 yrs	60,077	1541	482	63,244	128	37
		40-49 yrs	12,338	296	459	13,127	37	53
		50+ yrs	1220	34	574	1290	5	78
Family composition	Family type at child's birth	Both biological parents	100,261	2455	453	105,233	228	39
		Other	7579	201	484	7962	15	34
	Family type at child's birth (not 1991-1995)	Both biological parents	58,794	1954	450	61,495	147	32
		Single-parent family	2221	81	504	2285	7	42
		Other	2448	82	457	2543	4	21
	Number of full siblings†	0	20,384	560	519	20,934	55	48
		1	57,810	1465	483	61,047	124	38
		2	30,406	710	428	32,802	76	42
		3	8940	183	367	9409	15	28
		4	2577	43	300	2459	7	50
		5+	1630	10	112	1446	3	39
Number of half siblings†	0	82,358	1881	432	87,033	173	37	
	1	17,393	487	518	18,256	44	44	
	2	11,333	326	531	11,968	31	47	
	3	5713	149	475	5764	19	59	
	4	2806	74	464	2843	7	44	
	5+	2144	54	448	2233	6	47	
Twin, triplet or quadruplet	No	118,796	2913	458	125,397	272	40	
	Yes	2951	58	388	2980	8	55	

† Results presented separately by sibling type (older brothers/older sisters/younger brothers/younger sisters) in the supplementary material.

Results

We recorded 2971 cases of ED among females and 280 cases of ED among males during the follow-up period. The overall incidence rate of ED was 457/100,000 person-years in females and 40/100,000 person-years in males. All substantive findings were unchanged in sensitivity analyses, restricting our outcome measure to the 82% of cases who met our more restrictive definition of ED (see Supplementary Tables 1 and 2).

Risk factors for ED in females

Higher maternal and higher paternal education were both associated with an increased rate of ED in daughters, with a clear dose-response relationship across all six educational categories. The association with maternal education appeared somewhat stronger than the association with paternal education, but both associations persisted after mutual adjustment and after adjustment for other parental and family characteristics (Table 2). Given that higher parental education is common in Sweden, these strong effect sizes also translated into a sizeable population impact. For example, assuming the observed associations were fully causal, these results suggest that 24% of all ED cases in females would be averted if all cohort members were born to mothers with only 9-10 years education (population attributable fractions calculated using HRs from adjusted model 2).

We also observed a In contrast to these positive associations with education, the independent effect of income was such that the ED rate was highest in the lowest income quarter (Table 2). Parental unemployment and family type did not show statistically significant associations with rate of ED in daughters and the association between parental age and ED rate was not statistically significant after adjusting for parental education (Table 2).

There was a moderately strong inverse association between increasing numbers of full siblings and rate of ED in females. All types of full siblings (older or younger, sisters or brothers) were associated with a lower rate of ED in females (see supplementary material). By contrast, an increasing number of half-siblings was associated with a higher rate of ED. These divergent effects of full versus half siblings were seen both in minimally-adjusted analyses and in analyses adjusted for other parental and family characteristics (adjusted model 2, Table 2). Assuming these associations were fully causal, 5.6% of ED cases would be averted if all cohort members had two full siblings and 3.3% if all cohort members had zero half siblings.

Table 2: Hazards ratios for eating disorders: females (N=121,747)

