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Mood changes during pregnancy and the postpartum period: development of a biopsychosocial model

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Abstract

Objective: Women are vulnerable to mood changes during pregnancy and the postpartum period. We set out to empirically test the hypothesis that biological and psychosocial variables interact to result in this vulnerability.

Method: Using structural equation modeling techniques, we developed an integrative model of perinatal mood changes from clinical, psychosocial, hormone and mood data collected from 150 women in late pregnancy and at 6-weeks postpartum.

Results: In the prenatal model, biological variables had no direct effect on depressive symptoms. However, they did act indirectly through their significant effects on psychosocial stressors and symptoms of anxiety. The same model did not fit the postpartum data, suggesting that different causal variables may be implicated in postpartum mood.

Conclusion: This model demonstrates the importance of considering both biological and psychosocial variables in complex health conditions such as perinatal mood disorders.

After more than two decades of research, it is now clear that the risk for affective disorders in women is at least as high, if not higher, during the perinatal period as at other times (1–3). While many researchers have hypothesized that changes in sex hormone concentrations that occur during pregnancy and the postpartum period contribute to this vulnerability (4–6), other lines of evidence have pointed to key roles for non-biological risk factors such as lack of social support, relationship difficulties, and stressful life events (7, 8). Most researchers and clinicians agree that perinatal mood changes are probably best accounted for by a combination of biological and psychosocial risk factors (9); however, this hypothesis has yet to be empirically tested.

In order to study the effects of biopsychosocial interactions in psychiatry, powerful statistical techniques are required which are capable of detecting and quantifying subtle and indirect effects of variables. Structural equation modeling (SEM) allows for simultaneous testing of both direct and indirect effects of multiple variables by examining their patterns of variances and covariances (10). SEM techniques are widely used in the behavioural and social sciences (11). In recent years, SEM has also been used to examine relationships among putative causes of psychiatric conditions (12) including depression during pregnancy and postpartum (13). To our knowledge, Kendler and colleagues (12) were the first to use SEM to test relationships between biological (specifically, genetic) and non-biological risk factors in a psychiatric condition (12). However, to our knowledge, no published studies have made use of SEM techniques in examining the interrelationships between other biological (e.g. hormonal) and environmental factors in mental health conditions, particularly during the perinatal period.

Aim of the study

In this study, we sought to develop a biopsychosocial model of perinatal mental health by quantifying the complex interactions between biological and non-biological determinants of perinatal mood using SEM techniques.

Material and methods

Participants

Participants were 150 obstetrical patients at the Women's College Campus of Sunnybrook and Women's College Health Sciences Centre (SWCHSC), Toronto, Canada, or at other area hospitals or midwifery clinics. Two recruitment strategies were used: flyers were posted around hospital buildings and community services for pregnant women; and, for SWCHSC patients, patients waiting for prenatal appointments in their obstetrician's offices were invited to participate. All women were at least 18 years of age, in good physical health, and

which written informed consent was obtained. All study procedures were approved by the SWCHSC Research Ethics Board. Of the 150 participants, none withdrew from the study.

The prevalence of clinical depression and depressive symptoms in this sample was in the range reported in studies of other community samples (1). Using scores on the Edinburgh Postnatal Depression Scale (EPDS) (14) as indicators of depressive symptoms, 19 (12.8%) women scored over the recommended cut-off score of 12 at both the end of the third trimester and at 6-weeks postpartum. The sample described here was 86% Caucasian, 93% married or in an analogous stable relationship, 61% primiparous, and 84% working outside of home prior to the maternity leave, with a mean age of 33.4 years.

Procedures

Participants were assessed prenatally between 36 and 42 weeks gestation, and postpartum at approximately 6 weeks following delivery. At the prenatal assessment, participants completed questionnaires assessing demographic variables and personal and family psychiatric history, and at both the prenatal and postpartum assessment, participants completed measures of marital adjustment, social support, life stress, and mood (see below). In addition, participants had a blood sample drawn for hormone analysis at both visits.

