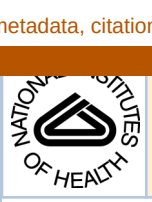

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Birth Outcomes in Women with Eating Disorders in the Norwegian Mother and Child Cohort Study (MoBa)

Cynthia M Bulik, Ph.D.[1,2], **Ann Von Holle, M.S.**[1], **Anna Maria Siega-Riz, Ph.D., R.D.**[2,3], **Leila Torgersen, Ph.D.**[5], **Kari Kveim Lie, M.D.**[6], **Robert M. Hamer, Ph.D.**[1], **Cecilie Knoph Berg, M.A.**[4], **Patrick Sullivan, M.D., FRANZCP**[1,4], and **Ted Reichborn-Kjennerud, M.D.**[5,7,8]

[1]Department of Psychiatry, University of North Carolina at Chapel Hill

[2]Department of Nutrition, University of North Carolina at Chapel Hill

[3]Department of Epidemiology, University of North Carolina at Chapel Hill

[4]Department of Genetics, University of North Carolina at Chapel Hill

[5]Division of Mental Health, Norwegian Institute of Public Health

[6]Division of Epidemiology, Norwegian Institute of Public Health

[7]Department of Psychiatry, University of Oslo, Norway

[8]Columbia University, Department of Epidemiology, New York, New York

Abstract

Background—We explored the impact of eating disorders on birth outcomes in the Norwegian Mother and Child Cohort Study (MoBa).

Method—35,929 pregnant women in the MoBa included women with broadly defined anorexia nervosa (AN; n=35), bulimia nervosa (BN; n=304), binge eating disorder (BED; n=1,812), and EDNOS-purging type (EDNOS-P; n=36) in the six months prior to or during pregnancy and the referent group--women who reported no eating disorders (no-ED; n=33,742).

Results—Pre-pregnancy BMI was significantly lower in mothers with AN and higher in mothers with BED than the referent. Mothers with AN, BN, and BED reported greater weight gain during pregnancy and more mothers with eating disorders reported smoking during pregnancy than the referent. Women with BED had higher birth weight babies, lower risk of small for gestational age babies, and higher risk for large for gestational age babies and cesarean section than the referent.

Conclusions—BED influences birth outcomes. The absence of differences in birth outcomes in women with AN and EDNOS-P may reflect small sample size and differential severity of illness in population versus clinical samples. The detection of eating disorders in pregnancy could help identify modifiable factors (e.g., binge eating, smoking) that could influence birth outcomes.

Studies based on both clinical and community samples reveal substantial adverse effects of some eating disorders on the pregnant woman and her unborn child.[1–11] Higher rates of miscarriages[1, 2] have been noted in both women with anorexia nervosa (AN)[3] and bulimia nervosa (BN)[9] and higher rates of cesarean section deliveries have been noted in AN.[1, 2] Infants of women with eating disorders have been reported to have greater likelihood of stillbirth, low birth weight, low Apgar scores, breech presentation, and cleft lip and palate.[4–10]

Correspondence to: Dr Bulik, Department of Psychiatry, University of North Carolina at Chapel Hill, 101 Manning Drive, CB #7160, Chapel Hill, NC 27599-7160, Voice: (919) 843 1689 Fax: (919) 966-5628, cbulik@med.unc.edu.

In a population-based study using data from the Avon Longitudinal Study of Parents and Children (ALSPAC), Micali et al.[9] reported significantly lower birth weights in offspring of women with AN; however, this effect was primarily accounted for by lower pre-pregnancy BMI. Danish registry data, which identified women based on prior hospitalizations for eating disorders and presumably indexing more severe cases, suggest that eating disorders are associated with low-birth weight, pre-term delivery, and small for gestational age (SGA) infants.[11] Kouba et al.[10] identified nulliparous non-smoking women with and without histories of AN and BN in prenatal clinics and found lower birth weight, smaller head circumference, and greater risk for microcephaly and SGA among offspring of women with eating disorders. However, this study did not control for confounding of maternal variables or for multiple comparisons. Collectively, these data raise considerable concern regarding AN and BN, yet several questions remain unanswered. Among these are the effect of eating disorders beyond AN and BN on birth outcomes and specifically, the impact of BED which we have shown to both persist and onset during pregnancy.[12]

