

# Association of Higher Parental and Grandparental Education and Higher School Grades With Risk of Hospitalization for Eating Disorders in Females: The Uppsala Birth Cohort Multigenerational Study

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## Abstract

Eating disorders are a leading cause of disease burden among young women. This study investigated associations of social characteristics of parents and grandparents, sibling position, and school performance with incidence of eating disorders. The authors studied Swedish females born in 1952–1989 ( $n = 13,376$ ), third-generation descendants of a cohort born in Uppsala in 1915–1929. Data on grandparental and parental social characteristics, sibling position, school grades, hospitalizations, emigrations, and deaths were obtained by register linkages. Associations with incidence of hospitalization for eating disorders were studied with multivariable Cox regression, adjusted for age and study period. Overall incidence of hospitalization for eating disorders was 32.0/100,000 person-years. Women with more highly educated parents and maternal grandparents were at higher risk (hazard ratio for maternal grandmother with higher education relative to elementary education = 6.5, 95% confidence interval: 2.2, 19.3, adjusted for parental education). Independent of family social characteristics, women with the highest school grades had a higher risk of eating disorders (hazard ratio = 7.7, 95% confidence interval: 2.5, 24.1 for high compared with low grades in Swedish, adjusted for parental education). Thus, higher parental and grandparental education and higher school grades may increase risk of hospitalization for eating disorders in female offspring, possibly because of high internal and external demands.

anorexia nervosa; eating disorders; education; family; parents; siblings; social class

Abbreviations: ICD, *International Classification of Diseases*; USCos, Uppsala Birth Cohort.

## INTRODUCTION

There are concerns about increasing rates of mental illness such as eating disorders, self-inflicted injuries, and self-reported stress among young women in Sweden (1) and other industrialized parts of the world (2, 3). According to the *Diagnostic and Statistical Manual of Mental Disorders: DSM-IV* (4), eating disorders are divided into the main diagnoses of anorexia nervosa, bulimia nervosa, eating disorder not otherwise specified, and binge eating disorder. Together, these disorders constitute one of the leading causes of disease burden in terms of years of life lost through death or disability for young women (5), with a peak age at onset occurring at a developmentally sensitive time in the mid-teens. Respective lifetime prevalence rates for full and partial anorexia nervosa in women range from 0.9% to 4.3% (6–8) and from 1.5 % to 7% for full and partial bulimia nervosa (6, 9). Studies of time trends in eating disorders show that the incidence and prevalence of anorexia nervosa and bulimia nervosa are stabilizing in Western countries (10), whereas the prevalence of eating disorder not otherwise specified and of binge eating disorder continue to increase (11, 12).

Eating disorders are usually explained by multifactorial models including psychological, social, and biologic risk factors. However, little research has attempted to bring together information about various types of risk factors and explore interactions among parental health and social characteristics, early-life environment, and individual aspirations in influencing the risk and long-term outcome of eating disorders.

Common risk factors for anorexia nervosa and bulimia nervosa are gender, ethnicity, family history of eating disorders or depression, elevated weight and shape concerns, negative self-evaluation, abuse, and general psychiatric morbidity (13, 14). Additional risk factors for anorexia nervosa include childhood sleeping problems, overanxious parenting, obsessive-compulsive personality traits, perfectionism, family discord, high parental demands, and high levels of physical exercise (13, 15). Evolutionary biologists (16–18) have argued that people restrict their eating as a result of comparing themselves with those who are successful and in response to fears of loss of status. Studies in humans support a role for social rank in eating pathology (19), and results from twin studies confirm the role of genetic factors in the development of eating disorders (20).

Evidence that the conditions in which children live have long-term effects on their health is well established (21) and lends support to policies to reduce child disadvantage as part of strategies to address health inequalities. However, earlier studies on family social circumstances and eating disorders in daughters have not provided conclusive results: a review of eating disorders and socioeconomic position concluded that eating disorders were not restricted to certain social groups (22), whereas a cohort study from Sweden showed a higher prevalence of anorexia nervosa in higher socioeconomic groups. Results from the latter study also showed that children with an earlier foster-home placement or children of parents who had been treated for psychiatric disorders were at increased risk of developing eating disorders (23). Moreover, families with a higher prevalence of deaths of a first-degree relative and with a higher prevalence of depression in mothers were overrepresented among anorexia nervosa cases (24).