Theoretical model

The theoretical model tested in this work, shown in **Fig. 1**, was developed on the basis of previously published literature and our hypotheses about the relationships between variables likely to be determinants of mood changes during pregnancy and postpartum. The model tests the interrelationships between two classes of risk factors (Psychosocial Stress, Biological Risks) and two classes of symptomatology (Anxiety and Depression) using three to five indicator variables for each construct.

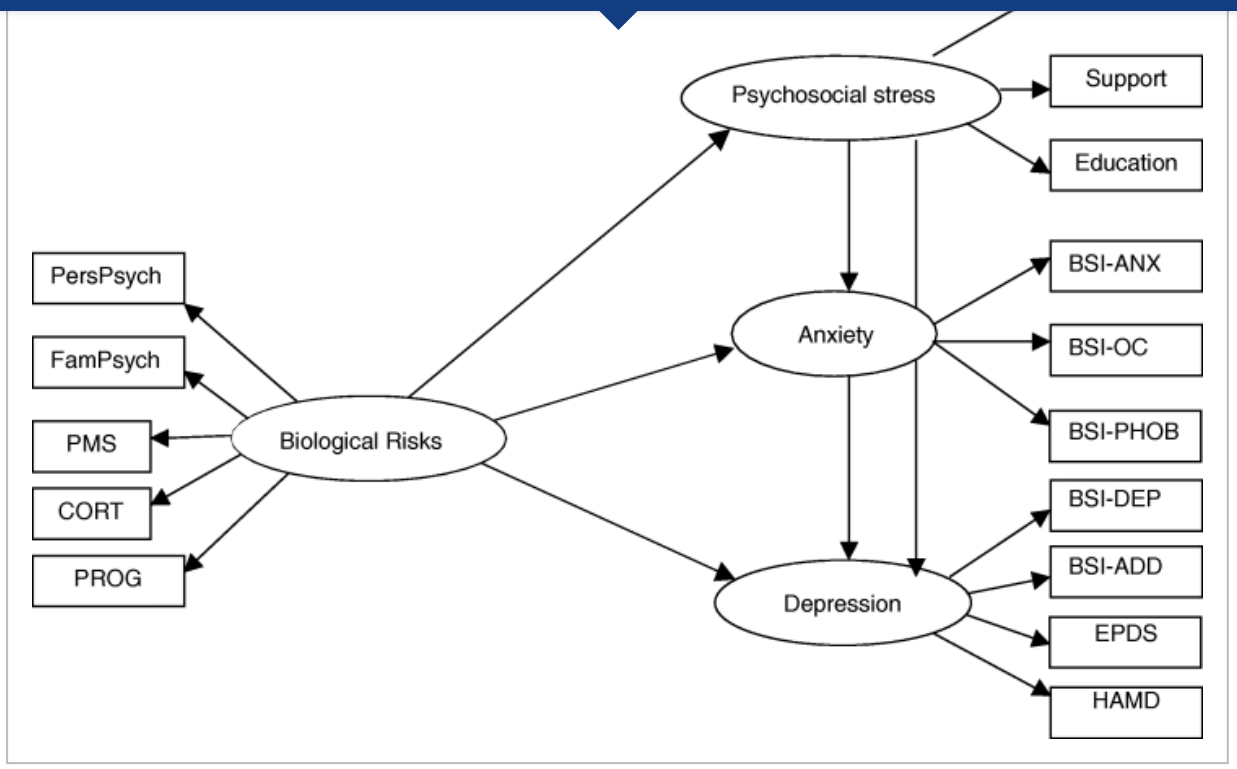

Figure 1

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Theoretical model of perinatal mood changes. Ovals represent latent (not directly measurable) variables, while rectangles represent observable indicator variables. Arrows represent the postulated relationships between these variables. Abbreviations: PersPsych: personal history of depression; FamPsych: history of depression or anxiety in first degree relatives; PMS: personal history of mood symptoms related to the menstrual cycle; CORT: plasma cortisol; PROG: plasma progesterone; BSI: Brief Symptom Inventory; ANX: anxiety subscale; OC: obsessive-compulsive subscale; PHOB: phobic anxiety subscale; DEP: depression subscale; ADD: additional items; EPDS: Edinburgh Postnatal Depression Scale; HAMD: Hamilton Rating Scale for Depression.

