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Patterns of remission, continuation, and incidence of broadly defined eating disorders during early pregnancy in the Norwegian Mother and Child Cohort Study (MoBa)

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Abstract

Background: We explored the course of broadly defined eating disorders during pregnancy in the Norwegian Mother and Child Cohort Study (MoBa) at the Norwegian Institute of Public Health.

Method: 41,157 pregnant women, enrolled at approximately 18 weeks gestation, had valid data from the Norwegian Medical Birth Registry. We collected questionnaire based diagnostic information on broadly defined anorexia nervosa, and bulimia nervosa (BN), and eating disorders not otherwise specified (EDNOS). EDNOS subtypes included binge eating disorder (BED) and recurrent self-induced purging in the absence of binge eating (EDNOS-P). We explored rates of remission, continuation, and incidence of BN, BED and EDNOS-P during pregnancy.

Results: Pre-pregnancy prevalence estimates were 0.1% for anorexia nervosa, 0.7% for BN, 3.5% for BED, and 0.1% for EDNOS-P. During early pregnancy, estimates were 0.2% (BN), 4.8% (BED), and 0.1% (EDNOS-P). Proportions of individuals remitting during pregnancy were 78% (EDNOS-P), 40% (BN purging), 39% (BED), 34% (BN any type), 29% (BN non-purging type). Additional individuals with BN achieved partial remission. Incident BN and EDNOS-P during pregnancy were rare. For BED, the incidence rate was 1.1 per 1000 person-weeks equating to 711 new cases of BED during pregnancy. Incident BED was associated with indices of lower socioeconomic status.

Conclusions: Pregnancy appears to be a catalyst for remission of some eating disorders, but also a vulnerability window for the new onset of broadly defined BED especially in economically disadvantaged individuals. Vigilance by health care professionals for continuation and emergence of eating disorders in pregnancy is warranted.

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A complex interaction exists between pregnancy and eating disorders. Early studies suggested that pregnancy in women with anorexia nervosa (AN) was rare due to the endocrinological, psychological, and psychosocial features of the disorder (Strimling, 1984, Weinfeld et al., 1977). However, some, but not all outcome studies of AN and BN suggest that fertility may not differ from the general population in those who survive the illness (Kohmura et al., 1986, Bulik et al., 1999, Crow et al., 2002). No population-level studies exist that explore the course of eating disorders during pregnancy.

Our current knowledge about pregnancy and eating disorders comes primarily from clinical samples. These studies suggest that the impact of eating disorders on the pregnant woman and her unborn child can be significant—especially when the eating disorder is not detected or persists despite treatment (Abrams and Laros, 1986, van der Spuy et al., 1988, Stewart et al., 1987, Treasure and Russel, 1988, Brinch et al., 1988, Lacey and Smith, 1987, Conti et al., 1998). Increased complications from conception through the postpartum period have been described that adversely impact maternal and fetal well-being. Maternal complications that have been noted to occur at a higher prevalence in women with AN include miscarriages and cesarean deliveries (Bulik et al., 1999, Franko et al., 2001). High cesarean rates have also been reported in women with bulimia nervosa (BN) (Mitchell et al., 1991). Features that are characteristic of eating disorders such as low pre-pregnancy weight and low gestational weight gain are associated with adverse birth outcomes as well (Abrams and Newman, 1991, Siega-Riz et al., 1996) and a higher incidence of congenital malformations (van der Spuy et al., 1988). Poor birth outcomes that impact the infants of women with eating disorders include stillbirth, low birth weight, low Apgar scores, higher occurrence of breech presentation, and cleft lip and palate (Stewart et al., 1987, Treasure and Russel, 1988, Brinch et al., 1988, Lacey and Smith, 1987, Conti et al., 1998). Crow et al. (Crow et al., 2004) reported that bulimic symptoms tended to improve during pregnancy, pregnant women with past or active BN have been reported to be at greater risk for hyperemesis, and delivering infants with smaller head circumference, microcephaly, and who are small for gestational age, compared to healthy controls (Kouba et al., 2005). Less is known about the course and impact on pregnancy of eating disorders not otherwise specified (EDNOS). In this paper, we are particularly interested the impact of two forms of EDNOS, namely binge eating disorder (BED) and a clinical presentation marked by self-induced purging behavior in the absence of binge eating which we refer to as EDNOS-P. Although only recently the subject of scientific inquiry, this presentation has been described as a clinically significant phenomenon (Keel et al., 2005) and identified in a population-based sample of Australian twins (Wade, 2007).