Family structure and sibling position have also been studied as potential risk factors for eating

disorders. Eating disorders have been shown to be more common in families with poor structure and multiple conflicts (25). No clear associations between birth order or sex of siblings and eating disorders were found in a study by Gowers et al. (26), whereas higher birth order and fewer brothers were associated with anorexia nervosa in a study by Eagles et al. (27). A qualitative study of women recovering from anorexia nervosa found that the relationship between patients and siblings tended to be characterized by antagonism, rivalry, and little warmth (28). Canetti et al. (29) reported that anorectic patients, compared with healthy controls, perceived their parents as less caring and their fathers as more controlling.

While changes in cognitive ability and neuropsychological functioning in patients with chronic, severe eating disorders have been documented (30, 31), much less is known about school performance and the incidence of eating disorders. Earlier research on cognitive functioning and eating disorders mostly focused on IQ, suggesting that patients with eating disorders have IQ scores within or slightly higher than the normal range (32, 33). Furthermore, Dura and Bornstein (34) found that school achievement, as measured by reading, spelling, and arithmetic, of patients with anorexia nervosa was much better than predicted by their IQ scores.

We hypothesized that high and low school achievement may be common among females at risk of eating disorders and may partly reflect the role of family educational background in the etiology of eating disorders. The aim of our study was to investigate social and family background factors and school performance in relation to incidence of hospitalization for eating disorders among females in a large, multigenerational Swedish cohort.

## **MATERIALS AND METHODS**

### **Study participants and register linkage**

We studied associations of family social characteristics and school performance with incidence of hospitalization for eating disorders among Swedish females, third-generation descendants of the Uppsala Birth Cohort (UBCoS). The original UBCoS consists of 14,193 males and females born in Uppsala University Hospital from 1915 to 1929 (first generation). All subsequent generations born to UBCoS members until 2002 have been traced through the Multi-Generation Register that made it possible to identify the offspring of the original cohort if they were born in 1932 or later and resident and registered in Sweden at least once during 1961–2002. The Multi-Generation Register also provided information about the identity of the other biologic (or adoptive) parent of the children (second generation) and grandchildren (third generation), and we could thus reconstruct the family links for all subsequent generations born until the end of 2002. Register-based data on grandparental and parental social characteristics, school grades at age 15 years, and hospitalizations for eating disorders were obtained by linkages through unique personal identification numbers and covered a follow-up period from 1960 to 2002 (35).

There were 14,338 females in the third generation of UBCoS, who were born in 1952–1989. We excluded 330 (2.3%) who were adopted into UBCoS families and an additional 414 (2.9%) who were born outside of Sweden themselves or whose parent(s) were born outside of Sweden.

Because the diagnostic criteria for anorexia nervosa include amenorrhea, the start of the study

period was set to age 12 years. Another 218 (1.5%) women who had died, emigrated, or already been hospitalized for an eating disorder by this age were also excluded, leaving data on 13,376 women in the analysis.

Information on parental educational level was obtained from the census and the Longitudinal Database for Education, Income and Occupation databases and was analyzed in 3 categories as “elementary” (<10 years), “secondary” (11–12 years), and “postsecondary” (?13 years). Mother’s and father’s gross income was obtained from the Longitudinal Database for Education, Income and Occupation and was analyzed in tertiles. Grandparental education was obtained from census data and was analyzed in 2 categories as “elementary” or “higher.” Information on sibling position and sex of siblings was generated from information obtained from the Multi-Generation Register.

### **School performance**

Information on school performance was obtained from the Swedish National Agency for Education and concerned the grades earned during the last year of compulsory school (typically age 15 years). The data were based on 2 different grading systems. In the first study period, Sweden had a national relative grading system. Grades ranged from 1 to 5; 5 was the highest. Teachers were guided through national achievement tests given in English (year 8) and mathematics and Swedish (year 9). In the school year 1995–1996, a new criterion-referenced grading system was introduced, with grades divided into 4 levels: IG (not pass), G (pass), VG (pass with distinction), and MVG (pass with special distinction) and prespecified skills required for different levels (36). Because there was no obvious objective way to reconcile the grades over the 2 periods, we chose to analyze them separately. However, because there were very few eating disorders events among females with school grades from the later period (3 or 4, depending on the school subject), we ultimately analyzed grades from the earlier period only. Grades for Swedish language, biology, physics, history, mathematics, and English, and a combined average grade, were used in this analysis.