In developing this model, we first hypothesized that psychosocial stress would directly affect symptoms of depression, as has been demonstrated in previous literature (15–17). However, based on our finding of a high prevalence of anxiety symptoms in this population (18), together with findings of others that anxiety symptoms are closely related to depressive disorders in women (19–21), we also hypothesized that psychosocial stressors would exert part of their effects on depressive symptoms indirectly, through effects on anxiety.

The particular focus of this work, however, was to quantify the relationship between the 'Biological Risks' factor and the other variables in the model. On the basis of the research literature suggesting a relationship between ovarian hormones and depressive symptoms

hypothesized that biological factors would exert part of their effects indirectly, by mediating vulnerability to psychosocial stressors.

Model variables

Psychosocial stress factor. Demographic and psychosocial variables that were significantly associated with depression scores in univariate analyses of these data were considered for inclusion in the 'Psychosocial Stress' factor. Specifically, this included the following demographic variables, as assessed at the prenatal visit: relationship status (current stable relationship vs. no current stable relationship), highest level of education achieved (rated on a six point scale from 'high school incomplete' to 'LLB, MD, PhD or other professional degree completed'), household income (rated on a 7-point scale), and unplanned pregnancy (yes or no).

Scores on measures of stressful life events, relationship adjustment, and social support were also included as part of the 'Psychosocial Stress' factor. The list of threatening experiences (LTE) (24), a 12-question measure of stressful life events (e.g. serious illness or assault, death of close relative) was included as our stressful life events inventory. It has been demonstrated to have adequate reliability and validity, and in particular, during pregnancy (25). The test–retest reliability coefficient for at least one event occurring in the past 3 months is 0.88 (24). The Dyadic Adjustment Scale (DAS) (26), a widely used measure of marital adjustment, was used to quantify relationship satisfaction. Cronbach's alpha coefficient for the DAS has been reported to be 0.96 (26). Finally, the Medical Outcome Study Social Support Survey (MOS) (27) was chosen as the social support measure. The MOS is a brief self-report measure of availability of tangible support, affection, positive social interaction, and emotional or informational support. The survey has high internal consistency ($\alpha = 0.97$) and test–retest reliability of 0.78 (28).

In consideration of the relatively small sample size (see discussion below), the above variables were reduced into a smaller number of indicator variables using principal components analysis (PCA). In brief, PCA with promax rotation on the prenatal data revealed that three factors had eigenvalues ≥0.99, and these three factors accounted for 67% of the total variance in scores. DAS and MOS only loaded on factor 1 (factor loadings > 0.85), so this factor was termed 'Support'. On factor 2, LTE scores (factor loading $= 0.88$), relationship status (factor loading $= 0.57$), and income (factor loading $= 0.69$) loaded strongly, so this factor was termed 'Life Stress'. Finally, education (factor loading $= 0.83$) and unplanned pregnancy (factor loading $= 0.68$) loaded on factor 3, which was termed 'Education'. This procedure was repeated on the postpartum dataset, which included the same values for demographic variables as the prenatal dataset, but scores from the postpartum assessment for the DAS, MOS and LTE. Three factors were again extracted, with the same composition as for the prenatal data. Prenatal and 6-week postpartum factor

variables, resulting in a total of three indicator variables for 'Psychosocial Stress', as shown in Fig. 1.