Most studies of eating disorders and pregnancy have been retrospective reports on clinical samples. Some women with active eating disorders report a temporary or permanent improvement in eating disorders symptomatology during pregnancy (Lacey and Smith, 1987, Lemberg and Phillips, 1989, Morgan et al., 1999, Namir et al., 1986). However, the eating disorder, and a renewed obsession with slimness, may become acute in the postpartum period (Willis and Rand, 1988, Lacey and Smith, 1987, Lemberg and Phillips, 1989, Morgan et al., 1999). This may be offset by successful treatment, as we have previously found improved symptomatology in women with BN who became pregnant in the interval following treatment (Carter et al., in press).

Although the aforementioned studies are intriguing, they have important methodological limitations (e.g. clinical samples, retrospective data) and are therefore unable to shed adequate light on illness course during pregnancy. The present study advances this body of preliminary knowledge with information from the initial assessments in a prospective population-based birth cohort study. The Norwegian Mother and Child Cohort Study (MoBa) is designed to recruit a sample of 100,000 pregnant women between 1999 and 2008 or until the target is reached. This study collected information on eating disorders and eating disordered behavior

of mothers assessed during pregnancy about their behavior both prior to and during pregnancy. We report the prevalence of eating disorders and disordered eating behavior prior to pregnancy as well as the patterns of remission, continuation, and incidence of eating disorders during pregnancy of 41,157 women entered into this cohort.

METHOD

Participants

The data collection was conducted as part of the Norwegian Mother and Child Cohort Study (MoBa) at the Norwegian Institute of Public Health (Magnus et al., 2006). The study has been approved by the appropriate regional committees for ethics in medical research and the Norwegian National Data Inspectorate.

In brief, MoBa is a prospective pregnancy cohort study. Pregnant women are recruited through a postal invitation after registering for a routine prenatal ultrasound at about 18 weeks gestation. Participating women sign informed consent to take part in a longitudinal study, donate blood and urine samples, and receive a questionnaire. The present study is based on the first questionnaire, and includes assessment of a range of exposures and health outcome variables. The MoBa cohort is linked to Norwegian health registries, particularly the Medical Birth Registry (MBR) (Irgens et al., 2000) to capture pregnancy outcome variables.

The analysis population for this report included MoBa participants who: a) had information from both the MoBa Questionnaire 1 and the MBR, b) did not complete an early pilot version of Questionnaire 1 ($n=2,599$), c) had valid values for self-reported age, weight, and height, d) returned Questionnaire 1 before delivery, and e) had a singleton birth. If a woman enrolled in MoBa more than once (due to additional pregnancies), only the first pregnancy was included. Of the initial 54,714 pregnancies enrolled in MoBa, 41,157 (75%) met the five criteria above and were included in this report. Overall, from 1999-2006, ~42% of invited mothers have agreed to participate in MoBa.

Measures

Medical Birth Registry of Norway (MBR)—The MBR was established in 1967 (Irgens et al., 2000). All stillbirths and live births after 16 weeks of pregnancy are reported to the MBR through mandatory notification by midwives and doctors (Irgens et al.). National identification numbers of child and mother are recorded for all births. Variables from the MBR used for this report included maternal age, marital status, region of birth, and gestational age of baby at birth. Of all MoBa pregnancies, 4.5% did not have a MBR record and were excluded. Data from the MBR have been used for prior biomedical research (Skjaerven et al., 2005, Skjaerven et al., 2002, Lie et al., 2001).