During 1988–1996, mathematics and English were offered at 2 different levels, basic and advanced. The lack of an objective method for combining the grades across levels again led us to analyze the levels separately. Because there were very few eating disorders events among females taking the basic level (8 in mathematics and 3 in English), we analyzed only those females taking the advanced level.

### **Definitions of eating disorders**

Data on eating disorders were obtained from the hospital discharge register, and we combined the information from all versions of the *International Classification of Diseases (ICD)* (37) used during the study period. The study subjects were classified as having eating disorders if any of the following diagnoses and codes were included on the discharge record for hospitalizations at age 12 years or older: ICD, Eighth Revision: 306.5 (feeding disturbances); ICD, Ninth Revision: 307.1 (anorexia nervosa) and 307.5 (other and unspecified disorders of eating); ICD, Tenth Revision: F50 (eating disorders), F50.0 (anorexia nervosa), F50.1 (atypical anorexia), F50.2 (bulimia nervosa), F50.3 (atypical bulimia), F50.4 (overeating), F50.5 (vomiting associated with other psychosocial disturbances), F50.8 (other eating disorders), and F50.9 (eating disorders, unspecified). Separate analysis for a subgroup of patients with anorexia nervosa was based on diagnoses from ICD, Ninth Revision (code 307.1) and ICD, Tenth Revision (codes F50.0, F50.1).

## Statistical methods

Associations of social characteristics and school performance with incidence of eating disorders were studied by using multivariable Cox regression models, adjusted for age and study period. The standard errors of the estimates were calculated allowing for the potential correlations caused by subjects sharing the same parents or grandparents. Because school grades were observed at age 15 years, the study period in analyses concerning school performance did not begin until this age. For all other risk factors, exposure began at age 12 years. Data for women who died or emigrated before the end of follow-up (the end of December 2002) were censored at the date of death or emigration. All analyses were performed by using Stata software: Stata/IC version 10.0 for Macintosh and Stata/SE version 10.0 (38).

## RESULTS

The 13,376 third-generation UBCoS females included in the analyses were born between 1952 and 1989 to 9,648 different mothers. Tables 1 and 2 summarize the available data for each risk factor considered. The proportion of missing data ranged from 1.9% to 4.0% for the parental social characteristics and from 1.2% to 3.5% for the grandparental education variables. Regarding school subjects offered at only a single level, grades were missing for 8.5%–18.2% of females (calculated as a percentage of those born within the period for which school grades based on the earlier grading system were available (January 1973–July 1982)). In mathematics and English, grades were missing for 9.7% and 11.3%, respectively, of females who did not have basic-level grades. In each analysis, the sample size was further affected by the need to maintain a consistent sample when adjusting for additional risk factors.

In the analyses concerning parental social characteristics, grandparental education, and sibling position, where the study period began at age 12 years, 454 females died or emigrated before the end of follow-up. There were 55 cases of eating disorders recorded among the 13,376 study subjects, who altogether completed 171,837 years of follow-up. The overall incidence rate of eating disorders was 32.0 per 100,000 person-years. Anorexia nervosa accounted for 50.9% of the recorded cases of eating disorders.

In age- and period-adjusted analyses, both higher parental education and higher parental income were associated with increased risk of eating disorders in daughters, although the trend for father's income was not statistically significant (Table 3). The effect became weaker and not statistically significant after additional adjustment for the other parent's characteristic, with the exception of the linear trend of risk of eating disorders over tertiles of maternal income that remained borderline significant even when adjusted for father's income (Table 3). We did not find evidence of interaction between the respective parental social characteristics regarding their associations with eating disorders in daughters ( $P = 0.18$  for maternal and paternal education and  $P = 0.89$  for maternal and paternal income).

Risk of eating disorders was substantially increased for women with more highly educated maternal grandmothers (Table 4). This increased risk was only partly confounded by maternal grandfather's and parents' education. The fully adjusted hazard ratio associated with a higher than elementary compared with an elementary education for maternal grandmothers was 6.5 (95%

confidence interval: 2.2, 19.3). Age- and period-adjusted analysis also indicated an increased risk of eating disorders for women with maternal grandfathers with a higher education; however, that association became weaker and nonsignificant after adjustment for maternal grandmother's education.