We extend the existing body of research by exploring birth outcomes among women with and without eating disorders in the initial 35,929 women entered into the Norwegian Mother and Child cohort (MoBa). Four eating disorders presentations were defined as: broadly defined AN, BN, binge eating disorder (BED), and the purging subtype of eating disorders not otherwise specified (EDNOS-P) relative to the referent group comprising women with no eating disorders (no-ED). Based on previous investigations, we hypothesized a “cycle of risk” in AN[13] in which AN would be associated with a greater risk of preterm birth, SGA, lower gestational weight gain, and lower birth weight. Moreover, given the association between BED and obesity, we predicted elevated risk for greater gestational weight gain, higher birth weight, and large for gestational age (LGA) infants.

METHOD

Participants

Data collection was conducted as part of the Norwegian Mother and Child Cohort Study (MoBa) conducted by the Norwegian Institute of Public Health.[14] The study has been approved by the appropriate regional committees for ethics in medical research and the Norwegian National Data Inspectorate. Briefly, 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 and fourth questionnaires, 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 of Norway (MBRN)[15] to capture pregnancy outcome variables.

The current study is based on version 3 of the quality-assured data files released in 2007. The analysis population for this report included MoBa participants who: a) had information from MoBa Questionnaires 1 and 4 and the MBRN, b) gestational age between 20 and 44 weeks, c) gestational weight gain between -10 and 100 kg, d) non-missing weight value for Questionnaire 4, e) did not complete an early pilot version of Questionnaire 1 (n=2,599), d) had valid values for self-reported age, weight, and height, f) returned Questionnaire 1 before delivery, g) had a singleton birth, and h) had a non-missing eating disorder subtype before pregnancy. If a woman enrolled in MoBa more than once (due to additional pregnancies), only the first pregnancy was included. Of the initial 74,200 mother-child records reported in MoBa, 35,929 (48%) met the 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 (MBRN)—The MBRN was established in 1967.[15] Information on pregnancy, delivery and health of the neonate are reported to MBRN for all deliveries after 16 weeks of gestation through mandatory notification by midwives and doctors.[15] The Norwegian Mother and Child Cohort study is linked to MBRN through the personal identification number. Of all MoBa pregnancies, 4.5% did not have a MBRN record and were excluded. Data from the MBRN have been used for prior biomedical research.[16–18]

Eating Disorders—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[19–23] and were designed in accordance with DSM-IV criteria for AN, BN, and EDNOS.[24] In the analysis population, respondents completed Questionnaire 1 at a median of 18.4 weeks gestation (inter-quartile range 17.1–20.3 weeks and range 6.0–42.0 weeks).

Diagnostic algorithms and hierarchies were constructed from the questionnaire items to define the presence of eating disorders in the six months prior to pregnancy and/or during pregnancy. Our final categories included: broadly defined AN, defined as meeting DSM IV criteria for AN (with the exception of amenorrhea); broadly defined BN, endorsing at least weekly frequency of binge eating and either purging (vomiting, laxatives) or non-purging (exercise, fasting) compensatory behaviors; broadly defined 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 the feeling of loss of control. Purging was assessed specifically to be differentiated from nausea and vomiting of pregnancy. As the symptoms picture for many women changed in the interval before pregnancy and during pregnancy, the order for our diagnostic hierarchy was: AN, BN, EDNOS-P, BED, and no-ED. All individuals who met AN criteria before pregnancy were categorized as AN regardless of presentation during pregnancy. Those who met BN criteria either before or during pregnancy and who did not meet AN criteria prior to pregnancy were categorized as BN. If not classified as AN or BN, those who met criteria for EDNOS-P before or during pregnancy *and* did not endorse binge eating at either time were categorized as EDNOS-P. Similarly, individuals who endorsed BED and did not endorse purging during or before pregnancy were included in the BED group. Group assignment was only made when all responses were available to ensure accurate classification.