MoBa Questionnaire 1—Questionnaire 1 included items on eating disorders and behaviors that were previously used for studies of eating disorders in the Norwegian Institute of Public Health Twin Panel (Harris et al., 2002, Reichborn-Kjennerud et al., 2003, Reichborn-Kjennerud et al., 2004a, Reichborn-Kjennerud et al., 2004b) and were designed in accordance with DSM-IV criteria for AN, BN, and EDNOS (American Psychiatric Association, 1994). Diagnostic algorithms were constructed from the questionnaire items to define eating disorder subtypes: broadly defined anorexia nervosa, defined as meeting DSM IV criteria for AN (with the exception of amenorrhea and also endorsing a BMI <18.5 at the time of low weight); broadly defined bulimia nervosa (endorsing at least weekly frequency of binge eating and purging and categorized as BN any type, BN purging type, BN non-purging, type); broadly defined binge eating disorder (BED, at least weekly frequency of binge eating in the absence of compensatory behaviors), and EDNOS-P (purging at least weekly in the absence of binge eating). Questions

for binge eating included both eating an unusually large amount of food and feeling out of control. AN was assessed before pregnancy only due to practical difficulties in determining low weight in the presence of pregnancy-related weight gain. BN, BED, and EDNOS-P were assessed for both 6 months prior to pregnancy (retrospective assessment) and at the time of survey completion. The respondents were specifically asked to distinguish between pregnancy-related nausea and vomiting and self-induced vomiting as a purging method. Self-reported weight and height were used to calculate pre-pregnancy body mass index (BMI, kg/m²) and BMI at the time of assessment. Respondents completed questionnaire 1 at a median of 18.1 weeks gestation (inter-quartile range 16.7-20.1 weeks and range 5.3-41.9 weeks).

Eating Disorder Classifications

In this report, we studied 16 diagnostic groupings defined by diagnosis and time in relation to pregnancy. Fifteen of these are defined by the combinations of five broad diagnoses (BN-any type, BN-purging type, BN-non-purging type, BED, and EDNOS-P) and three periods of time (before pregnancy, during pregnancy, and before and during pregnancy). Each criterion had missing observations. If a person was missing one of the diagnostic criteria and if there were no negative responses to any of the other criteria, then that eating disorder group for that person was classified as missing.

According to the DSM-IV criteria, BED and BN are mutually exclusive diagnoses as are BN-purge and BN-non-purge. The category BN-any type is not a simple summation of BN purging and BN non-purging types as individuals who responded to questions that defined non-purging BN (i.e. fasting and exercise), but had missing values for the questions that defined purging BN (i.e. laxatives and vomiting) were included in the BN any type category.

Definition of remission, incidence and continuation

Remission refers to those individuals who reported an eating disorder before pregnancy and no eating disorder during pregnancy. Specifically with reference to core symptoms, for BN, this means the absence of binge eating and the absence of compensatory behaviors and for BED, the absence of binge eating. Partial remission refers to those individuals who met criteria for BN prior to pregnancy, but who reported the absence of compensatory behaviors but the continued presence of binge eating during pregnancy. Continuation refers to those individuals who report having had an eating disorder before pregnancy and continued to have the same eating disorder during pregnancy. Finally, incidence refers to the number of new cases of broadly defined eating disorders occurring during early pregnancy in the group of mothers who reported no eating disorder in the 6 months before pregnancy.

Statistical Analysis

We had four analytic goals for this report. First, we described the demographic characteristics using contingency tables. Second, we described the prevalence of the eating disorder diagnoses at 16 time points (as defined above) using contingency tables.

The third goal was more complex, as we wished to report remission, continuation, and incidence rates (defined above) for eating disorders during pregnancy. These rates are measured in events per person-weeks, and must account for (a) the different ages of subjects and (b) the different gestational weeks at which respondents completed the surveys. The most appropriate statistical technique was Poisson regression because it can produce estimates of discrete counts divided by units of exposure (Stokes et al., 2000). In this paper, events represent the discrete counts and “weeks of gestation until survey completion” represents the units of exposure. For example, a model to estimate incidence densities has an incident case as an event and units of exposure equals the total number of gestational weeks until the survey for those without an eating disorder before pregnancy. Age was included as a covariate to correct for any possible

age confounding. To determine robust standard errors for the Poisson regression coefficients of interest, generalized estimating equations (GEE) were used. Our use of GEE here is different from the more typical use of GEE to account for clustered data (e.g., measures on the same subject at multiple time points or for twin pairs). We used GEE in order to estimate robust standard errors using its “empirical” (rather than model-based) approach (Stokes et al., 2000, Carter et al., 2005, Zou, 2004).