In the analyses concerning parental social characteristics and grandparental education, we found no evidence that included subjects differed from those who were not included in terms of the crude incidence of eating disorders. However, excluded subjects were often born a little earlier (results not shown).

Analyses of sibling position and sex of siblings indicated a protective effect of a larger family and the presence of older siblings and brothers in particular, but none of the effects were statistically significant (results not shown). The hazard ratio associated with having one or more younger sisters was of borderline significance (hazard ratio = 1.68, 95% confidence interval: 0.97, 2.91).

In the analyses concerning school performance, where the study period began at age 15 years, 28 cases of eating disorders were observed among the 5,632 eligible study subjects during 56,235 person-years of follow-up, giving an incidence rate of 49.8 per 100,000 person-years. Anorexia nervosa accounted for 42.9% of the recorded cases of eating disorders.

The trend in risk of eating disorders over average school grades indicated some evidence of higher risk for females in the highest grade category (Table 5), a result that became weaker after adjustment for parental education. Of the specific subjects studied, we noted a markedly increased risk of eating disorders for females with the highest grade in the Swedish language. Receiving the highest grades in biology, physics, history, mathematics, and English was also associated with a higher risk of incidence of eating disorders in study subjects. These effects were slightly reduced after adjustment for parental education. The linear trends in increasing risk of eating disorders over increasing grades in physics, history, mathematics, and English remained borderline statistically significant after adjustment for parental education, whereas the associations with grades from other subjects studied lost statistical significance.

There was some evidence that crude incidence rates of eating disorders were higher among subjects not included in the school performance analyses compared with those included (results not shown). However, the number of events of eating disorders in the excluded subjects was small.

Despite the fact that the statistical power of analyses restricted to females with anorexia nervosa was very limited, we repeated the above analyses by restricting the outcome to anorexia nervosa only for all risk factors apart from school grades (where the reduced sample size meant that insufficient eating disorders events were observed, Table 1). Results of these analyses were largely consistent with the reported associations of social characteristics and sibling position with any diagnosis of eating disorders.

## **DISCUSSION**

The most important finding in the present study was the increased risk of hospitalization for an eating disorder among daughters of more highly educated parents. There was a doubled risk of eating disorders associated with postsecondary education relative to elementary education, and a significant linear trend was observed. It is noteworthy that, in line with this trend, a higher than elementary education for maternal grandmothers indicated a 6-fold increased risk of eating disorders relative to elementary education, a result that was significant even when adjusted for grandfather's and parental education. Thus, parental social characteristics and maternal social background appeared to be associated with risk of hospitalization for eating disorders among daughters. Together, those findings indicate a role of high internal and external demands in the etiology of eating disorders.

Furthermore, we found a higher risk of eating disorders among females with the highest grades. Even after adjusting for parental education, there was a marked increase in risk of eating disorders among females with the highest grades in Swedish language and some evidence of increased risk of eating disorders with increasing grades in several other subjects.

A main limitation of our study is that we could study the risk of only hospitalization for eating disorders and could not include cases of eating disorders diagnosed and treated in outpatient care. This limitation necessarily meant that our analysis was restricted to the most severe cases of eating disorders that required hospitalization. On the other hand, it is most likely that, during the period of follow-up, and the earlier years of follow-up in particular, clinically diagnosed eating disorders cases would be admitted to inpatient care. The analyses presented in this paper were adjusted for age and calendar period and thus also indirectly address problems with changing diagnostic and treatment practices over the period of our study.

There is a slight possibility that our outcome was influenced by differential health-care-seeking behavior by families with different social positions or different levels of parental education. It is plausible that, for example, parents with a higher education might seek medical help for their daughters earlier or be more active in requesting access to inpatient care. Latzer et al. (39) found a higher educational level among parents of eating disorders patients than in the general population in Israel and interpreted this finding as a likely consequence of systematic differences in help-seeking patterns. Earlier research on help-seeking behavior regarding eating disorders also showed that individuals from ethnic minorities were less likely to seek help (39–41). Although subjects born outside of Sweden themselves or with parents who were born outside of Sweden were excluded from our study, it is possible that the results also partly reflect differences in help-seeking behavior rather than differences in the incidence of eating disorders. Although concerns about equity in access to health care have been voiced in Sweden recently (42), access to health services during the period of follow-up was generally good, with no financial barriers to use of hospital care in particular.