		Minimally-adjusted for birthyear	Adjusted 1	Adjusted 2
Mother's highest educational level 2006	Pre-secondary, ≤8 yrs	0.49 (0.34, 0.71)	0.46 (0.30, 0.70)	0.47 (0.30, 0.75)
	Pre-secondary, 9-10 yrs	1*** [***]	1***	1***
	Secondary, ≤2 yrs	1.08 (0.94, 1.26)	1.11 (0.95, 1.30)	1.10 (0.93, 1.29)
	Secondary, 3 yrs	1.24 (1.06, 1.46)	1.28 (1.08, 1.53)	1.31 (1.09, 1.57)
	Post-secondary, ≤2 yrs	1.44 (1.24, 1.68)	1.45 (1.22, 1.71)	1.47 (1.23, 1.76)
	Post-secondary, 3+ yrs	1.72 (1.49, 1.98)	1.67 (1.42, 1.97)	1.69 (1.42, 2.02)
Father's highest educational level 2006	Pre-secondary, ≤8 yrs	0.81 (0.63, 1.05)	0.94 (0.71, 1.23)	1.00 (0.75, 1.32)
	Pre-secondary, 9-10 yrs	1*** [***]	1***	1***
	Secondary, ≤2 yrs	1.04 (0.91, 1.18)	1.01 (0.89, 1.16)	1.02 (0.88, 1.17)
	Secondary, 3 yrs	1.19 (1.03, 1.38)	1.11 (0.95, 1.29)	1.14 (0.98, 1.34)
	Post-secondary, ≤2 yrs	1.24 (1.07, 1.43)	1.10 (0.94, 1.27)	1.14 (0.98, 1.34)
	Post-secondary, 3+ yrs	1.58 (1.39, 1.80)	1.32 (1.14, 1.53)	1.32 (1.13, 1.54)
Family disposable income, 2004	Quarter 1 (low)	1***	1***	1***
	Quarter 2	0.84 (0.76, 0.93)	0.81 (0.72, 0.90)	0.82 (0.73, 0.92)
	Quarter 3	0.83 (0.75, 0.92)	0.76 (0.68, 0.84)	0.76 (0.67, 0.85)
	Quarter 4 (high)	0.95 (0.86, 1.05)	0.74 (0.66, 0.83)	0.77 (0.69, 0.87)
Any parent unemployed at child's birth	No	1	1	1
	Yes	1.02 (0.88, 1.17)	1.01 (0.87, 1.17)	1.00 (0.85, 1.17)
Mother's age at child's birth	<20 yrs	1.10 (0.87, 1.39)	1.21 (0.92, 1.60)	1.17 (0.87, 1.57)
	20-29 yrs	1** [***]	1	1
	30-39 yrs	1.13 (1.05, 1.22)	1.02 (0.93, 1.11)	1.00 (0.91, 1.10)
	40+ yrs	1.22 (0.98, 1.52)	1.16 (0.91, 1.49)	1.16 (0.90, 1.51)
Father's age at child's birth	<20 yrs	0.75 (0.44, 1.28)	0.70 (0.39, 1.27)	0.65 (0.34, 1.24)
	20-29 yrs	1* [**]	1	1
	30-39 yrs	1.13 (1.05, 1.23)	1.00 (0.91, 1.10)	1.01 (0.92, 1.12)
	40-49 yrs	1.07 (0.94, 1.22)	0.92 (0.79, 1.08)	0.89 (0.75, 1.05)
	50+ yrs	1.35 (0.96, 1.90)	0.94 (0.57, 1.56)	0.86 (0.50, 1.48)
Family type at child's birth	Both biological parents	1		1
	Other	1.07 (0.93, 1.24)		1.00 (0.84, 1.18)
Number of full siblings	Change per sibling	0.87 (0.84, 0.90)***		0.92 (0.88, 0.97)***
Number of half siblings	Change per sibling	1.04 (1.01, 1.07)**		1.05 (1.01, 1.09)**
Twin, triplet or quadruplet	No	1		1
	Yes	0.84 (0.65, 1.09)		0.89 (0.67, 1.20)

*p<0.05. **p<0.01. ***p<0.001. P-values for categorical variables are from tests for heterogeneity, with tests for linear trend in brackets for minimally-adjusted analyses. Minimally-adjusted analyses adjust for only for year of birth, adjusted models 1 and 2 additionally include all variables presented in the column

Table 3: Hazards ratios for eating disorders: males (N=128,097)

		Minimally- adjusted for birth year	Adjusted 1	Adjusted 2
Mother's highest educational level 2006	Pre-secondary, ≤8 yrs	0.77 (0.34, 1.75)	0.89 (0.38, 2.07)	0.71 (0.24, 2.12)
	Pre-secondary, 9-10 yrs	1 [n.s.]	1	1
	Secondary, ≤2 yrs	0.97 (0.64, 1.46)	0.97 (0.63, 1.49)	1.07 (0.66, 1.73)
	Secondary, 3 yrs	0.79 (0.49, 1.28)	0.76 (0.46, 1.26)	0.82 (0.47, 1.44)
	Post-secondary, ≤2 yrs	0.79 (0.49, 1.25)	0.72 (0.43, 1.20)	0.83 (0.48, 1.45)
	Post-secondary, 3+ yrs	0.78 (0.50, 1.21)	0.69 (0.42, 1.14)	0.73 (0.42, 1.28)
Father's highest educational level 2006	Pre-secondary, ≤8 yrs	0.98 (0.45, 2.13)	0.97 (0.43, 2.16)	0.87 (0.33, 2.35)
	Pre-secondary, 9-10 yrs	1 [n.s.]	1	1
	Secondary, ≤2 yrs	1.03 (0.68, 1.55)	1.12 (0.73, 1.71)	1.40 (0.87, 2.25)
	Secondary, 3 yrs	1.17 (0.74, 1.86)	1.40 (0.87, 2.27)	1.60 (0.94, 2.75)
	Post-secondary, ≤2 yrs	1.32 (0.84, 2.05)	1.65 (1.03, 2.65)	1.86 (1.09, 3.18)
	Post-secondary, 3+ yrs	1.04 (0.68, 1.60)	1.40 (0.85, 2.28)	1.70 (0.98, 2.93)
Family disposable income, 2004	Quarter 1 (low)	1	1	1
	Quarter 2	0.94 (0.68, 1.30)	0.91 (0.65, 1.28)	1.05 (0.72, 1.53)
	Quarter 3	0.79 (0.56, 1.10)	0.79 (0.56, 1.12)	0.93 (0.63, 1.37)
	Quarter 4 (high)	0.81 (0.58, 1.13)	0.79 (0.54, 1.14)	0.87 (0.57, 1.32)
Any parent unemployed at child's birth	No	1	1	1
	Yes	1.22 (0.83, 1.79)	1.22 (0.82, 1.82)	1.13 (0.72, 1.77)
Mother's age at child's birth	<20 yrs	2.18 (1.23, 3.85)	1.88 (0.95, 3.69)	2.07 (1.00, 4.26)
	20-29 yrs	1**	1	1
	30-39 yrs	1.05 (0.81, 1.34)	1.00 (0.74, 1.36)	0.99 (0.71, 1.38)
	40+ yrs	2.35 (1.38, 4.00)	1.87 (0.97, 3.64)	1.38 (0.63, 3.04)
Father's age at child's birth	<20 yrs	2.39 (0.88, 6.49)	1.34 (0.39, 4.62)	0.51 (0.07, 3.92)
	20-29 yrs	1 [n.s.]	1	1
	30-39 yrs	0.92 (0.71, 1.20)	1.01 (0.74, 1.39)	0.96 (0.68, 1.34)
	40-49 yrs	1.28 (0.88, 1.86)	1.20 (0.75, 1.94)	1.32 (0.80, 2.19)
	50+ yrs	1.89 (0.77, 4.64)	2.23 (0.78, 6.38)	1.40 (0.33, 5.97)
Family type at child's birth	Both biological parents	1		1
	Other	0.88 (0.52, 1.48)		0.82 (0.45, 1.47)
Number of full siblings	Change per sibling	0.95 (0.84, 1.07)		1.01 (0.88, 1.17)
Number of half siblings	Change per sibling	1.10 (1.01, 1.20)*		1.08 (0.96, 1.21)
Twin, triplet or quadruplet	No	1		1
	Yes	1.30 (0.64, 2.63)		1.60 (0.75, 3.41)