The fourth goal was to investigate the correlates of remission, continuation, and incidence. To do this, we again used Poisson regression (with GEE) by adding the term of interest as a covariate to the regression models described above. The null hypothesis was that the effect is the same across all levels in the group (Figure 1). As this goal included a sizeable number of statistical tests, we controlled for multiple comparisons using the false discovery rate (FDR) (Westfall et al., 1999, Benjamini and Hochberg, 1995).

All analyses were done using SAS/STAT® software for Windows and Solaris (v9.1) (SAS Institute Inc., 2004).

RESULTS

Sample demographics

Table 1 presents the demographic composition of the sample. Consistent with initial reports on the MoBa sample (Magnus et al., 2006), the majority of the 41,157 women represented here were between the ages of 25-34, 94.7% were native Norwegian speakers, 52% had at least one live birth before the index child (born prior to the MoBa study), and 97% were married or cohabitating. In addition, the sample was relatively highly educated with 58% attending some form of college and over 91% reporting a combined income greater than the equivalent of \$33,000 per year. Behaviorally, whereas 50% reported having smoked at some time, 32% reported smoking (occasionally or daily) in the three months before pregnancy and 11% during pregnancy. Over 95% reported having ever consumed alcohol.

Prevalence of eating disorders

As shown in Table 2, 37 women (0.1%) reported the presence of broadly defined AN prior to pregnancy. BN was more common with 290 women (0.7%) reporting BN prior to pregnancy, 96 women (0.2%) reporting BN during pregnancy, and 67 women (0.2%) reporting BN both before and during pregnancy. Broadly defined BED was the most commonly reported eating disorder with 1,405 (3.5%) of women reporting its presence prior to pregnancy, 1,856 (4.8%) during pregnancy, and 779 (2.0%) both prior to and during pregnancy. EDNOS-P was reported in 42 women (0.1%) prior to pregnancy and 12 (< 0.1%) of women during pregnancy.

Remission, continuation, and incidence

Table 3 presents age-adjusted rates of remission, continuation, and incidence for five categories of eating disorders (BN any type, BN purging type, BN non-purging type, BED, and EDNOS-P). Rates of remission varied across disorders and were highest for EDNOS-P (42 per 1000 person-weeks) followed by BED (20 per 1000 person-weeks), BN purging (19 per 1000 person-weeks), BN any (17 per 1000 person-weeks), and BN non-purging (15 per 1000 person-weeks). The percentages of individuals remitted were: EDNOS-P (78%), BN purging (40%), BED (39%), BN any (34%), and BN non-purging (29%). The lowest continuation rates during pregnancy were for EDNOS-P (6 per 1000 person weeks) and BN non-purging type (8 per 1000 person weeks), followed by BN any type (16 per 1000 person weeks) and BN purging (20 per 1000 person weeks). Notably, continuation rates in individuals with BED were considerably higher (32 per 1000 person weeks). The percentage of individuals continuing were: EDNOS-P (10%), BN non-purging (16%), BN any type (29%), BN purging (38%), and

BED (61%). The percentages of individuals with BN achieving partial remission (i.e. absence of compensatory behaviors) were: BN non-purging (53%), BN any (34%), and BN purging (18%). Incident cases during pregnancy for all types of BN and EDNOS-P were rare. For BED, incident cases were more common (1.1 per 1000 person weeks) equating to 711 new cases of BED in the sample during the observation period.