Because information used for our analysis is based on data included in routine registers, validity of our results depends on the quality and completeness of the register data and a correct linkage of data between the respective registers. The hospital discharge register is unlikely to be complete; however, we believe that the relatively small proportion of missing data did not lead to serious bias in our analysis.

Although we would have preferred to use school-grade data from both school grading systems, the lack of an objective method of combining the grades and the scarcity of eating disorders events during the more recent period meant that grades from only the earlier period could be utilized. Future analysis of associations between school grades under the more recent grading system and eating disorders risk is important to assessing whether the trends we observed are common to both grading systems. Our inability to analyze basic-level mathematics and English grades because of a small number of events could also potentially have introduced bias. Reassuringly, the results obtained were in line with those observed for other school subjects.

It is unlikely that misclassification of the risk factors differed between subjects with and without eating disorders (nondifferential misclassification), meaning that any misclassification would lead to attenuation of the hazard ratios. Although we would not expect substantial misclassification of risk factors, this possibility suggests that the true associations may be greater than those observed. Because the outcome we considered was hospitalization for eating disorders, it is unlikely that significant outcome misclassification occurred, although the possibility of true eating disorders cases being misdiagnosed does exist. This misclassification would result in the true incidence of eating disorders being higher than that observed and, if misdiagnosis was associated with the risk factors examined, could bias the results.

The proportion of subjects missing from analyses involving parental and grandparental risk factors was low, so the effects of any selection bias are unlikely to be substantial. Excluded subjects were generally similar to included subjects, although, in analyses of school performance, there was some evidence of a higher incidence of eating disorders among excluded subjects. If we thought that the excluded subjects were likely to be those with lower school attainment, then their exclusion could explain some or all of the observed trends of increasing risk of eating disorders with increasing school grades. However, the number of eating disorders events among excluded subjects was low, so these observations should not be overinterpreted.

The unique feature of our study was our ability to combine data on 2 social indicators of both mothers and fathers and to study associations with the educational level of maternal and paternal grandparents. Moreover, we are not aware of other large studies of the incidence of eating disorders in groups with different levels of school performance.

Our finding of a higher risk of eating disorders for females with high school grades may indicate high ambition in those females, partly supporting the findings of relatively higher school achievement than expected by IQ scores among patients with anorexia nervosa (34). Our findings, particularly regarding the high risk of eating disorders among females with high grades in Swedish language, may also, more speculatively, reflect cognitive strategy preferences among females with eating disorders, especially those with anorexia nervosa, for details/accuracy closely related to traits reflecting control and perfectionism (43).

Although the results concerning siblings and birth order are not conclusive, there is some indication that having older brothers may reduce the risk of eating disorders, whereas having younger sisters may increase the risk. However, these findings should be interpreted cautiously because of the lack of statistical significance. Early studies have suggested that patients with eating disorders have more sisters (44), and more recent reports found that anorexia nervosa

patients had fewer brothers than controls did (27). The number of siblings might be related to other aspects of the family setting such as internal relationships, family climate, and attitudes.

A negative childhood climate has been suggested as a risk factor for development of eating disorders, especially bulimia nervosa (45), and physical or psychological abuse in childhood is recognized as a risk factor for psychiatric disorders in general (46, 47). Our results on associations between eating disorders and family characteristics are consistent with earlier findings (23) in that we also found an indication of a higher risk of eating disorders for females from socially advantaged families. Although we do not think that quality of parenting is systematically worse among highly educated parents or mothers with high income, we cannot exclude the possibility that problems regarding certain aspects of parenting, such as time and parental engagement during sensitive periods of development, may mediate the effects seen in our analysis. In our view, the increased risk of eating disorders for granddaughters of more highly educated maternal grandmothers further supports a role of high internal and external demands in shaping the incidence patterns of eating disorders found in our study.

Despite the improving survival of adolescent patients with anorexia nervosa in Sweden (48), it is generally believed that our lack of understanding of etiology still hampers the development of more effective treatments for eating disorders (49, 18). The results of the present study emphasize the importance of multigenerational information and longitudinal research, as highlighted by others (49, 50), and point to the need for future research on various types of risk factors and interactions between parental health and social characteristics, early-life environment, and individual psychological factors in assessing the risk and long-term outcome of eating disorders.