n.s.=non-significant. *p<0.05. **p<0.01. ***p<0.001. P-values for categorical variables are from tests for heterogeneity, with tests for linear trend in brackets for minimally-adjusted analyses. Minimally-adjusted analyses adjust for only for year of birth, adjusted models 1 and 2 additionally include all variables presented in the column

Risk factors for ED in males and gender differences in risk factors

There was strong evidence of an interaction between gender and maternal education ($p < 0.001$) and weak evidence of an interaction between gender and paternal education ($p = 0.04$). These interactions reflected the fact that although higher parental education was a strong risk factor for ED in females, it showed no association with ED in males (Table 3).

Otherwise there was little evidence of interactions between gender and non-socio-economic risk factors (see supplementary material). The magnitude of the point estimate for the association between number of half siblings and rate of ED in males was similar to that observed in females ($p = 0.33$ for gender interaction), but the association did not reach statistical significance in adjusted analyses. Associations of twin/triplet status and number of full siblings with rate of ED in males were likewise statistically non-significant (Table 3) and showed little consistency with patterns of associations observed among females. However, these variables also showed no evidence of an interaction by gender ($p > 0.19$). This suggests these data are underpowered to examine how far males differ from females in the effect of sibling size upon ED rate, and are also somewhat underpowered generally to examine predictors of ED in males.

Discussion

In this population-based cohort study of Swedish-born adolescents living in Stockholm County, we found maternal and paternal education to be strongly related to ED rate in females but not in males. Furthermore, increasing number of full siblings was associated with a lower rate of ED in females, while increasing number of half-siblings was associated with an increased rate.

With respect to the associations between parental education and rate of ED in females, our results replicate and extend the findings from two previous, smaller Swedish cohorts (11; 12.). This association is intriguing since it represents a reversal of the social gradient observed for most child health outcomes (33.). Given the large and growing fraction of Swedish parents who are highly educated, we demonstrate that this association may have a substantial impact upon population-level ED (e.g. an overall population attributable fraction of 24% for maternal education). Future research into mechanisms underlying this association is therefore warranted. It has been suggested that high demands (both external and internal), high parental expectations and parental control play a role in the development of ED (9; 34-36.). Education is one area where children may be particularly subject to parental expectations and demands, and this may be especially true for the children of highly-educated parents. In Sweden, as in many other settings, higher parental education has been shown to predict better grades among offspring (37; 38.). This relationship is likely to reflect several underlying factors, but one plausible factor is an expectation among highly educated parents that their children should also do well academically. This may include not only explicit wishes from parents, but also indirect demands that may cause daughters in well-educated families to feel overwhelmed by a pressure to live up to expectations.

Why then, are the same effects not seen in males? One possibility is that the gender difference reflects, at least in part, differences in the composition of ED diagnoses between males and females. A related explanation is suggested by the authors of one previous paper reporting on gender differences in SEP associations; they speculate that males show a different association because their ED symptoms are more likely to reflect global

psychological impairments (10.). There is also some evidence that sons are less likely than daughters to be the target of parental weight concerns (39.). If so, this might also render males less vulnerable to the cultural values and orientations of high SEP families (e.g. valuing healthy food, self-restraint and high achievement) which have been argued to form a backdrop for many female cases of ED (40.). Addressing these and other related questions may suggest important targets for public health interventions, as well as perhaps shedding light onto the aetiology of ED in both males and females.

Parental demands are also a plausible candidate explanation for our finding of a protective effect of having larger number of siblings. Previous studies have shown that having a large number of siblings is associated with poorer outcomes across a range of educational and health measures (38.). This has typically been hypothesised to reflect a ‘dilution’ effect whereby parents with many children have less time and resources to devote to each child. Nevertheless, it is possible that such a dilution might be beneficial with respect to ED if it softens the magnitude of the parental demands and expectations placed on each child. Indirect support for this hypothesis comes from our observation that the protective effect of having more siblings was seen regardless of the type of sibling (older or younger, brothers or sisters). This perhaps suggests that the effect is mediated by total family size rather than by the ways in which different types of siblings interact with each other.