Outcomes and Covariates

Self-reported weight and height were used to calculate pre-pregnancy body mass index (BMI, kg/m^2) and BMI at the time of assessment. Weight gain during pregnancy was calculated using maternal reported weight before pregnancy from Questionnaire 1 subtracted from weight at birth as indicated in Questionnaire 4 (after pregnancy).

Information obtained from MBRN included pregnancy complications (gestational diabetes, pregnancy-induced hypertension, pre-eclampsia, HELLP syndrome (hemolysis, elevated liver enzyme levels, low platelet count), vaginal bleeding, placenta praevia, fetal “lie” (breech and non-vertex cephalic presentation), mode of delivery (induction of labor by medication or amniotomy, caesarean section, use of general anaesthesia and epidural anaesthesia), slow progress in labor, birth weight, length of gestation, and Apgar scores after one and five minutes.

Preterm birth was defined as length of gestation less than 37 weeks. Small for gestational age (SGA) and large for gestational age (LGA) were calculated from MBRN variables using an algorithm stratifying by gestational age and sex of baby[25]. The birth weight from the MBRN was standardized using the mean and standard deviation corresponding to the stratum as determined by.[25] This z-score then was assigned a percentile according to the standard normal distribution. If the percentile exceeded the 90th percentile then the baby was classified as LGA and if the percentile was below the 10th percentile then the baby was classified as SGA. Outcomes were designated as primary (birth weight, preterm birth, SGA, LGA, preeclampsia, caesarean, gestational age Apgar scores, slow progress) and secondary (anesthetics at birth, assisted vaginal breech presentation, HELLP, non-vertex cephalic presentation, placenta previa).

Covariates included self-reported smoking during pregnancy (coded as a dichotomous variable), household income, education, parity (defined as total number of live births), and maternal age. With the exception of maternal age, which was from the MBRN, all covariates originated from the first MoBa maternal Questionnaire. Although it is known that maternal pre-pregnancy weight and maternal weight gain influence birth outcomes,[26–28] given the complete confounding of pre-pregnancy BMI with eating disorders diagnostic status, we treated these variables as outcome measures rather than covariates in the analyses.

Statistical Analysis

To determine differences in pregnancy outcomes across eating disorder subtypes, a series of nested models estimated eating disorder subtype effects with and without adjustment for covariates. False discovery rate[29] control for each model was employed to avoid type I error inflation. The majority of outcomes were dichotomous and served as the response variables in separate univariate Poisson regressions. A Poisson regression was used to estimate relative risk and 95% confidence intervals (CIs) of birth outcomes by eating disorder subtype with and without adjustment. This method provides direct estimates of relative risk unlike logistic regression and odds ratio estimates. Given evidence of under-dispersion of the data, in which the variance of the data is less than what is estimated in a standard Poisson regression, a quasi-likelihood approach with GEE was used to robustly estimate the variance of the relative risk estimates.[30–32]

We conducted a series of pre-planned models which systematically included additional covariates. This approach was taken in order to control for the impact of covariates that are known to influence birth outcome.[33–35] All models contain eating disorder subtype as the primary covariate to measure its effect on an outcome. The first model included eating disorder subtype as the primary covariate with no other covariates. This model allowed us to explore basic differences across eating disorder subtypes in comparison to the referent group without accounting for additional sociodemograhic or behavioral factors. The second model added five standard covariates known to influence birth outcome: maternal age, gestational age, parity, household income, and maternal education. These covariates were based on extant literature documenting the effects of these factors on birth outcome. The third model retained the covariates from the second model and added a behavioral covariate, smoking during pregnancy (coded as a dichotomy). This model was run given the known independent effects of smoking on birth outcomes[36] and the observed differences in maternal smoking across eating disorder subtypes and the referent group.

Assumptions for parametric tests were not met for birth weight, gestational age and Apgar score after one and five minutes making the series of nested models with adjustment infeasible. Given the unequal sample sizes between the no-ED group and eating disorder subtypes, there is the possibility of variance heterogeneity and type I error inflation.[37] Before conducting the nonparametric tests, population dispersion was assessed to evaluate

variance homogeneity between eating disorder subtype assumptions. The ratio of standard deviations between each eating disorder subtype and the no-ED referent group did not exceed 1.1 for any variable. The non-parametric tests precluded any adjustment as done in the parametric analyses.