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## REFERENCES

1. SOU. Adolescents, stress and psychiatric health. Analyses and suggestions for action [in Swedish]. Stockholm, Sweden: Statens Offentliga Utredningar; 2006. (Swedish Government official report no. SOU 2006:77).
2. van Son GE, van Hoeken D, Bartelds AI, et al. Time trends in the incidence of eating disorders: a primary care study in the Netherlands. *Int J Eat Disord*. 2006;39(7):565–569.
3. Hoek HW. Incidence, prevalence and mortality of anorexia nervosa and other eating disorders. *Curr Opin Psychiatry*. 2006;19(4):389–394.
4. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders: DSM-IV*. 4th ed. Washington, DC: American Psychiatric Association; 1994.
5. Mathers C, Vos T, Stevenson C. *The Burden of Disease and Injury in Australia*. Canberra, Australia: Australia Institute of Health and Welfare; 1999. (AIHW cat. no. PHE 17). (<http://www.aihw.gov.au/publications/phe/bdia/bdia.pdf>).
6. Treasure J. Eating disorders. *Medicine*. 2008;36(8):430–435.
7. Hudson JI, Hiripi E, Pope HG Jr, et al. The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. *Biol Psychiatry*. 2007;61(3):348–358.
8. Wade TD, Bergin JL, Tiggemann M, et al. Prevalence and long-term course of lifetime eating disorders in an adult Australian twin cohort. *Aust N Z J Psychiatry*. 2006;40(2):121–128.
9. Favaro A, Ferrara S, Santonastaso P. The spectrum of eating disorder in young women: a prevalence study in a general population sample. *Psychosom Med*. 2003;65(4):701–708.
10. Currin L, Schmidt U, Treasure J, et al. Time trends in eating disorder incidence. *Br J Psychiatry*. 2005;186:132–135.
11. Darby A, Hay P, Mond J, et al. The rising prevalence of comorbid obesity and eating disorder behaviors from 1995 to 2005. *Int J Eat Disord*. 2009;42(2):104–108.
12. Hay JP, Mond J, Buttner P, et al. Eating disorder behaviors are increasing: findings from two sequential community surveys in South Australia [electronic article]. *PLoS ONE*. 2008;3(2):e1541.
13. Jacobi C, Hayward C, de Zwaan M, et al. Coming to terms with risk factors for eating disorders: application of risk terminology and suggestions for a general taxonomy. *Psychol Bull*. 2004;130(1):19–65.
14. Fairburn CG, Harrison, PJ. Eating disorders. *Lancet*. 2003;361(9355):407–416.
15. Schmidt U. Aetiology of eating disorders in the 21(st) century: new answers to old questions. *Eur Child Adolesc Psychiatry*. 2003;12(suppl 1):I30–I37.
16. Morrison T, Waller G, Meyer C, et al. Social comparison in the eating disorders. *J Nerv Ment Dis*. 2003;191(8):553–555.
17. Gilbert N, Meyer C. Social anxiety and social comparison: differential links with restrictive and bulimic attitudes among nonclinical women. *Eat Behav*. 2003;4(3):257–264.
18. Gatward N. Anorexia nervosa: an evolutionary puzzle. *Eur Eat Disord Rev*. 2007;15(1):1–12.
19. Troop NA, Baker AH. The specificity of social rank in eating disorder versus depressive symptoms. *Eat Disord*. 2008;16(4):331–341.
20. Bulik CM, Sullivan PF, Tozzi F, et al. Prevalence, heritability and prospective risk factors for anorexia nervosa. *Arch Gen Psychiatry*. 2006;63(3):305–312.
21. Graham H, Power C. Childhood disadvantage and health inequalities: a framework for policy based on lifecourse research. *Child Care Health Dev*. 2004;30(6):671–678.
22. Gibbons P. The relationship between eating disorder and socioeconomic status: it's not what

