



Research paper

Prolactin, a potential mediator of reduced social interactive behavior in newborn infants following maternal perinatal depressive symptoms



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ABSTRACT

Background: The hormone prolactin (PRL) plays a crucial role for the initiation and maintenance of maternal behavior, and is also associated with the etiology of mood disorders in women, especially for depression. The present study aimed to determine whether maternal peripheral prolactin would be associated with newborn behavior disorders following maternal perinatal depressive symptoms, and further to explore the efficacy of the Newborn Behavioral Observations (NBO) in improving newborn social interactive behavior.

Methods: Interview and the 24-item Hamilton Rating Scale for Depression (HAM-D) were used to assess the hospitalized pregnant women waiting for delivery at 37–42 weeks of gestation. A total of 255 subjects were recruited, diagnosed with depression (n=135), and control group (n=120). Within 2 weeks postpartum, mothers were asked to fill with Maternal Attachment Inventory (MAI) to measure maternal care. Neonatal Behavioral Assessment Scale (NBAS) were used to evaluate newborn behavior. The depressed mother-newborns dyad was randomly assigned to NBO intervention and control group. Serum prolactin in mothers and cortisol in mothers and newborns were measured.

Results: The newborns of mothers exposed to maternal perinatal depressive symptoms displayed the reduced newborn social interactive behavior accompanied by decreased maternal serum PRL as well as increased maternal and neonatal serum cortisol. The NBO could be an effective intervention tool.

Limitations: Our study could not be double-blind. The mothers knew which group their infant were in.

Conclusions: Maternal peripheral PRL had the potential to be a mediator in reduced social interactive behavior in newborn infants following maternal perinatal depressive symptoms.

1. Introduction

Pregnant women exposed to stressful life events, such as domestic affairs, financial or relational problems, and serious illness, are particularly vulnerable to depressive symptoms. Prenatal maternal depression has been a significant public health concern, affecting about 10–25% of women (Teixeira et al., 2009). The symptoms of prenatal maternal depression can continue into the postpartum period (Vesga-Lopez et al., 2008). The newborn period is one that is particularly vulnerable for both mothers and their infants, as maternal perinatal depressive symptoms can impair a mother's ability to respond timely to her infant's cues and to engage in sensitive and passionate interactions with her infant that are associated with optimal newborn neurobehavioral development. Increasing evidence suggests that maternal depression results in newborn behavior disorders, such as attentional, emotional and behavioral problems, which would later be noted in

adolescence and adulthood (Feldman et al., 2009). For example, maternal depression has also been related to less responsiveness to stimulation in the neonate (Smith et al., 2011). However, the mechanism underlying newborn behavior disorders resulting from maternal perinatal depressive symptoms remains unclear.

It is generally believed that impaired maternal behavior is responsible for newborn behavioral disorders. While maternal depression may be a proximal cause of impaired maternal behavior (Murgatroyd and Nephew, 2013; Smith et al., 2004), neuroendocrine hormone may exert distal influences on maternal behavior and parenting capacity. The neuropeptide prolactin (PRL) plays a crucial role in the initiation and maintenance of maternal behavior (Larsen and Grattan, 2012), whereas blocking PRL secretion with bromocriptine significantly delays the onset of maternal behavior and impairs parenting capability. Administration of prolactin to nulliparous rats significantly promotes the rapid onset of maternal behavior (Bridges and Ronsheim, 1990). In

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Table 1
Demographic characteristics in pregnant women.

	Normal group (n=120)	Depression group (n=135)	Values	p value
Maternal age (mean ± SD)	29.31 ± 4.194	30.20 ± 3.937	$z=-1.819$	0.069
Gestational age (mean ± SD)	38.09 ± 3.590	37.64 ± 1.713	$z=-1.104$	0.270
Residence			$\chi^2=0.740$	0.390
Rural area	36 (30%)	34 (25.19%)		
Urban area	84 (70%)	101 (74.81%)		
Education			$\chi^2=2.041$	0.564
Middle school	30 (25.0%)	30 (22.22%)		
High school	36 (30.0%)	33 (24.44%)		
Bachelor's degree	41 (34.17%)	52 (38.52%)		
Higher than bachelor's degree	13 (10.83%)	20 (14.81%)		
Family income			$\chi^2=0.698$	0.705
< 2000 RMB/m/person	24 (20.0%)	23 (17.04%)		
2000–5000 RMB/m/person	67 (55.83%)	74 (54.81%)		
> 5000 Yuan/m/person	29 (24.17%)	38 (28.15%)		
Professional status			$\chi^2=0.350$	0.554
Employed	22 (18.33%)	21 (15.56%)		
Unemployed	98 (81.67%)	114 (84.44%)		
Matrimonial status			$\chi^2=0.860$	0.354
Married/cohabiting	113 (83.70%)	123 (91.11%)		
Single	7 (16.30%)	12 (8.89%)		