Characteristics associated with remission, continuation, and incidence

In exploratory analyses, we determined whether specific demographic factors were associated with remission, continuation, and incidence of BN and BED. These analyses were not conducted for EDNOS-P due to the small number of incident cases. Variables tested included maternal age, pregravid maternal BMI and BMI at the time of assessment, maternal education, median combined household income, ever smoked, previous pregnancies, total number of births, total number of abortions, native language (Norwegian or other), and region of birth. For BN, no demographic variables were significantly associated with either remission, continuation, or incidence with the exception of a negative association between remission and smoking (FDR $p = 0.05$). For BED, higher BMI both before pregnancy and during pregnancy were associated with remission (FDR $p < 0.001$), lower BMI prior to and during pregnancy were associated with continuation (FDR $p < 0.001$), and higher BMI during pregnancy only was associated with incidence (FDR $p = 0.019$). Several additional variables were associated with BED incidence (Figure 1). Lower maternal education (FDR $p < 0.001$), ever smoked (FDR $p < 0.001$), at least one pregnancy (FDR $p < 0.001$), at least one abortion (FDR $p < 0.001$), at least one live birth (FDR $p < 0.001$), lower minimum combined income (FDR $p = 0.009$), and a native language other than Norwegian (FDR $p = 0.009$) were associated with new onsets of BED.

DISCUSSION

To our knowledge, this is the first investigation to document both the prevalence and the patterns of remission, continuation, and incidence of broadly defined eating disorders in a large cohort of pregnant women. In this sample, pre-pregnancy prevalence estimates were 0.1% for AN, 0.7% for BN, 3.5% for BED, and 0.1% for EDNOS-P. Due to the complications of assessing the weight criterion for AN during pregnancy, we only have prevalence data for BN, BED and EDNOS-P during pregnancy. The estimates were 0.2% for BN, 4.8% for BED, and $< 0.1\%$ EDNOS-P. Previous reports from Norway in the general population (i.e. not selected for pregnancy), reported prevalences of 0.3% for AN, 0.7% for BN and 1.5% for BED (Götestam and Agrav, 1995).

Our observed pre-pregnancy prevalence of AN is on the low end of that reported in epidemiologic investigations of women not selected on the basis of pregnancy (Hoek, 2006). This may reflect the fact that at least some individuals with AN have impaired fertility and/or social/interpersonal deficits decreasing their likelihood of becoming pregnant (Strimling, 1984, Weinfeld et al., 1977, Stewart et al., 1990), or alternatively, the stress of pregnancy in individuals with AN could make them less likely to agree to participate in a study such as MoBa.

Examining the course of eating disorders during pregnancy, we found the most common pattern for BN and EDNOS-P to be remission or partial remission of symptoms during pregnancy—with combined remission rates surpassing continuation rates for all types of BN and EDNOS-P. This suggests that for many, but clearly not all women, pregnancy is a powerful stimulus to decrease bulimic symptomatology. This can take the form of reductions in both binge eating and compensatory behavior, or in the case of those partially remitted women, elimination of compensatory behaviors only (which may be viewed as potentially more harmful to the fetus). This parallels similar observations of spontaneous quitting in pregnancy for both alcohol use

and smoking (Bruce et al., 1993, Ockene et al., 2002, Kruse et al., 1986, Solomon and Quinn, 2004). Whether the observed decreases are limited to the period of pregnancy or are more lasting will be addressed in future waves of the MoBa study. In addition, although incident cases of BN during pregnancy were reported, they were rare.

Unexpectedly, we found the course of BED to be different than that of BN. For BED, continuation of symptoms during pregnancy was more common than remission. Most notably, new onsets of BED were much more common than the other eating disorders (1.12 per 1000 person-weeks) or 711 new cases in our sample. We are unaware of previous reports of incident BED during pregnancy and our finding raises the important issue of the validity of questions regarding binge eating during pregnancy. An important topic for further detailed investigation is whether the terms associated with the evaluation of binge eating (e.g. eating and unusually large amount of food, feeling out of control) are more likely to be endorsed by women who are experiencing appetite changes associated with pregnancy. The magnitude of our observed incident cases suggests that this is an important area of inquiry to determine the extent to which the reported behavior truly represents pathological eating behavior.