All analyses were done using SAS/STAT® software for Windows and AIX (v9.1).[38]

RESULTS

Maternal characteristics

Table 1 presents maternal characteristics by eating disorders category. After multiple comparisons correction, significant differences emerged for several variables between eating disorders groups and the referent. Mothers with AN were significantly younger than the referent group. Prior to pregnancy, as expected based on diagnosis, self-reported BMI was significantly lower in mothers with AN than the referent group and significantly higher in mothers with BED than the referent group. Mothers in all eating disorders groups except EDNOS-P reported greater weight gain during pregnancy than mothers without eating disorders. Significantly more mothers with AN, BN, BED, and EDNOS-P reported smoking during pregnancy than the referent group.

Primary Birth and Pregnancy Outcomes

Table 2 presents means and standard errors or percents and Table 3 the statistical comparisons for the primary birth outcome variables (birth weight, weeks gestation, caesarean section, gestational diabetes, LGA, SGA, preeclampsia, preterm birth, slow progress, and Apgar scores at one and five minutes) across eating disorders subgroups.

Complete results from the Poisson regressions for Models 1–3 are presented in Figure 1 and in Tables 4–6 which are included as supplementary material to this manuscript online. In Model 1 (eating disorder subtype as the primary covariate with no other covariates), BED was associated with significantly higher birth weight than the referent group. Women with BED had significantly lower risk of having SGA infants than the no-ED referent group. The opposite pattern emerged for LGA babies, with BED associated with higher risk. No other significant differences emerged under Model 1.

In Model 2, we controlled for gestational age, maternal age, income, education, and parity. In this model, the observed differences for SGA and LGA between BED and the referent group remained. Unlike model 1, risk for cesarean section was significantly elevated in women with BED relative to the referent. The same pattern of results remained when smoking (Model 3) was added to the model as a covariate.

Secondary Birth Outcomes

In exploratory analyses, we ran the three models as described above for our secondary outcome variables: narcotics, assistance with vaginal breech presentation, epidural, HELLP, induction, non-vertex cephalic presentation, and placenta previa. Percents are presented in Table 2. In Model 1, women with BN had significantly higher rates for assistance at vaginal breech presentation and women with AN had greater risk of receiving an epidural. In Model 2 and Model 3, BN was associated with significantly greater risk of induction and assistance at vaginal breech presentation while AN and BED had greater risk of receiving an epidural than the referent group.

DISCUSSION

In this large population-based study, eating disorders in the six-months prior to or during pregnancy were associated with various adverse birth and pregnancy behaviors and outcomes compared to the referent group of women without eating disorders. As expected based on diagnosis, pre-pregnancy BMI differed across groups being lowest in women with AN and highest in women with BED—both significantly different from women with no eating disorder. In addition, with the exception of women with EDNOS-P, all women with eating disorders gained significantly more weight during pregnancy than the referent group. What remains unclear is whether the factors underlying the elevated weight gain differed across eating disorders groups. Women with AN started at a lower pre-pregnancy weight and therefore had more weight to gain to reach healthy weights during pregnancy. Higher weight gain in women with BN and BED may have been more related to the nature of their food intake. We have shown previously that binge eating persists in many women with BN and BED during pregnancy[12] and, perhaps relatedly, that women with BED both before and during pregnancy had higher intakes of total energy and higher total mono-saturated and saturated fat than women without eating disorders.[39] Thus, increased weight gain in mothers with AN relative to the referent may be appropriate given their lower pre-pregnancy weight, whereas increased weight gain in BN and BED women relative to the referent may be more-related to intake differences that reflect ongoing eating pathology.