addition, PRL is implicated in the etiology of mood disorders in women, especially for symptoms of depression (Sjoeholm et al., 2011). Compared to non-depressed controls, the depressed patients showed lower levels of plasma and salivary PRL (Faron-Gorecka et al., 2013; Lykouras et al., 2011). Serum PRL concentration is related to severity of depressive symptoms in women (Slopien et al., 2015). Therefore, prenatal maternal depression results in decreased maternal peripheral PRL accompanied by impaired maternal parenting capability and mother-infant relationship (Grattan and Kokay, 2008).

Moreover, increased maternal peripheral PRL concentration during pregnancy may help attenuate the potentially harmful effects of overexpression of maternal cortisol due to prenatal depression on fetal development. Prenatal maternal depression is associated with hyperactivity of the HPA axis and increased cortisol is a product of this alteration of the HPA axis. Field et al. (2006) found that depressed pregnant women had elevated prenatal cortisol levels mediating the effects of depression on fetal brain development, activity, and growth delays. PRL is often regarded as a “stress hormone” because various stressors stimulate its secretion from the adenohypophysis into peripheral circulation (Torner et al., 2002). The increased PRL is referred to as an adaptation to stress (Torner et al., 2004) and attenuates neuronal stress circuitries (Donner et al., 2007). Thus, PRL mediates the suppression of HPA axis hyperactivity following stress exposure (Torner et al., 2002), implying that PRL inhibits the overexpression of cortisol. Otherwise, the decreased maternal peripheral PRL following prenatal depression is associated with increased maternal and neonatal cortisol concentrations linked with newborn behavioral disorders. Furthermore, peripheral PRL concentration may be implicated in impaired social behavior following chronic social stress. For example, an animal study reported that the offspring of dams exposed to chronic social stress exhibited impaired social behavior accompanied by decreased basal concentrations of plasma prolactin (Babb et al., 2014). However, the above study demonstrated that impaired social behavior in the offspring was associated with their own decreased peripheral prolactin levels. Additionally, there are few clinical studies on the subject. Less is known whether maternal peripheral prolactin concentration is associated with newborn behavioral disorders following maternal perinatal depressive symptoms.

There is a need to develop an effective, easily implemented intervention to ameliorate newborn behavior disorder resulting from maternal perinatal depressive symptoms. The Newborn Behavioral Observations (NBO) system, designed to enhance positive interactions between mothers and their infants and help improve mothers' parent-

ing capacities and perceived confidence (McManus and Nugent, 2014), provides clinicians a short, flexible, and cost-effective intervention. It has been successfully used to reduce symptoms of postpartum maternal depression. Considering NBO as an infant-centered relationship-based tool, NBO has the potential to be an intervention target for moderating newborn social behavior disorder following maternal perinatal depressive symptoms.

The present study aimed to determine whether maternal peripheral prolactin concentration is associated with newborn behavioral disorders following maternal perinatal depressive symptoms, and further to explore the efficacy of the NBO system in enhancing positive interactions between mothers and their infants and thus improving newborn social interactive behavior.

2. Method

2.1. Sample

The subjects were recruited from the hospitalized pregnant women waiting for delivery from the department of gynecology and obstetrics of the first affiliated hospital, medical school of Xi'an Jiao tong university between January 2014 and August 2015. In order to be eligible for this study, pregnant women had to be 18 years of age or older, within 37–42 weeks of gestation, and expecting to deliver a single infant. An additional inclusion criterion was the ability to respond to questionnaires in Chinese. Women were excluded from the sample if they delivered preterm (before 36 weeks of gestation), and their infants were admitted to neonatal intensive care. A total of 255 women were recruited, of whom 135 were diagnosed with depression (n=135), with the rest belonging to a control group (n=120). Inclusion criteria were applied: 1. mother aged 18–40 years; 2. First-time mother, full-term and singleton pregnancy; 3. the birth with no intrapartum complications; and 4. Apgar score > 8 at the first, fifth, and tenth minutes after birth. Exclusion criteria were as follows: any maternal medical illness, hypertension, advanced liver disease, renal failure, cancer, valvular heart disease, heart failure, stroke, atrial fibrillation, peripheral arterial disease, and other severe diseases. Preterm infants (< 37 weeks gestation) and multiple births were excluded from the current analysis. All the subjects completed the infant behavior assessment by the NBAS and NBO interventions, and were followed to 8 weeks postpartum. Demographic characteristics of the sample are shown in Table 1.

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