Although our analyses were unable to detect any demographic parameters associated with the course of BN except for smoking, for BED, new cases were associated with an array of variables reminiscent of the “matrix of disadvantage” (Mullen et al., 1993). Women with higher BMIs during pregnancy, more previous pregnancies, at least one abortion, lower income and education, greater smoking, and whose native language was not Norwegian were more likely to report developing BED during pregnancy. Although additional data are needed to characterize these individuals more fully, from this assessment it appears that general indices of economic and social disadvantage may be associated with the development of this symptom cluster during pregnancy.

Pregnancy is a powerful biopsychosocial event. For some, pregnancy appears to be a window of opportunity for discontinuing bulimic behaviors that could be harmful to both mother and child. For a smaller number of women, however, their bulimic behaviors (binge-eating and purging) persist during pregnancy. We will explore the impact of these persistent behaviors on pregnancy outcome and child development in future waves of data analysis in this cohort.

The somewhat surprising number of incident cases of BED during pregnancy raises several questions. For these women, rather than a window of opportunity, pregnancy appears to be a window of vulnerability. Several potential explanations for the emergence of BED during pregnancy emerge. Biologically, the myriad of adaptive neuroendocrine changes that occur in pregnancy that affect multiple brain functions influencing metabolism, appetite, and mood (Russell et al., 2001) may render pregnancy a high risk period for developing binge-eating. Behaviorally, women who fail to increase food intake according to the increased metabolic needs of pregnancy may experience unexpected and intense hunger which may lead to compensatory overeating and ultimately trigger a pattern of appetite dysregulation. This is perhaps less likely given that changes were seen relatively early in pregnancy prior to appreciable increases in metabolic requirements. Psychologically, especially given the pattern of social disadvantage associated with incident cases in this sample, the psychosocial stress of pregnancy may serve as an environmental trigger for binge eating.

These findings must be considered within the context of several limitations. First, although the diagnostic questions we used had been used in previous epidemiologic studies in Norway, they nonetheless were self-report questions and targeted broadly defined disorders. Direct diagnostic interviews may have yielded richer diagnostic information; however, given the magnitude of the sample, this was not practical. Second, although our frequency criteria for binge eating and purging differed from current DSM criteria, we emphasize that the established

criteria have not been empirically supported (Sullivan et al., 1998). Third, women may have been hesitant to admit to eating disorders behaviors during pregnancy which would have led to an underestimation of continuation rates and an overestimation of remission. Fourth, BMI measures were self-report rather than measured—a common approach given the logistical complications and cost of measured weight in large samples such as MoBa. Although there are no comparable data from Norway, data from other large samples suggest a correlation of 0.94 between measured and self-reported height and 0.98 between measured and self-reported weight for women (Bulik et al., 2001). Fifth, 42% of women invited agreed to participate in MoBa. Although this response rate is low, it is not uncommon to large epidemiologic studies and does not necessarily imply a biased sample (Hartge, 2006). The determination of bias is complex and would occur if and only if individuals who chose not to participate in this study differed markedly from those who did participate on the variables of interest. Initial comparisons of the MoBa cohort to the Norwegian population indicates lower rates of preterm birth (7.2% vs 7.7%) and low birth weight (< 2500g) (4.6 % vs 5.1%) possibly reflecting a socioeconomic gradient associated with participation (Magnus et al., 2006). Further exploration of this using education data suggests that the MoBa participants may be somewhat more educated than the general Norwegian population with 58% attending some form of college in comparison to 46% of women between 25-29 years and 43% of women between 30-39 years reported to have higher education in 2005 by Statistics Norway. Finally, our data reflect changes in eating disorders symptomatology in the early stages of pregnancy. It is possible that such changes may not reflect patterns of remission, continuation, and incidence across the entire gestational period.

Nonetheless, our results point to additional questions and cautions. Using data that are currently being collected, in future papers we will explore personality and psychiatric features that are associated with remission, continuation, and incidence of eating disorders during pregnancy. Clinically, given that some data suggest that women who have eating disorders are unlikely to report them to their obstetricians (Lemberg and Phillips, 1989), our results suggest that physicians, nurses, and midwives working with pregnant women should be alert to the possibility of enduring and emergent eating disorder symptoms. For women with known eating disorders, the onset of pregnancy does not necessarily ensure discontinuation of symptoms, and regular follow-up regarding persistence of symptoms should be considered. Moreover, as part of routine prenatal inquiries about nutrition, health care providers should also ask about the presence of binge eating. As we do not yet know the impact of binge eating during pregnancy, detection is a minimum first step that could facilitate referral for appropriate intervention if clinically indicated.