Biological risks factor. Plasma progesterone and plasma cortisol concentrations were assayed for all 150 participants at both the prenatal and 6-week postpartum assessments. The values were obtained using a solid-phase competitive chemiluminescence commercial laboratory immunoassay (IMMULITE 2000). The coefficient of variation for the progesterone assay over three concentrations ranged from 12 to 16% with approximately 400 replications, and the coefficient of variation for the cortisol assay over three concentrations ranged from 11 to 15% with approximately 400 replications. We attempted to combine the values for plasma progesterone and plasma cortisol into one 'Hormonal Risks' factor; however, confirmatory factor analysis revealed that concentrations of these hormones were not correlated with one another, and as such could not be combined into one factor. As a result, these variables were combined into a factor together with psychiatric history variables; specifically, personal psychiatric history, history of premenstrual mood symptoms (PMS), and family psychiatric history. Family psychiatric history was a dichotomous variable: women were scored as 'yes' if they had either family history of depression or family history of anxiety, and 'no' if they had no such history. Personal psychiatric history and history of PMS were also coded as dichotomous yes/no variables. In total, there were five indicators (two hormonal + three psychiatric history) for the 'biological risks' factor. Hormone concentrations from the prenatal and postpartum assessments were used in the prenatal and postpartum datasets, respectively, while prenatal assessments of psychiatric history were used in both datasets.

Anxiety factor. Three subscales of the Brief Symptom Inventory (BSI), a 53-item self-report scale developed from the longer Symptom Checklist 90-Revised (29), were used as indicators of anxiety. The subscales used for this factor were the 'Anxiety' subscale (BSI–ANX, includes items related to feeling suddenly scared for no reason, feeling fearful, nervousness or shakiness inside, spells of terror or panic, feeling restless, and feeling tense or keyed up); the 'Obsessive Compulsive' subscale (BSI–OC, includes items related to feeling blocked in getting things done, having to check and double-check things, trouble remembering things, the mind going blank, trouble concentrating, and difficulty making decisions); and the 'Phobic Anxiety' subscale (BSI–PHOB, includes items about specific fears, e.g. feeling nervous when left alone, feeling afraid in open spaces or on the streets). Each item of the BSI is rated on a five-point scale of distress, ranging from 'not at all' (0) to 'extremely' (4) distressing over the past week. Internal consistency coefficients for these three subscales have been reported by the scale's authors as follows: BSI–ANX = 0.81, BSI–OC = 0.83, and BSI–PHOB = 0.77. Test–retest reliability coefficients for these subscales are also high, with reported values of BSI–ANX = 0.79, BSI–OC = 0.85, and BSI–PHOB = 0.91 (29). Prenatal scores on these subscales were used in the prenatal dataset, and postpartum scores in the postpartum dataset.

version of the Hamilton Rating Scale for Depression (HAMD) is a widely used observer-rated scale concerned primarily with the behavioural and somatic features of depression (30) which has been validated for use in perinatal populations (31). The HAMD was administered using Williams' Structured Interview Guide (32), either by a psychiatrist (M.K.R.), psychiatric nurse, or trained graduate student (L.E.R.). Although inter-rater reliability was not assessed in this study, test–retest reliability for the HAMD using the Structured Interview Guide has been reported to be 0.81, even among minimally trained raters from multiple disciplines (32). For self-report ratings of depressive symptoms, the EPDS (14), which was designed specifically for screening for perinatal depression in community populations, was chosen. The EPDS is a 10-item self-report scale with well-established sensitivity and specificity (33, 34), which has also been validated for use during pregnancy (35). Participants are asked to underline the response that is most true for them over the past 7 days, ranging on a four-point scale from 'No, not at all' (0) to 'Yes, most of the time' (3). The scale's authors report the EPDS to have split-half reliability of 0.88 and a standardized alpha coefficient of 0.87 (14). The final indicators for the 'Depression' factor were drawn from the BSI (29). Firstly, the BSI Depression subscale (BSI–DEP) was used, which includes items assessing anhedonia, feelings of hopelessness and loneliness, and other hallmark affective symptoms of depression. Secondly, the four Additional Items of the BSI (BSI–ADD), which include poor appetite, trouble falling asleep, thoughts of death or dying, and feelings of guilt, were combined as an indicator of depression, since these four symptoms have been reported to be common in depressed perinatal women (36). These subscales were scored in the same manner as has previously been described for the BSI subscales included in the Anxiety factor. BSI–DEP has an internal consistency alpha coefficient of 0.85 and a test–retest reliability coefficient of 0.84 (29). Psychometric data are not available on the BSI–ADD, as the scale's authors acknowledge that these items load on multiple BSI factors. However, they were justified for inclusion in the original measure, and are included here, because these symptoms represent important vegetative and clinical indicators of mood states. Prenatal scores on all depression measures were used in the prenatal dataset, and postpartum scores in the postpartum dataset.