This paper also makes the novel observation that although having full siblings seemed to have a protective effect, having half-siblings represented a risk factor for ED in females. In seeking to understand this finding, it may be useful to consider research into environmental characteristics predicting ED. Klump *et al.* (41.) studied non-shared environmental factors in individuals with disordered eating. The results indicated that differential family relationships partly explained differences in eating pathology in one sibling versus another. The adverse effect of half siblings observed in the present study could be a marker for former adverse life events (e.g. parental divorce) or on-going family tensions (e.g. following the introduction of new siblings). The inclusion of half-siblings in the family is also frequently associated with the presence of a step-parent, who may sometimes have more contact or higher-quality contact with their biological children than with their step-children. Further quantitative and qualitative research is needed to confirm this association with number of half-siblings and, if replicated, to investigate the possible underlying mechanisms.

Strengths and limitations

One of the key strengths of this study is its comparatively large sample of both males and females, and its use of data collected prospectively from a well-defined population. Another strength of our study is that it identifies ED using an unusually wide range of healthcare registers, and is therefore likely to have identified a more representative group of ED patients than previous studies which used only hospitalisation data (11-13.). Nevertheless, although an improvement upon previous studies, our use of register data is an important limitation. Firstly, like all register-based studies, we were not able to standardise the process of making ED diagnoses. Instead we were reliant upon ‘real-world’ diagnoses made by multiple clinicians in multiple clinics and using both ICD and DSM criteria. Our findings are, however, consistent with a previous Swedish study, which used only ICD diagnoses and which addressed some similar research questions (e.g. regarding the association between parent SEP and ED risk in daughters)(11.).

Secondly, although medical care in Sweden is available free of charge or at relatively low cost, previous research has demonstrated that individuals from socio-economically disadvantaged groups are less likely to seek healthcare, or else seek help at a later stage (42.). Yet although this raises the possibility that some of our findings could reflect differential help-seeking behaviour, we believe that the gender-specificity of the association between parental education and ED provides some evidence against this interpretation. Over-ascertainment of cases in higher socio-economic groups would also be expected to affect the associations with others indicators of SEP (e.g. income and employment), but those did not show the same association with ED as education.

A third limitation of our study is that it reports on ED as an entire group, because we lacked full information on specific diagnoses. We believe that analyses based upon combining ED into a single group are still of value, particularly given the on-going debate as to the value of different subtypes (43; 44.), and given the extent to which patients fluctuate between diagnoses (45.). Nevertheless we recognise that it would be informative in future studies to distinguish between different types of ED, in order to examine how far this may explain the gender differences observed with respect to SEP (10; 46.). Finally, although comparatively large, this study lacked power to examine associations with rare exposures (e.g. twin status) and to examine associations in males. It would therefore also be valuable to replicate and extend this work using still larger samples, for example by using national rather than regional total-population data.

Conclusion

In a review of risk factors for disordered eating, Jacobi *et al.* (47.) concluded that interventions ought to be aimed at women with high weight and shape concerns, a history of critical comments about eating, weight and shape, and a history of depression. We accept that these may be the strongest proximate correlates of ED, and therefore the most important targets for individual-level or clinical interventions. Nevertheless we believe that the observed associations with parental education and family type may also be relevant for the design of broader public health preventive measures. Specifically, we believe that by understanding the mechanisms underlying these associations and, crucially, by understanding why only some children are adversely affected (daughters not sons, half siblings not full siblings) it may ultimately be possible to design interventions that seek to prevent ED and their symptoms at the population level.