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Figure 1.

Table 1
Demographic Characteristics of 41,157 women.

Maternal Age	
<24	5,106 (12.4%)
25-29	14,099 (34.3%)
30-34	15,378 (37.4%)
35+	6,574 (16.0%)
Total Number of Previous Live Births	
0	16,603 (47.6%)
1	13,747 (33.4%)
2+	7,807 (19.0%)
Number of Children Who Live with the Mother	
0	18,750 (45.6%)
1	13,889 (33.7%)
2	6,827 (16.6%)
3+	1,691 (4.1%)
Marital Status	
Married	20,388 (50.2%)
Cohabiting	18,894 (46.5%)
Unmarried/single	1,208 (3.0%)
Divorced/Separated/ Widowed	99 (0.2%)
Other	65 (0.2%)
Maternal Education	
<3 years high school	3,908 (10.0%)
Vocational high school	5,778 (14.8%)
3-year high school general studies, junior college	6,578 (16.9%)
Regional technical college/4-year university degree	15,617 (40.1%)
University, technical college, more than 4 years	7,032 (18.1%)
Combined Minimum Income	
0-\$33 (200,000NOK)	3,622 (9.6%)
>\$33 (200,000NOK)	21,882 (57.9%)
>\$82 (500,000NOK)	8,069 (21.4%)
>\$114 (700,000 NOK)	4,214 (11.2%)
Native Language of Mother	
Norwegian	38,049 (94.7%)
Other	2,146 (5.3%)
Ever Smoked	
Yes	20,424 (50.0%)
Smoking < 3 months before pregnancy	
Yes	13,024 (32.0%)
Smoking during pregnancy	
Yes	4,538 (11.1%)

Ever Drank Alcohol	
Yes	38,590 (95.2%)

Table 2

Prevalence of Eating Disorders Before and During Pregnancy

Disorder	Before pregnancy N (%)	During Pregnancy N (%)	Before and During Pregnancy N (%)
Anorexia nervosa (broad)	37 (0.1%)	--	--
Bulimia nervosa (any type) *	290 (0.7%)	96 (0.2%)	67 (0.2%)
Bulimia nervosa (purging)	118 (0.3%)	52 (0.1%)	37 (0.1%)
Bulimia nervosa (non-purging)	109 (0.3%)	30 (0.1%)	17 (< 0.1%)
BED (broad)	1,405 (3.5%)	1,856 (4.8%)	779 (2.0%)
EDNOS-P	42 (0.1%)	12 (< 0.1%)	5 (< 0.1%)

* BN any type also includes those individuals who could not reliably be categorized as purging or non-purging due to missing data.

Table 3
Age adjusted rates of remission, continuation, and incidence in 1,000 person-weeks (95% CI).

Pattern	Bulimia nervosa (any type)	Bulimia nervosa (purging)	Bulimia nervosa (nonpurging)	BED (broad)	EDNOS-P*
Remission**	17.4 (14.09,21.43)	19.4 (14.33,26.28)	14.8 (10.54,20.87)	20.3 (18.83,21.79)	41.5 (42.30,55.68)
Partial Remission***	18.0 (14.69,21.89)	9.9 (6.21,15.60)	26.3 (21.26,32.59)		
Continuation	16.0 (13.04,19.72)	20.1 (15.43,26.16)	8.3 (5.04,13.60)	32.2 (30.73,33.67)	5.8 (1.86,18.19)
Incidence	0.03 (0.02,0.04)	0.01 (0.00,0.02)	0.01 (0.01,0.03)	1.12 (1.04,1.20)	0.01 (0.00, 0.02)

* EDNOS-P and incidence calculations except for BED not age adjusted due to small sample size.
** Remission indicates rate of no eating disorder at time of survey completion during pregnancy.
*** Partial remission in BN indicates absence of compensatory behaviours during early pregnancy