In addition all women with eating disorders were more likely to smoke during pregnancy than the referent women. The frequency of smoking during pregnancy amongst eating disordered women ranged from 14 – 37% compared to 9% in women with no eating disorder. Somewhat surprisingly given previous reports of smoking across eating disorder subtypes,[40] numerically the highest percentage was among women with AN. Although we had initially hypothesized in previous studies that smoking in eating disorders would be more commonly associated with desire for weight and appetite control, we found higher scores on scales of nicotine dependence in women with eating disorders suggesting that giving up smoking may be more difficult for these women, even during pregnancy. [40]

Our modeling approach for both primary and secondary outcome variables began with an uncontrolled model reflecting differences across eating disorder subtypes and the referent group which was followed by two additional models controlling for covariates. Our baseline uncontrolled models revealed significant differences in the primary outcome variables indexing offspring birth weight. On the continuous birth weight measure, women with BED had significantly heavier babies than the referent group. On the dichotomous SGA and LGA variables, women with BED had higher risk of LGA and lower risk of SGA infants. This pattern largely remained in subsequent controlled models with the results for LGA and SGA remaining significant even under controlled conditions. In addition, when controlling for relevant covariates, cesarean sections became significantly elevated in women with BED.

The modeling of the secondary outcome variables with covariates suggested some possible indices of birth difficulties with BN women having significantly greater risk of induction and assistance at vaginal breech presentation while AN and BED had greater risk of receiving an epidural than the referent group. As these variables are simply recorded on the Medical Birth Registry, greater detail regarding the circumstances at the time of delivery was not available.

The observed pregnancy outcome in women with BED is a new addition to the literature. In terms of anorexia nervosa, our failure to confirm our “cycle of risk” hypothesis in AN may reflect both sample size and statistical power issues as well as the population-based the nature of our sample. In the population-based study of Micali et al.,[41] they reported lower

birth weight among offspring of women with AN, which was accounted for by pre-pregnancy weight differences, although their average pre-pregnancy BMI for women with AN was higher than observed in AN women in the MoBa. Kouba et al. [10] also reported low birth weight and SGA, but their results were uncorrected for covariates or multiple comparisons. Sollid et al.[11] reported greater risk of low-birth weight, pre-term delivery, and SGA amongst women who had been hospitalized for an eating disorder. These latter two studies did not present pre-pregnancy BMI. One obvious difference across these four studies is the method of case definition. We focused on maternal self-report of eating disorders in the six months prior to and during pregnancy based on symptom-level self-report questions. Micali et al. asked women "Have you ever had anorexia nervosa" and defined their groups accordingly, yielding a higher percentage of women with AN than observed in our larger cohort. Sollid et al.[11] relied on hospitalization records, and although they were unable to make finer eating disorder subgroup distinctions, the fact that women had required hospitalization suggests that theirs might have been a more severely ill sample. The observed differences in outcome across studies may reflect the severity of the eating disorders in each sample, with the MoBa reflecting a population-based sample, and the Kouba sample reflecting a clinical population. Indeed, it has been shown that AN exists on a continuum of severity in population based samples.[42] Thus, although our hypotheses regarding a "cycle of risk" were not confirmed in this population-based study, they may not be false, but may only apply to a more severely ill group of women. This gradient of severity is not unexpected given well-documented differences in severity and comorbidity across population and clinic-based investigations.[43–46] Therefore, the magnitude of the impact of AN on birth outcomes maybe proportional to the severity of the illness in the mother.

Our observation of elevated cesarean rates only in BED also differ from previously reported studies on clinical samples which identified higher rates of cesarean sections in women with AN,[1, 2] although, previous studies did not include women with BED and focused primarily on AN and BN. In the MoBa cohort, risk was only elevated in women with BED and was numerically lower in women with AN than the referent group. Again, this could reflect the severity of the AN group as well as cross-national trends in cesarean rates.

Our birth weight results may also have been influenced by demographics of Norway in general and of the MoBa cohort in particular. In 2005, the mean birth weight in Norway was 3,521g. The mean birth weight of the referent group in this MoBa study was 3,613 g and overall birth weight in the MoBa tends to be higher than in the general Norwegian population.[14] Indeed, the birth weights of the babies of women with AN in the MoBa were higher than the mean birth weight of the referent population in the United Kingdom study,[9] suggesting that the community-identified cases of AN in the MoBa may be less severe than those identified either in the UK cohort or through clinic-based strategies.