Conflict of interest

None

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References

1. Kosidou K, Magnusson C, Mittendorfer-Rutz E, Hallqvist J, Hellner Gumpert C, Idrizbegovic S, et al. Recent time trends in levels of self-reported anxiety, mental health service use and suicidal behaviour in Stockholm. *Acta Psychiatr Scand* 2010;122:47-55.
2. Klump KL, Bulik CM, Kaye WH, Treasure J, Tyson E. Academy for eating disorders position paper: eating disorders are serious mental illnesses. *Int J Eat Disord* 2009;42:97-103.
3. Muike AM, Stein DG, Arbess G. Eating disorders in adolescent boys: a review of the adolescent and young adult literature. *J Adolesc Health* 2003;33:427-435.
4. Dominé F, Berchtold A, Akre C, Michaud P-A, Suris J-C. Disordered Eating Behaviors: What About Boys? *J Adolesc Health* 2009;44:111-117.
5. Hudson JI, Hiripi E, Pope HG, Jr., Kessler RC. The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. *Biol Psychiatry* 2007;61:348-358.
6. Nunez-Navarro A, Aguera Z, Krug I, Jimenez-Murcia S, Sanchez I, Araguz N, et al. Do men with eating disorders differ from women in clinics, psychopathology and personality? *Eur Eat Disord Rev* 2012;20:23-31.
7. Jacobi C, Hayward C, de Zwaan M, Kraemer HC, Agras WS. Coming to terms with risk factors for eating disorders: application of risk terminology and suggestions for a general taxonomy. *Psychol Bull* 2004;130:19-65.
8. Kinzl JF, Mangweth B, Traweger CM, Biebl W. Eating-disordered behavior in males: The impact of adverse childhood experiences. *Int J Eat Disord* 1997;22:131-138.
9. Rome ES. Eating disorders in children and adolescents. *Curr Probl Pediatr Adolesc Health Care* 2012;42:28-44.
10. Costa C, Ramos E, Severo M, Barros H, Lopes C. Determinants of eating disorders symptomatology in Portuguese adolescents. *Arch Pediatr Adolesc Med* 2008;162:1126-1132.
11. Ahrén-Moonga J, Silverwood R, Klinteberg BA, Koupil I. Association of higher parental and grandparental education and higher school grades with risk of hospitalization for eating disorders in females. *Am J Epidemiol* 2009;170:566-575.
12. Ahren JC, Chiesa F, Af Klinteberg B, Koupil I. Psychosocial determinants and family background in anorexia nervosa--results from the Stockholm Birth Cohort Study. *Int J Eat Disord* 2012;45:362-369.
13. Lindberg L, Hjern A. Risk factors for anorexia nervosa: a national cohort study. *Int J Eat Disord* 2003;34:397-408.
14. McClelland L, Crisp A. Anorexia nervosa and social class. *Int J Eat Disord* 2001;29:150-156.
15. Gibbons P. The Relationship Between Eating Disorders and Socioeconomic Status: It's Not What You Think. *Nutr Noteworthy* 2001;4.
16. Swanson S, Crow, SJ, Le Grange D, Swendsen J, Merikangas, KR. Prevalence and correlates of eating disorders in adolescents: Results from the national comorbidity survey replication adolescent supplement. *Arch Gen Psychiatry* 2011;68:714-723.
17. Frans EM, Sandin S, Reichenberg A, Lichtenstein P, Langstrom N, Hultman CM. Advancing paternal age and bipolar disorder. *Arch Gen Psychiatry* 2008;65:1034-1040.
18. Hultman CM, Sandin S, Levine SZ, Lichtenstein P, Reichenberg A. Advancing paternal age and risk of autism: new evidence from a population-based study and a meta-analysis of epidemiological studies. *Mol Psychiatry* 2011;16:1203-1212.

19. Sandin S, Hultman CM, Kolevzon A, Gross R, MacCabe JH, Reichenberg A. Advancing Maternal Age Is Associated With Increasing Risk for Autism: A Review and Meta-Analysis. *J Am Acad Child Adolesc Psychiatry* 2012;51:477-486.
20. Dalman C, Allebeck P. Paternal age and schizophrenia: further support for an association. *Am J Psychiat* 2002;159:1591-1592.
21. Halmi KA. Anorexia nervosa: demographic and clinical features in 94 cases. *Psychosom Med* 1974;36:18-26.
22. Dolan BM, Evans C, Lacey JH. Family composition and social class in bulimia - A catchment area study of a clinical and a comparison group. *J Nerv Ment Dis* 1989;177:267-272.
23. Boumann CE, Yates WR. Risk factors for bulimia nervosa: A controlled study of parental psychiatric illness and divorce. *Addictive Behaviors* 1994;19:667-675.
24. Cnattingius S, Hultman CM, Dahl M, Sparen P. Very preterm birth, birth trauma, and the risk of anorexia nervosa among girls. *Arch Gen Psychiat* 1999;56:634-638.
25. Vandereycken W, Van Vreckem E. Siblings of patients with an eating disorder. *Int J Eat Disord* 1992;12:273-280.
26. Britto DJ, Meyers DH, Smith JJ, Palmer RL. Anorexia nervosa and bulimia nervosa: sibling sex ratio and birth rank - a catchment area study. *Int J Eat Disord* 1997;21:335-340.
27. Eagles JM, Johnston MI, Millar HR. A case-control study of family composition in anorexia nervosa. *Int J Eat Disord* 2005;38:49-54.
28. Swedish National Board of Health and Welfare. In-patient diseases in Sweden 1987-2006. Stockholm, 2007.
29. Ludvigsson JF, Otterblad-Olausson P, Pettersson BU, Ekblom A. The Swedish personal identity number: possibilities and pitfalls in healthcare and medical research. *Eur J Epidemiol* 2009;24:659-667.
30. Ekblom A. The Swedish Multi-generation Register. *Methods Mol Biol* 2011;675:215-220.
31. Statistics Sweden. Longitudinal Integration Database for Health Insurance and Labour Market Studies 2009.
32. Axelsson O. The Swedish Medical Birth Register. *Acta Obstetrica et Gynecologica Scandinavica* 2003;82:491-492.
33. World Health Organisation. Closing the gap in a generation; health equity through action on the social determinants of health 2008.
34. Fairburn CG, Cooper Z, Doll HA, Welch SL. Risk factors for anorexia nervosa: three integrated case-control comparisons. *Arch Gen Psychiatry* 1999;56:468-476.
35. Wade TD, Gillespie N, Martin NG. A comparison of early family life events amongst monozygotic twin women with lifetime anorexia nervosa, bulimia nervosa, or major depression. *Int J Eat Disord* 2007;40:679-686.
36. Jauregui Lobera I, Bolanos Rios P, Garrido Casals O. Parenting styles and eating disorders. *J Psychiatr Mental Health Nurs* 2011;18:728-735.
37. Swedish National Board of Education. Summary of report 363. Facts and figures, 2011.
38. Goodman A, M.D. Gisselmann, and I. Koupil, . Birth outcomes and early-life social characteristics predict unequal educational outcomes: consistency across Swedish cohorts born 1915-1929 and 1973-1980. *Longitudinal and Life Course Studies* 2010;1:315-316.
39. Agras S, Hammer L, McNicholas F. A prospective study of the influence of eating-disordered mothers on their children. *Int J Eat Disord* 1999;25:253-262.
40. Darmon. The fifth element: Social class and the sociology of anorexia. *Sociology* 2009;43:717-733.