- you think. *Nutr Noteworthy*. 2001;4(article 3);1–5.
23. Lindberg L, Hjern A. Risk factors for anorexia nervosa: a national cohort study. *Int J Eat Disord*. 2003;34(4):397–408.
  24. Råstam M, Gillberg, C. The family background in anorexia nervosa: a population-based study. *J Am Acad Child Adolesc Psychiatry*. 1991;30(2):283–289.
  25. Claes L, Vandereycken W, Vertommen H. Family environment of eating disordered patients with and without self-injurious behaviours. *Eur Psychiatry*. 2004;19(8):494–498.
  26. Gowers S, Kadambari SR, Crisp AH. Family structure and birth order of patients with anorexia nervosa. *J Psychiatr Res*. 1985;19(2–3):247–251.
  27. Eagles JM, Johnston MI, Millar HR. A case-control study of family composition in anorexia nervosa. *Int J Eat Disord*. 2005;38(1):49–54.
  28. Bachner-Melman R. Siblings in the context of anorexia nervosa. *Isr J Psychiatry Relat Sci*. 2005;42(3):178–184.
  29. Canetti L, Kanyas K, Lerer B, et al. Anorexia nervosa and parental bonding: the contribution of parent-grandparent relationships to eating disorder psychopathology. *Clin Psychol*. 2008;64(6):703–716.
  30. Duchesne M, Mattos P, Fontenelle LF, et al. Neuropsychology of eating disorders: a systematic review of the literature. *Rev Bras Psiquiatr*. 2004;26(2):107–117.
  31. Tchanturia K, Anderluh MB, Morris RG, et al. Cognitive flexibility in anorexia nervosa and bulimia nervosa. *J Int Neuropsychol Soc*. 2004;10(4):513–520.
  32. Blanz BJ, Detzner U, Lay B, et al. The intellectual functioning of adolescents with anorexia nervosa and bulimia nervosa. *Eur Child Adolesc Psychiatry*. 1997;6(3):129–135.
  33. Gillberg IC, Råstam M, Wentz E, et al. Cognitive and executive functions in anorexia nervosa ten years after onset of eating disorder. *J Clin Exp Neuropsychol*. 2007;29(2):170–178.
  34. Dura JR, Bornstein RA. Differences between IQ and school achievement in anorexia nervosa. *J Clin Psychol*. 1989;45(3):433–435.
  35. Koupil I. The Uppsala studies on developmental origins of circulatory disease. *J Intern Med*. 2007;261(5):426–436.
  36. Björklund A, Lindahl M, Sund K. Family background and school performance during turbulent era of school reforms. *Swedish Econ Policy Rev*. 2003;10:111–136.
  37. World Health Organization. *The ICD-10 Classification of Mental and Behavioural Disorders. Diagnostic Criteria for Research*. Geneva, Switzerland: WHO; 1993.
  38. Stata. Stata Statistical Software, release 10. College Station, TX: StataCorp LP; 2007.
  39. Latzer Y, Vander S, Gilat I. Socio-demographic characteristics of eating disorder patients in an outpatient clinic: a descriptive epidemiological study. *Eur Eat Disord Rev*. 2008;16(2):139–146.
  40. Becker AE, Franko DL, Speck A, et al. Ethnicity and differential access to care for eating disorder symptoms. *Int J Eat Disord*. 2003;33(2):205–212.
  41. Cachelin FM, Striegler-Moore RH. Help seeking and barriers to treatment in a community sample of Mexican American and European American women with eating disorders. *Int J Eat Disord*. 2006;39(2):154–161.
  42. Burström B. Care choice—evidence and effects of “equal care” [in Swedish]? *Läkartidningen*. 2008;105(43):2992–2994.
  43. Southgate L, Tchanturia K, Treasure J. Information processing bias in anorexia nervosa. *Psychiatry Res*. 2008;160(2):221–227.
  44. Theander S. Anorexia nervosa: a psychiatric investigation of 94 female cases. *Acta Psychiatr*

*Scand Suppl.* 1970;214:1–194.