In the present study, we controlled for several relevant maternal and sociodemographic characteristics known to influence birth outcomes. Although both maternal pre-pregnancy weight and gestational weight gain are known to influence birth outcomes, our study placed us in a unique situation. First, maternal pre-pregnancy weight is entirely confounded with our diagnostic groupings and we therefore opted to include diagnostic subgroup as a covariate in all models. Second, our analytic plan treated gestational weight gain as an outcome variable rather than a covariate. Given that these variables are confounded with diagnostic subtype in this study, including them as covariates in our analyses would have yielded an overcontrolled model. As evidenced in our data, eating disorders are an important source of pre-pregnancy weight variation in women with, on one end of the continuum, AN and related conditions contributing to low pre-pregnancy BMI, and on the other end of the continuum, BED and related conditions contributing to higher pre-pregnancy BMI.

The strengths of this investigation are several. First, MoBa represents largest population-based study of the impact of eating disorders on birth outcome to date. Second, our ability to link the MoBa data to the Norwegian Medical Birth Registry ensures high quality data that are equivalent across sites in Norway. Third, the high retention rate of mothers across waves of assessment yields low drop-out rates for the cohort and reduces potential sources of participant bias.

Although our study has considerable strengths, limitations must also be considered. First, our diagnostic questions and questions about weight gain during pregnancy and pre-pregnancy BMI were based on maternal self-report. Although we used questions that reflected DSM criteria, direct interviews may have yielded richer diagnostic information and more accurate weight data. Second, we employed criteria for binge eating and purging which differ from current DSM criteria; however, the established criteria have not been empirically supported and continue to be questioned[47]. Third, 42% of women invited agreed to participate in MoBa. Although this response rate is low, it is typical for large epidemiologic studies and does not necessarily imply a biased sample.[48] Initial comparisons of participants versus nonparticipants suggest that MoBa participants have 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.[14] MoBa participants may also be somewhat more educated than the general Norwegian population.[12] Moreover, given the considerable effort required to participate in the various waves of the MoBa protocol, the women with eating disorders who do participate may represent the healthier end of the eating disorder severity spectrum.

Our pattern of results indicate possible self-selection effects with individuals with less severe eating disorders participating and relatively low prevalence of some eating disorder subtypes (e.g. AN and EDNOS-P) represented in the cohort. This selection could result in diminishing effect sizes not originally expected based on prior clinical literature with more severe cases. Combined, these two characteristics create a situation in which an acceptable level of power, at least 0.8, is difficult to achieve. Simulation studies done with parameter estimates obtained from current analyses (results not shown) indicate power far below 0.8 with an estimated sample size approximately double the current level, at 60,000 individuals. These results underscore the challenges in measuring eating disorder effects for certain outcomes that although not previously addressed in the literature, are important contributions to the knowledge base. Future analyses will include more qualitative approaches to evaluating birth outcomes in the smaller eating disorder subtypes to understand the impact of illness severity on birth outcomes.

