Original research paper

Effect of prenatal maternal depression on early speech sound acquisition: A preliminary study

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Objective: Speech sound disorders (SSD) are the most prevalent childhood communication disorders. Many cases of SSD have an unknown origin. The study investigated the effect of prenatal maternal depression on the offspring's speech sound production.
Method: Data from 26 mother–child dyads were included in the study. Prenatal maternal depression was assessed by a validated questionnaire during the third trimester of pregnancy. Speech sound production ability was assessed in terms of the number of atypical (non-developmental) speech errors produced in a standardized speech assessment when the children were 2-years-old.
Results: Six of the mothers' questionnaires suggested depression, whereas 20 were within normal limits. Hierarchical multiple regression analyses indicated that prenatal depression uniquely accounted for 30.8% of the variance in speech sound acquisition after controlling for the child's sex and postnatal maternal depression level.
Conclusions: Maternal prenatal depression was significantly associated with more atypical speech errors in the offspring at 2 years. The current findings contribute to understanding the etiology of SSD with unknown origin. At a clinical level, prenatal depression could be taken as a risk factor for SSD.

Keywords: Prenatal maternal depression, Functional speech sound disorders, Contributing factor

Speech sound disorders (SSD) are characterized by difficulty in pronouncing words correctly when compared to one's age peers. SSD are often diagnosed in early childhood from 3 to 6 years of age and are broadly categorized into organically based and functional SSD. While organically based SSD can be readily related to obvious clinical features such as cleft palate and Down syndrome, functional SSD cannot be attributed to any identifiable etiology and therefore have been described as having an unknown origin (e.g. Flipsen *et al.*, 2009). Given the unexplained deficits of functional SSD, this client group presents a challenge to researchers regarding its contributing factors and maintaining causes (Shriberg *et al.*, 1999), as well as to clinicians regarding its early identification and differentiation from typical development. In addition, children with SSD represent a heterogeneous group, differing in severity, types of errors, and the responsiveness to specific intervention approaches. The heterogeneity suggests that rather than one single underlying cause, there are a number of causal and maintenance factors that may be explanatory. In an epidemiological study in the USA, the prevalence of functional SSD based on 1328 monolingual English-speaking 6-year-old children was 3.8% (Shriberg *et al.*, 1999). Using the same diagnostic criteria, the prevalence for the younger counterparts at 3 years old was 15.6% (Cambell *et al.*, 2003). These prevalence estimates suggest that SSD are so highly prevalent in childhood that they occupy a significant proportion of the caseload among speech–language pathologists (American Speech-Language-Hearing Association, 2010).

A number of studies have been conducted to examine the nature of SSD in terms of their underlying mechanism. For example, Stackhouse and Wells (1997) developed a psycholinguistic framework to describe a child's speech ability and difficulties. Through a series of speech processing tasks, a child's speech production ability can be profiled. Besides the

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nature of SSD, many studies have investigated the etiology or risk factors for functional SSD. For example, at 3-years of age, children born to mothers with low education level are at a high risk of having SSD (Cambell *et al*., 2003). In another study, To, Cheung, and McLeod (2013) evaluated some environmental demographic risk factors (e.g. parental education, household income, number of siblings) in a Cantonese-speaking community that contributed to inferior speech development in preschoolers. The researchers found that some of the risk factors found to be significant explained less than 3% of the variance in speech sound ability, leaving a large percentage of unexplained variance. In a retrospective study, Fox *et al*. (2002) examined risk factors associated with specific types of speech disorders. Fox *et al*. reported that in general, a history of fluctuating hearing impairment was associated with delayed phonological development. Perinatal difficulties were associated with inconsistent speech disorder, and family history of SSD was the only factor associated with consistent phonological disorders.

Strong aggregation of SSD within families suggests that SSD may be heritable. Significant odds ratios for positive family history of developmental communication disorders were reported in the literature (Cambell *et al*., 2003; Fox *et al*., 2002; Lewis *et al*., 2007; Shriberg *et al*., 2005). In a systematic review, it was concluded that concordance rates of SSD were significantly higher for monozygotic (MZ) than dizygotic (DZ) twins (Lewis *et al*., 2007) implying a genetic link to the disorder. In addition, male sex is associated with a greater risk for SSD (Cambell *et al*., 2003; Shriberg *et al*., 1999). Molecular genetic studies aiming to locate the exact chromosomal genes contributing to the development of SSD emerged recently. Genome-wide linkage analysis and fine mapping of chromosome loci identified disruptions of the FOXP2 gene, which may be the cause of SSD in some affected individuals (Newbury and Monaco, 2010). However, such monogenic mutations could not be the sole attribution of SSD in the general population due to their scarcity (Lewis *et al*., 2007). In an extensive review of studies on the comorbidity rates among SSD, Pennington and Bishop (2009) found that children with SSD were found to be 2.3 to 6.1 and 2.6 times more likely to have a comorbid diagnosis of language impairment and reading disorder, respectively, than non-SSD controls. Such high rates of comorbidity may reflect overlapping genetic bases of these disorders.

