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Broader autism phenotype as a risk factor for postpartum depression: Hamamatsu Birth Cohort (HBC) Study

Ryosuke Asano [a], Kenji J. Tsuchiya [a,b,c,*], Nori Takei [a,b,d], Taeko Harada [a], Yumeno Kugizaki [a], Ryuji Nakahara [a], Chikako Nakayasu [a], Akemi Okumura [a], Yukiko Suzuki [a], Shu Takagai [a,b,c], Norio Mori [a,c], and HBC Study Team

[a] Research Center for Child Mental Development, Hamamatsu University School of Medicine, Hamamatsu, Japan
[b] Department of Child Development, United Graduate School of Child Development, Osaka University, Kanazawa University and Hamamatsu University School of Medicine, Hamamatsu, Japan
[c] Department of Psychiatry, Hamamatsu University School of Medicine, Hamamatsu, Japan
[d] Division of Psychological Medicine, Institute of Psychiatry, King's College, London, UK

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ABSTRACT

The broader autism phenotype (BAP), which refers to the expression of behavioral and cognitive propensities that are milder but qualitatively similar to those defining autism spectrum disorder, can play a crucial role in postpartum depression (PPD). We investigated whether pregnant women's BAP would increase the risk for PPD, using a representative birth cohort in Japan. Pregnant women were enrolled in the Hamamatsu Birth Cohort (HBC) Study during their mid-gestation ($N = 841$) and were followed up until 3 months after delivery. BAP was measured mainly during the 2nd trimester of the pregnancy by using the Broader Phenotype Autism Symptoms Scale. Participants scoring 9 points or higher on the Edinburgh Postnatal Depression Scale at least once during the first 3 months after childbirth were diagnosed with PPD. Among participants, 128 (15.2%) women were found to have PPD. Multiple logistic regression analyses showed that BAP were associated with PPD (OR = 1.19, 95% CI [1.07–1.31]), even after controlling for other potential confounders. In addition, the association was not moderated by history of depression and/or anxiety disorders, including concurrent depressive and anxiety symptoms during pregnancy. The findings suggest that pregnant women with BAP have an elevated risk for PPD.

1. Introduction

Postpartum depression (PPD) is one of the most commonly observed psychiatric conditions in women after childbirth (Kendell, Chalmers, & Platz, 1987; O'Hara & McCabe, 2013). In the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR: American Psychiatric Association, 2000), PPD had been defined as a depressive disorder with the specifier "postpartum onset". It has been reported that the prevalence of PPD ranges from approximately 10 to 20% in Western countries (Davey, Tough, Adair, & Benzies, 2011; O'Hara & McCabe, 2013) as well as in Asian countries (Matsumoto et al., 2011; Wan et al., 2009). However, a substantial proportion of women with PPD are overlooked

* Corresponding author at: Handayama 1 Higashiku, Hamamatsu 431-3192, Japan. Tel.: +81 53 435 2331; fax: +81 53 435 2291.
E-mail address: tsuchiya@hama-med.ac.jp (K.J. Tsuchiya).

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(Gjerdingen & Yawn, 2007). This is problematic because PPD leads to a variety of negative outcomes, including maternal health problems (e.g., lower levels of self-rated general health; Dennis, 2004), poor parenting (Field, 2010; Paulson, Dauber, & Leiferman, 2006), and delay in children's behavioral development in later life (Hay, Pawlby, Waters, & Sharp, 2008). Therefore, it is important to identify risk factors for PPD to maintain the well-being of mothers and their families.

Studies have reported that some psychosocial factors increase the risk for PPD. Such risk factors include history of psychiatric illness, lack of social support, advanced age, and primiparity (Matsumoto et al., 2011; Milgrom et al., 2008; Mori et al., 2011; O'Hara & McCabe, 2013; Robertson, Grace, Wallington, & Stewart, 2004).