Statistical analysis

Structural equation modeling analyses were performed using the software LISREL Version 8.54 (37). Standard criteria of a non-significant χ^2 (i.e. $P > 0.05$) and root-mean-square error of approximation (RMSEA) ≤0.05 were considered indicative of adequate model fit (10).

Since the indicator variables used were measured on a variety of different scales, it was necessary to provide a unit of measurement for each latent variable. This was accomplished by assigning one indicator per latent variable a path coefficient of 1.0, i.e. assigning the latent variable the scale of that indicator variable, and estimating the path coefficients of the

latent construct in preliminary confirmatory factor analyses (for Biological Risks: personal psychiatric history; for Psychosocial Stress: 'Life Stress'; for Anxiety: BSI–ANX; for Depression: BSI–DEP). These were the only parameters fixed; the remaining parameters were free to be estimated. As a result, the statistical model included 36 parameters to be estimated (including error estimates), with a total of 84 degrees of freedom.

Due to non-normality of indicator variables, the weighted least-squares (WLS) estimation method (10) was used. Since WLS requires both listwise treatment of missing data and large sample sizes, it was preferable to impute the few missing data points than to delete entire cases on which one or two data points were missing. This was accomplished using LISREL's default of matching missing data points using a vector of variables with incomplete data and a vector of variables with complete data (37). Data were imputed for all missing data points from both the prenatal and 6-week postpartum assessments that could reasonably be imputed, i.e. income, mood measures that had been missed accidentally or because of lack of time at the assessment, and family psychiatric history variables in women who were adopted and had no knowledge of the medical history of their biological relatives. A total number of 42 data points were imputed in this manner (24 of which were used in the prenatal modeling, and 30 of which were used in the postpartum modeling), with no more than five cases imputed for any given variable.

As has previously been recommended (38), a two-step approach was taken in the analyses that follow. In the first step, a measurement model, which tests how well the chosen indicator variables measure the latent constructs, was established and tested for fit using the prenatal data. After examining the fit of the model to the prenatal data, modifications were made as recommended by LISREL modification indices in order to develop an alternative derivative model which could better account for the data. LISREL uses the technique of specification searching to identify parameters or paths, which, if freed, would significantly improve the overall fit of the model (37). Once adequate fit (as defined above) was established for the prenatal measurement model, the prenatal structural model was tested, i.e. the full statistical model that is detailed in Fig. 1, including relationships between latent variables, was evaluated for fit against the prenatal dataset. Finally, the best-fitting structural model for the prenatal dataset was tested for fit against the 6-week postpartum dataset.

The correlation matrix, mean vlaues, SD, and distribution data for all of the variables in the model are provided in Table 1. More detailed information about the statistical treatment of the data is available from the first author.

Table 1. Correlation Matrices and Descriptive Statistics. Prenatal data is given in the upper-right half of the table; postpartum data is given in the lower-left half. For definitions of abbreviations, please refer to Fig. 1

Mean	0.00	0.00	0.00	0.000	0.000	0.000	0.443	0.128	0.
SD	1.00	1.00	1.00	1.000	1.000	1.000	0.443	0.261	0.
Skewness				1.132	2.098	0.861	1.509	3.162	1.
PMS		−0.276	−0.298	−0.168	0.015	0.105	−0.126	−0.022	0.
Psych	−0.270		0.345	0.265	0.160	−0.126	0.396	0.394	0.
Family	−0.314	0.368		0.229	0.257	0.139	0.219	0.212	0.
Support	−0.172	0.332	0.229		0.313	0.217	0.364	0.284	0.
Life stress	−0.057	0.145	0.180	0.276		0.315	0.116	0.211	0.
Education	0.111	−0.088	−0.079	0.053	0.192		−0.047	0.008	0.
BSI–ANX	−0.001	0.523	0.233	0.419	0.067	0.029		0.532	0.
BSI–PHOB	0.103	0.373	0.076	0.358	0.088	0.048	0.623		0.
BSI–OC	0.026	0.310	0.226	0.503	0.008	0.046	0.626	0.495	
BSI–ADD	0.010	0.447	0.322	0.445	0.199	−0.013	0.720	0.574	0.