It is generally accepted that SSD are attributed to complex interactions of polygenic and environmental risk, as well as protective factors (Lewis *et al*., 2007; Shriberg, 2010). However, the contributing factors that trigger the phenotype of SSD are still unknown (Shriberg, 2010). In a recent review of risk and protective factors for SSD in preschool children, Harrison and McLeod (2010) found only one study that explored prenatal factors as potential predictors of SSD was located (i.e. Fox *et al*., 2002). In that particular study, prenatal problems were examined retrospectively as a risk factor for SSD in a sample of 65 preschool German-speaking children with functional SSD and 48 normal peers without SSD. Children's speech sound production abilities were measured based on a picture naming task and a German version of the Inconsistent Test. Prenatal and perinatal difficulties were measured using a mother-reported questionnaire. Prenatal problems included extreme prenatal stress, maternal infections, and/or fetal-damaging drug exposure during pregnancy. The perinatal problems included different types of birth complications. In the logistic regression model, Fox *et al*. reported that prenatal and perinatal difficulties (birth factor) were one of the risk factors identified as significant. The odds ratio of this factor of SSD was 8.86 and the 95% confidence interval was 1.51–52.02, implying that the risk of SSD in children with exposure to the prenatal and perinatal difficulties was 8.86 times higher than those without such an exposure. This pattern of findings is generally consistent with the idea that SSD might be associated more with intrinsic biological factors rather than merely the outcome of environmental influences (Reilly *et al*., 2006).

Aim of the present study

Given the robust finding of prenatal complications in Fox *et al*. (2002), the objective of the present longitudinal study was to examine the unique contribution of prenatal depression during the third trimester on 2-year-old offsprings' speech sound abilities after the effect of postnatal depression and sex were controlled for. In other words, the research question to be addressed in the present study was whether prenatal exposure of maternal depression during the third trimester is associated with the number of atypical speech sound errors in the offspring. The present study focused on children at 2 years of age.

Methods

Participants

Participants in the present study were randomly selected from a community sample developed for a previous study examining prenatal maternal mental problems and mothers' eating disorders (Lee *et al*., 2007). The mother and their offsprings were invited to participate in the study. The only inclusionary criterion for the present study was that the offspring was at the age of 2-years-old at the time of testing.

The women were initially screened for depressive symptoms at three time points during pregnancy, namely the first trimester (around 12 weeks), the

second trimester (around 20 weeks), and the third trimester (around 36 weeks) of pregnancy. Prenatal inclusion criteria included Chinese ethnicity and an age of 18-years or older and exclusion criteria included significant medical illnesses, pregnancies conceived through *in vitro* fertilization, and women considering termination of pregnancy. The current study recruited 31 women and their offspring. Informed written consent was obtained from the mothers. Of the 31 children, 5 children were not able to complete 60% of the assessment given in the study due to their limited cognitive ability. This resulted in a total of 26 mother–child dyads. These 26 children included 7 boys and 19 girls from 24 to 28 months (M = 25.2 months, SD = 1.0). All the children were born full term.

Measures

Prenatal depression

The Edinburgh Depression Scale (EDS) (Murray and Cox, 1990), also known as The Edinburgh Postnatal Depression Scale (EPDS) (Cox *et al.*, 1987), was used for screening clinically significant depression during pregnancy (Bowen *et al.* 2009; Seimyr *et al.* 2009) and the postnatal period. The EDS or EPDS is a self-administered screening instrument. Women are asked to respond to each of the 10 items by rating their feelings over the past week on a 4-point Likert scale. The overall score on the instrument can range from 0 to 30 with increased scores reflective of increasing severity (Murray and Cox, 1990). The Chinese version of the scale has been validated among Chinese women in Hong Kong with satisfactory psychometric properties (Lee *et al.*, 1998). The established cut-off score of 11 or higher was used to define a probable case of prenatal depression (Bunevicius *et al.*, 2009). Using this cut-off, six women were screened as probable cases of prenatal depression.

Child's speech sound ability

The *Hong Kong Cantonese Articulation Test* (HKCAT) (Cheung *et al.*, 2006), a standardized assessment, was used to assess the children's speech sound ability. The normative sample of the HKCAT included children between the ages of 2;4 to 12;4 years. For the children who participated in the present study, the number of atypical speech errors was calculated. This measure was defined as error patterns realized by less than 5% of children in the normative sample of the HKCAT. The measure of atypical speech error was chosen as the best indicator of speech production ability because it has been previously shown to be predictive of a clinical diagnosis of SSD when children reach the age of 3 years (McIntosh and Dodd, 2008).