41. Klump KL, Wonderlich S, Lehoux P, Lilienfeld LR, Bulik CM. Does environment matter? A review of nonshared environment and eating disorders. *Int J Eat Disord* 2002;31:118-135.
42. Ahs AM, Westerling R. Health care utilization among persons who are unemployed or outside the labour force. *Health Policy* 2006;78:178-193.
43. Walsh BT, Sysko R. Broad categories for the diagnosis of eating disorders (BCD-ED): an alternative system for classification. *Int J Eat Disord* 2009;42:754-764.
44. Fairburn CG, Cooper Z. Eating disorders, DSM-5 and clinical reality. *Br J Psychiatry* 2011;198:8-10.
45. Ackard DM, Fulkerson JA, Neumark-Sztainer D. Stability of eating disorder diagnostic classifications in adolescents: five-year longitudinal findings from a population-based study. *Eat Disord* 2011;19:308-322.
46. Striegel-Moore RH, Bulik CM. Risk factors for eating disorders. *Am Psychol* 2007;62:181-198.
47. Jacobi C, Fittig E, Bryson SW, Wilfley D, Kraemer HC, Taylor CB. Who is really at risk? Identifying risk factors for subthreshold and full syndrome eating disorders in a high-risk sample. *Psychol Med* 2011;41:1939-1949.

Appendix**Appendix, Supplementary Table 1: Sensitivity analyses repeated using our restricted case definition (recorded ED diagnosis or at least 3 visits to a specialist ED clinic)**

			Rate per 100000			
			Females		Males	
			Broad definition ED†	Restricted definition ED	Broad definition ED	Restricted definition ED
TOTAL			457	381	40	28
Family SEP	Mother's highest educational level, 2006	Pre-secondary, ≤8 yrs	172	131	33	14
		Pre-secondary, 9-10 yrs	353	283	45	39
		Secondary, ≤2 yrs	386	301	44	31
		Secondary, 3 yrs	451	378	40	24
		Post-secondary, ≤2 yrs	513	441	37	28
		Post-secondary, 3+ yrs	610	534	37	24
	Father's highest educational level, 2006	Pre-secondary, ≤8 yrs	298	227	32	24
		Pre-secondary, 9-10 yrs	382	298	36	25
		Secondary, ≤2 yrs	402	328	39	24
		Secondary, 3 yrs	453	394	43	34
		Post-secondary, ≤2 yrs	478	395	50	37
		Post-secondary, 3+ yrs	602	524	38	26
	Family disposable income, 2004 (quarters)	Quarter 1 (low)	510	419	47	31
		Quarter 2	425	343	44	29
		Quarter 3	422	356	37	26
Quarter 4 (high)		480	414	34	26	
Any parent unemployed at child's birth	No	457	382	39	27	
	Yes	457	369	62	34	
Parent's age	Mother's age at child's birth	<20 yrs	471	335	77	53
		20-29 yrs	433	359	37	27
		30-39 yrs	487	415	39	26
		40+ yrs	528	404	87	64
	Father's age at child's birth	<20 yrs	315	225	92	46
		20-29 yrs	427	346	41	29
		30-39 yrs	482	410	37	26
		40-49 yrs	459	377	53	37
		50+ yrs	574	523	78	31
		Family composition	Family type at child's birth	Both biological parents	453	380
Other	484			385	34	20
Family type at child's birth (not 1991-1995)	Both biological parents		450	388	32	23
	Single-parent family		504	405	42	36
	Other		457	379	21	10
Number of full siblings†	0		519	427	48	30
	1		483	405	38	26
	2		428	359	42	33
	3		367	295	28	19
	4		300	251	50	36
	5+		112	112	39	13
Number of half siblings†	0		432	365	37	26
	1		518	430	44	30
	2		531	425	47	33
	3	475	392	59	34	
	4	464	364	44	25	
	5+	448	357	47	47	
Twin, triplet or quadruplet	No	458	382	40	28	
	Yes	388	328	55	41	

†The 'broad' definition of ED includes all those who received a diagnosis of ED or attended a specialist ED clinic, the 'restricted' definition is limited to those who received a diagnosis of ED or attended a specialist ED clinic at least 3 times.