45. Ahrén-Moonga J, Holmgren S, von Knorring L, et al. Personality traits and self-injurious behaviour in patients with eating disorders. *Eur Eat Disord Rev.* 2007;16(4):268–275.
46. Lang S, af Klinteberg B, Alm PO. Adult psychopathy and violent behavior in men with early neglect and abuse. *Acta Psychiatr Scand Suppl.* 2002;106(412):93–100.
47. Ramklint M, Ekselius L. Personality traits and personality disorders in early onset versus late onset major depression. *J Affect Disord.* 2003;75(1):35–42.
48. Lindblad F, Lindberg L, Hjern A. Improved survival in adolescent patients with anorexia nervosa: a comparison of two Swedish national cohorts of female inpatients. *Am J Psychiatry.* 2006;163(8):1433–1435.
49. Pratt BM, Woolfenden SR. Interventions for preventing eating disorders in children and adolescents [electronic article]. *Cochrane Database Syst Rev.* 2002;(2):CD002891.
50. Ricciardelli LA, McCabe MP. A biopsychosocial model of disordered eating and the pursuit of muscularity in adolescent boys. *Psychol Bull.* 2004;130(2):179–205.

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**Table 1.** Distribution of Parental Education and Income and of Grandparental Education Among the 13,376 Eligible Third-Generation Uppsala Birth Cohort Females Born in 1952–1989, Sweden

	No.	%	Eating Disorders	Anorexia Nervosa
Mother's education				
Maternal				
grandmother				
r				
	HR		95% CI	No. of Events P Value From Wald Test
Grandmother				
Grandmother				
r				

|| **HR** | **95% CI** | **P Value From Wald Test** | **P Value for Linear Trend** | **HR** | **95% CI** | **P Value From Wald Test** | **P value for Linear Trend** | Combined average | 4,965 | | | | | | | | | Low | | 1 | | | | 1 | | | | | Medium | | 1.17 | | 0.33, 4.16 | | 0.81 | | 1.04 | | 0.29, 3.80 | | 0.95 | | | High | | 2.66 | | 0.87, 8.10 | | 0.09 | | 0.06 | | 2.15 | | 0.71, 6.50 | | 0.17 | | 0.13 | | Swedish | 4,948 | | | | | | | | | Low | | 1 | | | | 1 | | | | | Medium | | 2.55 | | 0.87, 7.44 | | 0.09 | | 2.46 | | 0.84, 7.21 | | 0.10 | | | High | | 9.61 | | 3.20, 28.88 | | <0.001 | | <0.001 | | 9.28 | | 2.91, 29.56 | | <0.001 | | <0.001 | | Biology | 4,569 | | | | | | | | | Low | | 1 | | | | 1 | | | | | Medium | | 2.46 | | 0.80, 7.54 | | 0.12 | | 2.01 | | 0.56, 7.19 | | 0.28 | | | High | | 4.11 | | 1.09, 15.57 | | 0.04 | | 0.02 | | 3.01 | | 0.63, 14.39 | | 0.17 | | 0.14 | | Physics | 4,552 | | | | | | | | | Low | | 1 | | | | 1 | | | | | Medium | | 1.52 | | 0.48, 4.78 | | 0.48 | | 1.26 | | 0.37, 4.23 | | 0.71 | | | High | | 5.57 | | 1.75, 17.77 | | 0.004 | | 0.02 | | 4.28 | | 1.12, 16.32 | | 0.03 | | 0.08 | | History | 4,435 | | | | | | | | | Low | | 1 | | | | 1 | | | | | Medium | | 0.83 | | 0.21, 3.31 | | 0.8 | | 0.71 | | 0.19, 2.66 | | 0.62 | | | High | | 6.85 | | 2.32, 20.17 | | <0.001 | | 0.01 | | 5.34 | | 1.56, 18.28 | | 0.01 | | 0.04 | | Mathematics | 2,824 | | | | | | | | | Low | | 1 | | | | 1 | | | | | Medium | | 2.31 | | 0.71, 7.57 | | 0.17 | | 2.13 | | 0.64, 7.06 | | 0.22 | | | High | | 5.62 | | 1.49, 21.19 | | 0.01 | | 0.01 | | 4.79 | | 1.13, 20.32 | | 0.03 | | 0.04 | | English | 3,780 | | | | | | | | | Low | | 1 | | | | 1 | | | | | Medium | | 2.18 | | 0.81, 5.84 | | 0.12 | | 1.96 | | 0.70, 5.43 | | 0.20 | | | High | | 3.82 | | 1.10, 13.23 | | 0.04 | | 0.02 | | 3.19 | | 0.78, 13.06 | | 0.11 | | 0.08

|| **Abbreviations:** CI, confidence interval; HR, hazard ratio.

<sup>a</sup> Adjusted for age and study period.

<sup>b</sup> Additionally adjusted for maternal and paternal education.