However, other researchers have focused on the biological and genetic basis of PPD. For example, it has suggested that serotonin-system dysfunctions have been associated with risk of depression and PPD (Riccio et al., 2011; Skalkidou, Hellgren, Comasco, Sylvén, & Sundström Poromaa, 2012). Variability in the repeat sequence of HTTLPR, which is a promoter region of serotonin transporter gene (SLC6A4), is associated with autism spectrum disorder (ASD) and related conditions (Cook & Leventhal, 1996), particularly among multiplex families (Devlin et al., 2005). In addition, abnormalities in expression of the SLC6A4 have been specifically associated with PPD (Doornbos et al., 2009), but not with depressive symptoms at 32 weeks after giving birth (Sanjuan et al., 2008). These findings imply that PPD and ASD might share common biological and genetic mechanisms. One way to test this possibility is to investigate the possible association between PPD and the mother's ASD-like behaviors, also known as broader autism phenotype (BAP).

BAP refers to the expression of behavioral and cognitive propensities that are milder but qualitatively similar to those seen in ASD and is more common in relatives of individuals with ASD than in the general population (Piven, Palmer, Jacobi, Childress, & Arndt, 1997). BAP is considered a stable trait rather than a momentary state. Studies have suggested that individuals with BAP have deficits in social motivation and communication, impairments in facial processing and executive functioning, and lower levels of motor imitation and language (Dawson et al., 2002, 2005; Piven et al., 1997; Sucksmith, Roth, & Hoekstra, 2011); all of these characteristics are also seen in individuals with ASD. In addition, it has been shown that individuals with higher levels of BAP are at increased risk for psychiatric disorders, such as major depressive disorder and depressive symptoms (Ingersoll & Hambrick, 2011; Piven & Palmer, 1999; Yirmiya & Shaked, 2005). These findings suggest that pregnant women with BAP may be at an increased risk for developing PPD after giving birth.

The present study was designed to investigate the possible link between BAP and PPD among a representative sample of Japanese women. We are unaware of any studies that have investigated the possible risk of PPD conferred by BAP in pregnant women using birth cohort. Identifying such an association would also be beneficial in providing more efficacious intervention for a large number of PPD sufferers. We hypothesized that pregnant women with BAP, as defined in the Broader Phenotype Autism Symptoms Scale (BPASS; Dawson et al., 2007), would show an increased likelihood of developing PPD after controlling for known risk factors.

2. Method

This study was conducted as a part of an ongoing cohort study, the "Hamamatsu Birth Cohort for Mothers and Children" (HBC; Tsuchiya et al., 2010). A detailed summary of the methodology of the HBC is described below.

2.1. Participants

We consecutively contacted 962 pregnant women who were expected to give birth at our two research sites in Hamamatsu in mainland Japan, namely the Hamamatsu University Hospital and the Kato Maternity Clinic, and who gave birth between December, 2007 and December, 2010. Participants were representative of Japanese women in terms of age, socioeconomic status, and parity, and their children were representative in terms of birthweight and gestational age (Tsuchiya et al., 2010). All participants were given a complete description of the study and provided written informed consent to participate.

The participating women were followed from study entry, which took place during mid-pregnancy, to 3 months after childbirth. Participants were asked to complete an interview with our research team during mid-gestation and filled out the Edinburgh Postnatal Depression Scale (EPDS; Cox & Holden, 2003; Cox, Holden, & Sagovsky, 1987) to measure their depressive symptoms after childbirth. Following the literature (Evans, Heron, Francomb, Oke, & Golding, 2001; Kendell et al., 1987), participants were asked to complete the EPDS three times after delivery at 2–4, 5–7, and 8–12 weeks, and then to mail it back to our research center. Because the diagnosis of PPD was considered unreliable in respondents who completed the EPDS only once during the study period, 121 (12.6%) of the 962 participants were excluded from the analysis. The following values were derived for the group of women excluded ($n = 121$) and the group of women included ($n = 841$) in the analysis: the mean scores of the first observation of the EPDS (4.05 vs. 4.49 points), mean scores of the BPASS (13.42 vs. 13.24 points), mean age of the participants (29.9 vs. 30.9 years), mean age of the partners (32.5 vs. 32.8 years), average household income (5.62 vs. 6.04 million JPY), gender of the child (male 47.9% vs. 51.8%), and parity (primiparae 50.4% vs. 52.8%, respectively).

2.2. Outcome measures

At the time of our measurement, PPD was defined as a depressive disorder with a specifier of postpartum onset in the DSM-IV-TR (American Psychiatric Association, 2000), although this specifier was replaced with a new specifier "peripartum