With the backdrop of these strengths and limitations, our results are the first to suggest that BED is associated with significant differences in birth outcome variables indexing offspring weight parameters. Given increasing awareness of the prevalence of BED[49] and adverse health consequences[23, 50], and our previous observation that pregnancy may represent a risk window for incident BED,[12] screening for eating disorders in general, and BED in particular during pregnancy is warranted As additional data are collected from the MoBa cohort, we will be well-positioned to detail longitudinal effects of BED (and other eating disorders) on growth and development of offspring.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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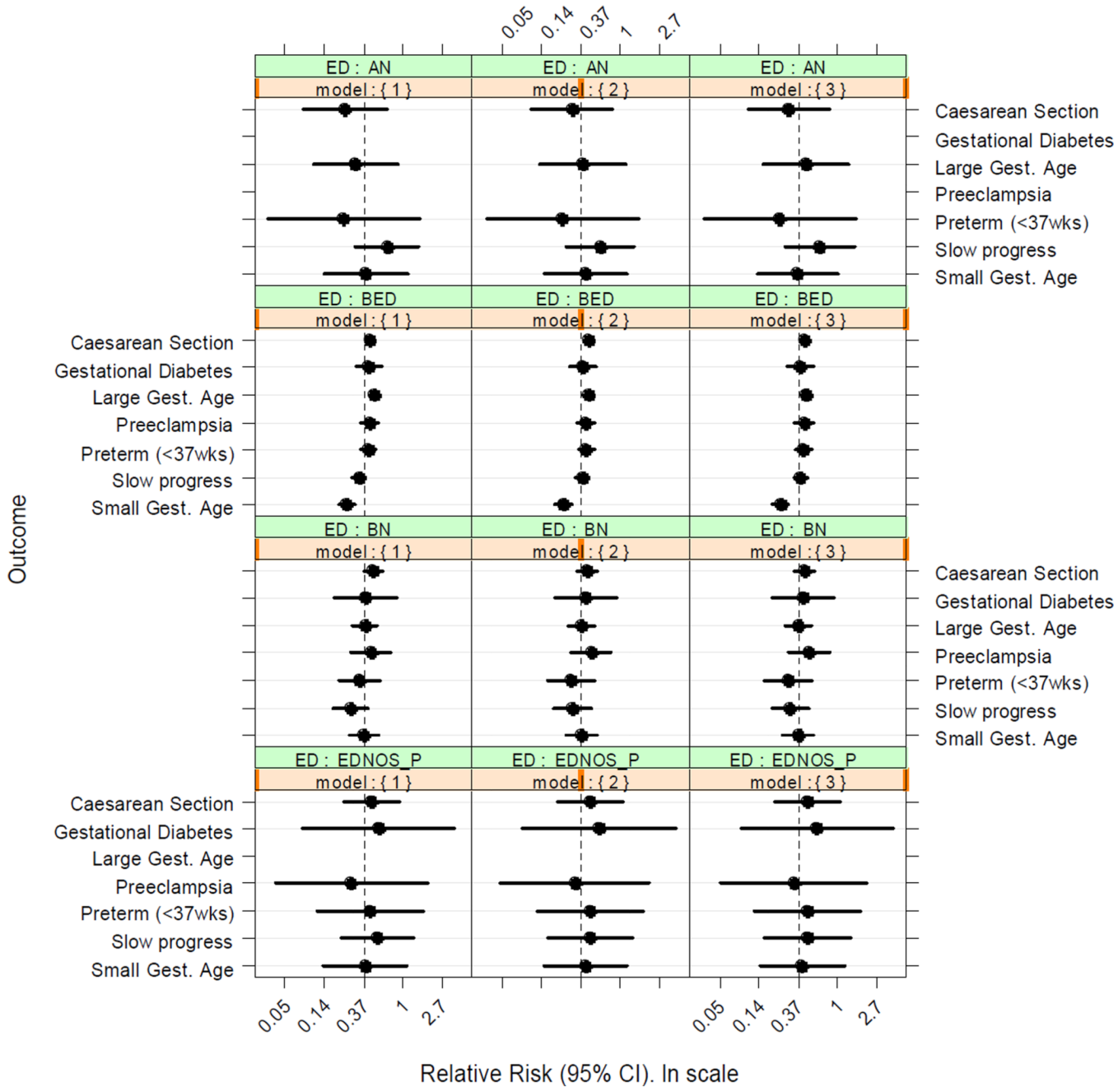

Figure 1.
Relative Risk of Outcome by ED subtype (versus non-ED).

Table 1

Maternal characteristics across eating disorders subtypes presented as Mean (SE) or percent (n)*.