Results

Measurement model of prenatal data

The initial measurement model (including all of the indicator variables described above) had poor fit to the prenatal dataset (χ^2 = 821.24, P = 0.00; RMSEA = 0.243). LISREL modification indices were used (37) to make changes to this model, as follows. Firstly, because the parameter estimate for PMS history was not statistically significant, and the parameter estimate for cortisol concentrations was only marginally statistically significant, these variables were removed from the model, improving model fit. Secondly, modification indices suggested adding error covariances between BSI–ADD and other indicator variables which were not theoretically justifiable. Because of our awareness of the scale's authors' cautions about the use of BSI–ADD as a subscale (29), we deleted this variable from the model, improving model fit. Similarly, modification indices proposed the addition of error covariance between Education and other variables that could not be justified. For this reason, this variable was also deleted from the model, with a further improvement in model

anxiety (BSI–ANX) and the self-report measure of life stress (LTE, scores on which primarily composed the Life Stress factor) were measuring a common construct to some degree, and as such may share some common error. This point will be further discussed below.

After making these changes, the resulting derivative measurement model had adequate fit to the prenatal dataset (χ^2 = 50.68, P = 0.0664; RMSEA = 0.050). This model was used to test the structural relationships.

Structural model of prenatal data

The initial structural model had identical fit to the prenatal dataset as the measurement model (χ^2 = 50.68, P = 0.0664; RMSEA = 0.050) and so met the fit criteria. However, two of the paths in this model did not reach statistical significance: the relationship between Psychosocial Stress and Depression, and the relationship between Biological Risks and Depression. Since the relationship between Biological Risks and Depression was furthest from statistical significance, we tested the model without this path, and found that the path did not contribute to the fit of the model: without it, model fit was slightly better (χ^2 = 51.41, P =0.072; RMSEA = 0.049). Further, with the path between Biological Risks and Depression no longer in the model, the path between Psychosocial Stress and Depression reached statistical significance.

The resulting derivative model, shown in Fig. 2, was determined to be the best fitting model for the prenatal dataset. In this model, Psychosocial Stress has both a direct effect on the Depression factor, and an indirect effect, mediated through its effects on the Anxiety factor. These relationships were in agreement with our hypotheses. Similarly, the Biological Risks factor had direct effects on both the Psychosocial Stress factor and the Anxiety factor, as we had proposed. However, contrary to our hypothesis, there was no statistically significant direct effect of the Biological Risks factor on the Depression factor: the relationship between these two factors was better accounted for by indirect effects of Biological Risks on Depression, as mediated through both Psychosocial Stress and Anxiety.

Figure 2

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Derivative statistical model of mood changes at 36–40 weeks gestation. Fit criteria for this model: χ^2 = 51.41, P = 0.072; RMSEA = 0.049. Standardized parameter estimates are given above or to the left of the arrows connecting variables to one another. Standardized error estimates are given to the left or right of each indicator variable. Estimates in italics were not statistically

Test of the model to postpartum dataset

The fit of the derivative prenatal structural model (Fig. 2) was then tested against the postpartum dataset. When we tested the hypothesis that both the factor loadings and the factor correlation patterns were equivalent between the prenatal and postpartum datasets, the hypothesis was rejected since the measurement model had very poor fit (χ^2 = 1470.48, P = 0.000; RMSEA = 0.299), and the structural model would not converge (i.e. no unique set of parameter estimates could be determined, suggesting very poor fit). Next, the factor loadings were allowed to vary, and the hypothesis that the factor correlation patterns were equivalent between the prenatal and postpartum datasets was tested. This hypothesis was also rejected, since the model would not converge.