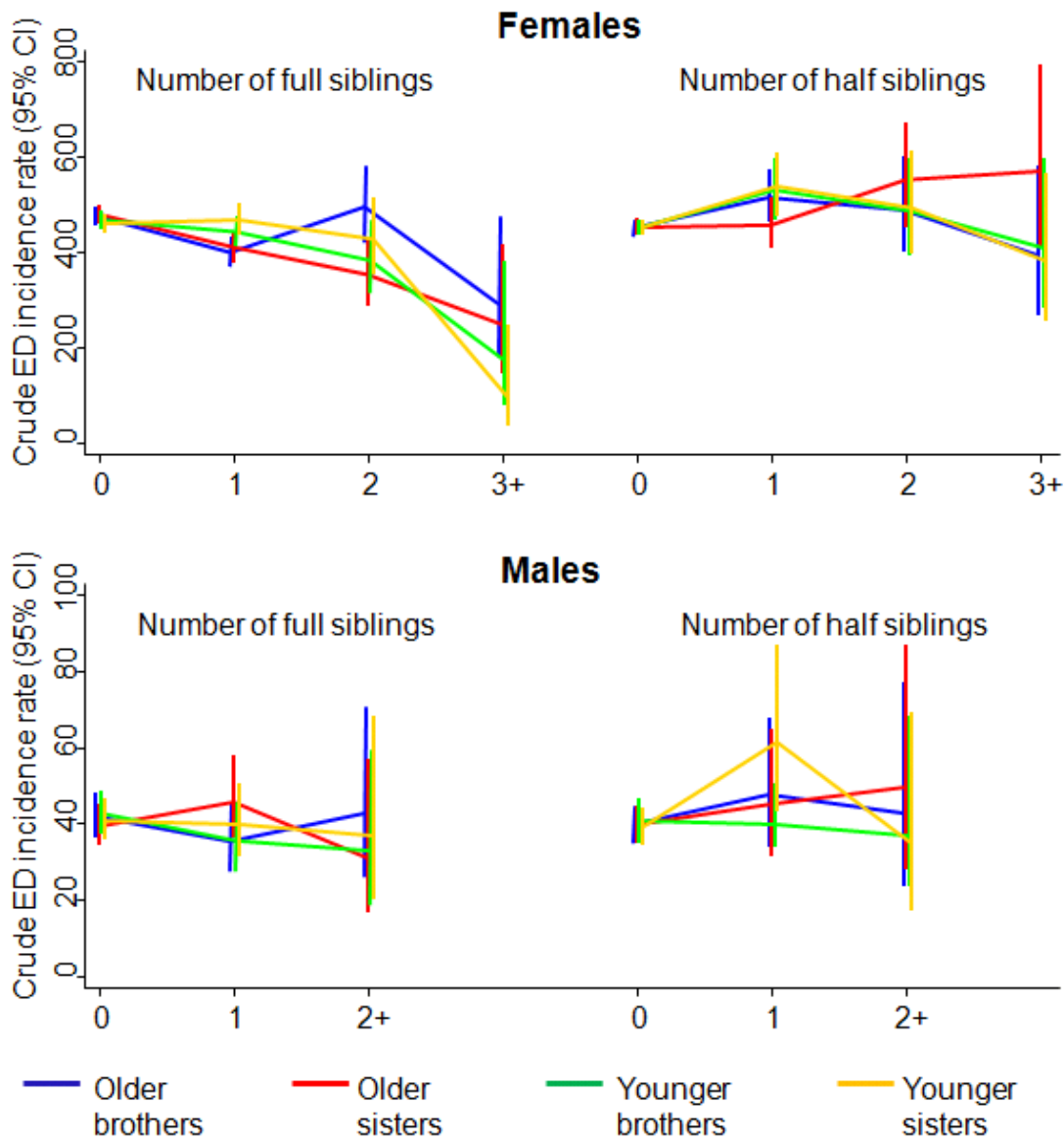
Appendix, Supplementary Table 2: Sensitivity analyses presenting adjusted hazards ratios for ED using restricted case definition (recorded ED diagnosis or at least 3 visits to a specialist ED clinic)