	AN (N=35)		BN (N=304)		BED (N=1,812)		EDNOS-P (N=36)		No ED (N=33,742)
	Mean (SE)	FDR p	Mean (SE)	FDR p	Mean (SE)	FDR p	Mean (SE)	FDR p	Mean (SE)
Maternal age (years)	26.6 (0.83)	<0.001	29.8 (0.28)	0.55	30.0 (0.11)	0.07	28.4 (0.83)	0.36	29.9 (0.02)
BMI Before Pregnancy (kg/m^2)	18.2 (0.10)	<0.001	24.4 (0.26)	0.50	25.9 (0.12)	<0.001	23.7 (0.68)	0.55	24.0 (0.02)
Weight gain during pregnancy (kg)	17.8 (0.97)	0.01	16.8 (0.42)	<0.001	16.6 (0.17)	<0.001	16.3 (1.15)	0.52	14.9 (0.03)
Smoking during pregnancy (n) % yes	(13) 37.1%	<0.001	(46) 15.1%	<0.001	(251) 13.9%	<0.05	(7) 19.4%	<0.001	(3,096) 9.2%
Total # live births	0: 68.6% 1: 22.9% 2+:8.6		0: 47.4% 1: 33.6% 2+: 19.1%		0: 44.2% 1: 36.1% 2+: 19.8%		0: 61.1% 1: 22.2% 2+:16.7%		0: 51.1% 1: 32.1% 2+:16.9%

*BMI and weight failed to meet ANOVA model assumptions so a non-parametric Wilcoxon was conducted. Smoking was a dichotomous response (y/n) and a Poisson regression was conducted.

Table 2

Birth outcomes across eating disorder subtypes reported in mean (SE) or %.

	AN (N=35)	BN (N=304)	BED (N=1,812)	EDNOS-P (N=36)	No ED (N=33,742)
Primary Outcome Variables					
Birth weight (g)	3,553 (79.7)	3,580 (30.7)	3,685 (13.1)	3,535 (71.9)	3,609 (3.0)
Weeks gestation	39.9 (0.22)	39.8 (0.09)	39.9 (0.04)	40.1 (0.24)	39.9 (0.01)
Preterm birth (%<37 wks)	2.9%	4.3%	5.4%	5.6%	4.7%
SGA -- <10% (%)	8.6%	8.2%	5.2%	8.3%	8.0%
LGA -- >90% (%)	8.6%	10.9%	13.6%	0%	10.4%
Preeclampsia (%)	0%	4.6%	4.5%	2.8%	3.8%
Caesarean (%)	8.6%	17.4%	15.8%	16.7%	13.6%
Gestational diabetes (%)	0%	2.0%	2.2%	2.8%	1.9%
Apgar score at 1 minute	8.9 (0.13)	8.7 (0.07)	8.7 (0.03)	8.8 (0.18)	8.7 (0.01)
Apgar score at 5 minutes	9.5 (0.10)	9.4 (0.05)	9.4 (0.02)	9.6 (0.10)	9.40 (0.00)
Slow progress (%)	14.3%	5.6%	7.0%	11.1%	7.7%
Secondary Outcome Variables					
Anaesthetics -- Narcotics	0%	3.9%	2.8%	5.6%	2.8%
Assistance at vaginal breech presentation	0%	2.6%	0.6%	2.8%	0.9%
Epidural	48.6%	28.0%	28.2%	38.9%	26.4%
HELLP	0%	0%	0.2%	0%	0.2%
Non-vertex cephalic	5.7%	10.9%	9.3%	11.1%	8.9%
Placenta previa	0%	0.3%	0.1%	0%	0.2%

Table 3

Primary Outcome Differences Across Eating Disorder Subtypes versus the non-ED group*.

	Model 1 (Covariate: ED subtype)		Model 2 (Covariates: add gestational age, maternal age, income, education, parity)		Model 3 (Covariates: add smoking)	
	FDR_p	**Pattern**	**FDR_p**	**Pattern**	**FDR_p**	**Pattern**
Birth weight **	<0.001	BED>REF				
Weeks gestation **		--				
Preterm birth (<37 wks)		--		--		--
SGA	<0.001	BED <REF	0.003	BED<REF	0.002	BED<REF
LGA	<0.001	BED>REF	0.02	BED>REF	0.02	BED>REF
Preeclampsia		--				--
Caesarean			0.02	BED>REF	0.02	BED>REF
Gestational diabetes		--		--		--
Apgar 1 min **		--				
Apgar 5 min **		--				
Slow progress		--		--		--

* With the exception of the non-parametric tests, all differences refer to outcome risk ratios.

** Non-parametric test statistic sources indicating a location shift between groups.