Finally, we attempted to develop a novel model based on the postpartum dataset using the same latent variables as the models described above and guided by LISREL modification indices. However, no measurement model including all four latent variables could be developed which had adequate fit to the postpartum dataset. In other words, the same variables that could adequately account for prenatal depressive symptoms could not explain, to a statistically significant degree, postnatal depressive symptoms.

Discussion

In this manuscript, we have described the results of applying SEM techniques to develop a multidimensional model of mood changes during the perinatal period. The major finding was that biological variables, including both genetic and hormonal factors, influence depressive symptoms during pregnancy indirectly, through their influence on vulnerability to psychosocial stressors and symptoms of anxiety. We interpret this to mean that biological variables, including both genetic and hormonal factors, make an individual more or less likely to respond to particular life situations with feelings of ‘stress’ or ‘anxiety’.

Limitations of the prenatal model

In interpreting these results, some limitations of the data for model building and fitting must be taken into account. Once cases with missing data were omitted, data on 146 participants were available for analysis. Considering that the final structural model tested involved estimating 28 parameters, the sample size was just adequate according to published guidelines of at least four subjects per parameter (39). In addition, several of the observed variables were not normally distributed, requiring use of distribution-free estimation methods. Although using WLS estimation is an improvement over distribution-dependent estimation methods in dealing with this type of data, it still may be deficient in correcting for the violation of multivariate normality, particularly in small sample sizes (40).

risk factors that clearly overlap between these categories, such as personal and family psychiatric history. Recurrence of major depression can occur for a variety of reasons, both biological and non-biological in nature (41). Similarly, while reports suggest that family history of mental illness is a risk factor for postpartum depression (42–44), it cannot be determined on the basis of the available data whether the risk is attributable to a genetic vulnerability to depression or, rather, to the effects of growing up in close proximity to relatives suffering from mental illness. The lack of correlation between the available hormonal measures (plasma progesterone and plasma cortisol concentrations) in this study made it necessary to factor these variables together with the psychiatric history variables. In future research, a preferred strategy would be to include data on several hormonal variables, which could then be combined into a discrete biological factor.

Due to budgetary restrictions, only two hormones could be measured in all study patients, with only one sample available from each assessment period. Because cortisol was not a primary outcome measure of this study, sampling times were not structured around its very significant diurnal fluctuations. Cortisol secretion is particularly variable during the first 4 h after waking, when a daily secretory surge typically occurs (45). Since our sampling generally occurred between 9:30 a.m. and 2:30 p.m., any potential effects of cortisol on mood could have been masked by diurnal changes.

Similarly, plasma progesterone concentrations are known to show substantial intra- and inter-individual variability during pregnancy. It therefore would have been preferable to have data from additional samples to include in the model. Even with this limitation, however, progesterone concentrations made a statistically significant contribution to the Biological Risks factor. This was despite the very low correlations between progesterone concentrations and all of the depression and anxiety indicators (see **Table 1**). A number of studies have reported an association between high progesterone concentrations and symptoms of postpartum depression (4, 23, 46–48), although these effects have tended to be of small magnitude and somewhat inconsistent, e.g. in women who bottle fed but not in those who breastfed their infants (4), or within-subjects but not between subjects (47). In the present study, the statistically significant contribution of progesterone concentrations to the Biological Risks factor, in the absence of significant correlations between progesterone and symptoms of depression or anxiety, provides support for the hypothesis that hormonal factors can play indirect and subtle roles in modulating mood in women.

Adequate fit to the data could only be achieved with the inclusion of an error covariance between BSI–ANX and the Life Stress factor, suggesting that the Psychosocial Stress and Anxiety factors did not constitute discrete constructs. Although others have noted the LTE to be a useful measure of stress in a perinatal population (25), the results of this modeling call into question whether the LTE is truly capturing life stress, or whether it might, to some degree, also be measuring an individual's current degree of distress.