		Females		Males	
		Broad definition ED†	Restricted definition ED	Broad definition ED	Restricted definition ED
Mother's highest educational level 2006	Pre-secondary, ≤8 yrs	0.47 (0.30, 0.75)	0.49 (0.30, 0.81)	0.71 (0.24, 2.12)	0.21 (0.03, 1.59)
	Pre-secondary, 9-10 yrs	1***	1***	1	1
	Secondary, ≤2 yrs	1.10 (0.93, 1.29)	1.01 (0.84, 1.21)	1.07 (0.66, 1.73)	0.88 (0.52, 1.49)
	Secondary, 3 yrs	1.31 (1.09, 1.57)	1.29 (1.06, 1.58)	0.82 (0.47, 1.44)	0.49 (0.25, 0.97)
	Post-secondary, ≤2 yrs	1.47 (1.23, 1.76)	1.45 (1.20, 1.76)	0.83 (0.48, 1.45)	0.67 (0.36, 1.25)
	Post-secondary, 3+ yrs	1.69 (1.42, 2.02)	1.71 (1.41, 2.06)	0.73 (0.42, 1.28)	0.51 (0.27, 0.97)
Father's highest educational level 2006	Pre-secondary, ≤8 yrs	1.00 (0.75, 1.32)	0.93 (0.67, 1.29)	0.87 (0.33, 2.35)	0.94 (0.27, 3.30)
	Pre-secondary, 9-10 yrs	1***	1***	1	1
	Secondary, ≤2 yrs	1.02 (0.88, 1.17)	1.05 (0.90, 1.23)	1.40 (0.87, 2.25)	1.38 (0.76, 2.48)
	Secondary, 3 yrs	1.14 (0.98, 1.34)	1.21 (1.02, 1.44)	1.60 (0.94, 2.75)	2.15 (1.13, 4.07)
	Post-secondary, ≤2 yrs	1.14 (0.98, 1.34)	1.15 (0.96, 1.37)	1.86 (1.09, 3.18)	2.33 (1.22, 4.42)
	Post-secondary, 3+ yrs	1.32 (1.13, 1.54)	1.37 (1.16, 1.62)	1.70 (0.98, 2.93)	1.98 (1.01, 3.86)
Family disposable income. 2004	Quarter 1 (low)	1***	1***	1	1
	Quarter 2	0.82 (0.73, 0.92)	0.78 (0.69, 0.89)	1.05 (0.72, 1.53)	0.99 (0.63, 1.58)
	Quarter 3	0.76 (0.67, 0.85)	0.75 (0.66, 0.86)	0.93 (0.63, 1.37)	0.98 (0.62, 1.56)
	Quarter 4 (high)	0.77 (0.69, 0.87)	0.77 (0.68, 0.87)	0.87 (0.57, 1.32)	0.95 (0.58, 1.55)
Any parent unemployed at child's at birth	No	1	1	1	1
	Yes	1.00 (0.85, 1.17)	0.96 (0.80, 1.15)	1.13 (0.72, 1.77)	0.85 (0.47, 1.57)
Mother's age at child's birth	<20 yrs	1.17 (0.87, 1.57)	1.04 (0.74, 1.47)	2.07 (1.00, 4.26)	2.05 (0.87, 4.83)
	20-29 yrs	1	1	1	1
	30-39 yrs	1.00 (0.91, 1.10)	1.01 (0.91, 1.12)	0.99 (0.71, 1.38)	0.85 (0.57, 1.27)
	40+ yrs	1.16 (0.90, 1.51)	1.10 (0.82, 1.48)	1.38 (0.63, 3.04)	1.71 (0.72, 4.05)
Father's age at child's birth	<20 yrs	0.65 (0.34, 1.24)	0.79 (0.40, 1.58)	0.51 (0.07, 3.92)	[omitted]‡
	20-29 yrs	1	1	1	1
	30-39 yrs	1.01 (0.92, 1.12)	1.04 (0.93, 1.16)	0.96 (0.68, 1.34)	0.92 (0.62, 1.37)
	40-49 yrs	0.89 (0.75, 1.05)	0.88 (0.73, 1.05)	1.32 (0.80, 2.19)	1.30 (0.71, 2.38)
	50+ yrs	0.86 (0.50, 1.48)	0.85 (0.46, 1.57)	1.40 (0.33, 5.97)	[omitted]‡
Family type at child's birth	Both biological parents	1	1	1	1
	Other	1.00 (0.84, 1.18)	0.96 (0.79, 1.16)	0.82 (0.45, 1.47)	0.75 (0.36, 1.56)
Number of full siblings	Change per sibling	0.92 (0.88, 0.97)***	0.92 (0.87, 0.97)**	1.01 (0.88, 1.17)	1.06 (0.90, 1.25)
Number of half siblings	Change per sibling	1.05 (1.01, 1.09)**	1.04 (1.00, 1.08)*	1.08 (0.96, 1.21)	1.11 (0.97, 1.27)
Twin, triplet or quadruplet	No	1	1	1	1
	Yes	0.89 (0.67, 1.20)	0.90 (0.66, 1.24)	1.60 (0.75, 3.41)	1.62 (0.66, 3.98)

*p<0.05. **p<0.01. ***p<0.001. All p-values from tests for heterogeneity. Minimally-adjusted analyses adjust for only for year of birth, adjusted models 1 and 2 additionally include all variables presented in the column.

†The 'broad' definition of ED includes all those who received a diagnosis of ED or attended a specialist ED clinic, the 'restricted' definition is limited to those who received a diagnosis of ED or attended a specialist ED clinic at least 3 times. ‡Cells omitted as based on only 2 cases each, resulting in unstable effect estimates

Appendix, Supplementary Figure 1: Crude rates of eating disorders by number of full and half siblings of different ages and genders.



Appendix, Supplementary Table 3: Interactions between risk factors and gender in predicting ED

		P for gender interaction	
		Adjusted For birth year	Adjusted for birth year + interaction with mother education
Family SEP	Mother's highest educational level, 2006	<0.001 (<0.001)	
	Father's highest educational level, 2006	0.10 (0.04)	0.45
	Fam. disposable income, 2004 (quarters)	0.61 (0.15)	
	Any parent unemployed at child's birth	0.35	
Parent's age	Mother's age at child's birth	0.007 (0.14)	0.08
	Father's age at child's birth	0.15 (0.64)	
Family composition	Family type at child's birth	0.37	
	Family type at child's birth (not 1991-1995)	0.36	
	Number of full siblings	0.25 (0.15)	
	Number of half siblings	0.78 (0.24)	
	Twin, triplet or quadruplet	0.20	

We tested all variables shown in Supplementary Table 3 for interaction with gender entering variables as categorical variables and, for the education, income, parent age and siblings variables, repeating the tests entering them as continuous variables. In minimally-adjusted analyses, the strongest evidence for an interaction with gender was for mother's education ($p < 0.001$ for gender interaction regardless of whether mother's highest education entered as a linear or a categorical term). In minimally-adjusted analyses there was also some evidence of an interaction with father's highest education entered as a linear term ($p = 0.04$) and mother's age as a categorical term ($p = 0.006$). Since both of these variables were strongly associated with mother's education, we additionally entered the interaction between mother's education and gender into the model. This caused both interactions to become non-significant.