Future research should employ structured interviews to collect this information. Further, inter-rater reliability for the HAMD was not assessed in the study, despite the fact that interviewers had differing levels of experience. Periodic checks of HAMD scoring for depressed patients assessed both by the study psychiatrist (M.K.R.) and another interviewer indicated that consistent scoring procedures were followed; however, the lack of formal inter-rater reliability assessments remains a limitation of this work.

The characteristics of this sample should also be acknowledged in drawing conclusions from these data. The research participants in this study were predominantly Caucasian, in a stable relationship, and well educated. The degree to which these findings can be generalized to more diverse samples of pregnant and postpartum women awaits further research.

Limitations of the postpartum model

Post-hoc model respecification, as was employed in the development of our prenatal model, risks capitalizing on peculiarities of the dataset used in model building (49). The preferred method of assuring that this has not occurred is to test the fit of the derivative model to an independent dataset. To this end, the fit of the derivative prenatal model was tested against the postpartum dataset; however, the prenatal model did not statistically account for the postpartum data.

Failure to find adequate fit in this case would be appropriate if depression at 6-weeks postpartum were to have a different etiological profile than depression during late pregnancy. While few studies have longitudinally investigated risk factors for depression during both pregnancy and postpartum, there is some support for the hypothesis that different risk factors may be relevant at different times, particularly insofar as sociodemographic risks are concerned. Gotlib and colleagues found that while several sociodemographic variables, including years of education and number of children at home, were significantly associated with prenatal depression, they were not associated with postpartum depression (50). Two classes of variables, relevant not to prenatal depression but possibly to postpartum depression, were not included in our analyses, and this may have contributed to our inability to model these data. Firstly, obstetrical complications may influence mental health during the postpartum period (44, 51, 52). Secondly, a recent meta-analysis found that both childcare stress and infant temperament had moderate effect sizes in their relationship with PPD (8). Future models of postpartum mood problems should include indicators of these variables.

Summary of findings and implications

The key finding of this study was that biological risk factors (progesterone concentrations, genetic risk) had no direct effect on prenatal depressive symptoms, but rather, had indirect

and symptoms of depression: the utility of personal and family history of depression in predicting future episodes of perinatal mood disorders is well known (53). Rather, these findings suggest that the variance in depressive symptoms can be best accounted for by the indirect effects of biological risk factors on psychosocial variables and anxiety. These biological variables could alter sensitivity to environmental stressors, such as lack of social support, and in this way, determine the threshold for developing symptoms of depression or anxiety during pregnancy. Further research is required to determine the key biological and psychosocial variables that will allow us to effectively model postpartum depressive symptoms in a similar fashion.

Our model provides support for the hypothesis that depression is a 'complex, multifactorial disorder' (12), and further supports the recommendation that both biological vulnerability and environmental triggers be considered in the study of depression in women (54). The lack of a direct relationship between biological risk factors, including progesterone concentrations, and symptoms of depression during pregnancy could explain the difficulties others have had in identifying a linear relationship between hormonal variables and depression during the perinatal period: it is conceivable that these relationships can only be elucidated in the context of relevant psychosocial stressors. Researchers in the area of perinatal mood disorders should strive to incorporate assessments of key psychosocial variables (social support, life stress, marital satisfaction), even when their primary outcome variables are biological in nature. Clarification of the etiology of postpartum depression through more comprehensive assessment of risk factors could facilitate identification of variables to be targeted in strategies for prevention of postpartum depression in high-risk women. This is particularly important in that available prevention strategies have shown limited effectiveness (55).

Conclusions

During pregnancy, biological variables may alter sensitivity to psychosocial stressors, such as lack of social support, and, in this indirect manner, determine the threshold for developing symptoms of depression. The relationship between biological risk factors and depression is mediated in part by the relationship between depression and anxiety, demonstrating that symptoms of anxiety are important contributors to perinatal mood problems.

The analyses reported here must be considered preliminary, in that the data collected in this study were not ideal for the application of SEM, due to limitations in sample size, non-normality in the outcome variables, and the omission of potentially important causal variables. Despite these limitations, however, this work demonstrates the importance of considering both biological and psychosocial variables in examining women's health issues, and provides a model for studying how relationships between biological and psychosocial

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