Potential covariates

Postnatal maternal depression when the children were at 2 years of age and the child's sex were included as potential covariates. Postnatal depression was screened using the EPDS and adopting a cut-off score of 13 or higher, since sensitivity and specificity were reported to be higher using this cut-off score (Cox *et al.*, 1996).

Procedures

Women in the database of the previous project were contacted by telephone and invited to participate in the present study when their children reached the age of 2 years. An appointment was made to assess the child either at their home or in a quiet room in the Division of Speech and Hearing Sciences at The University of Hong Kong. The assessors who administered the test were blinded to the mental health profiles of the mothers. Prior to the child assessment, parental consent was obtained and the mother filled in the EDS questionnaire. Upon establishing rapport, each child completed the HKCAT. If the child could not name the item spontaneously, a model was given to elicit an imitated response. Each child took approximately 10–45 minutes to complete the test depending on his/her responsiveness and cooperativeness. Speech samples were recorded on a Sony digital voice recorder (ICD-UX81F) for later transcriptions and subsequent reliability check. The individuals who transcribed the samples and conducted the reliability check were also blinded to the mental status of the mothers.

Reliability

To determine inter-rater transcription agreement, assessments of three randomly selected children (12% of the samples) were transcribed independently by another trained transcriber based on the recordings. The inter-rater and intra-rater point-to-point agreement for both consonants and vowels across transcriptions were 89 and 92% respectively, which were considered to be satisfactory. The original transcriptions of the investigator were used for analysis.

Results

Descriptive analyses

There were six children whose mothers scored above the cut-off on the EDS, indicating probable prenatal depression. These six mothers and their children were placed in the 'depressive' group. The remaining 20 mothers and their children comprised the 'non-depressive' group. The mean EDS scores of the prenatally depressive and non-depressive groups were 11.2 and 6.9 respectively. The mean numbers of atypical speech errors produced by children of the two groups (i.e. with and without exposure to prenatal depression) in both sexes are summarized in Table 1. There were more girls than boys in the current sample. There

Table 1 Number of atypical speech error patterns (Mean and (SD)) produced by children with and without prenatal exposure to maternal depression

	Without prenatal depression			With prenatal depression		
	Boys ($n = 5$)	Girls ($n = 15$)	Total ($n = 20$)	Boys ($n = 2$)	Girls ($n = 4$)	Total ($n = 6$)
Atypical patterns	11.6 (8.3)	4.7 (5.6)	6.4 (6.8)	29.0 (7.1)	12.5 (3.0)	18.0 (9.4)

were more children born to mothers who scored below the cut-off score for prenatal depression than at or above the cut-off. On average, boys produced more atypical speech errors than girls.

Hierarchical multiple regression analyses

Hierarchical multiple regression analyses were conducted to investigate the contributions of sex, prenatal, and postnatal exposure to maternal depression in predicting the offspring's speech ability. The predictor variables fitted in the regression equation (i.e. depression status and sex) were dichotomous and the dependent outcome variable (i.e. the number of atypical speech errors) was continuous. Regression analyses were used so as to control for potential covariates that might have associations with the dependent variable but were not the focus of the study. The variable of sex was coded as '1' for female and '0' for male and the variables of depression status were coded as '1' for above the cut-off and '0' for below the cut-off. Stepwise analyses were conducted. In the first step, sex was entered into the equation based on past evidence showing that sex is a significant risk factor for SSD (Harrison and McLeod, 2010). Postnatal depression level measured at the time of testing was entered next and followed by prenatal depression status in the final step.

The results of the multiple regression analysis for depression are displayed in Table 2. Tests for multicollinearity indicated a low level of multicollinearity (tolerance = 0.89, 0.86, and 0.94 for sex, postnatal depression, and prenatal depression, respectively). Sex accounted for 27.6% of the variance in atypical speech errors in the first step. In the second step, postnatal depression was added and did not significantly increase the amount of variance explained. When prenatal depression was added in the last step, the amount of variance accounted for rose to 58.8%. Controlling for sex and postnatal depression, prenatal depression uniquely accounted for 30.8% of the variance, indicating that prenatal depression was significantly associated with more atypical speech errors.

Discussion

The present study was a preliminary attempt to examine systematically the unique contribution of prenatal depression during late pregnancy on offspring's speech sound development after controlling for the child's sex and the mother's postnatal depression. Sex explained nearly 30% of the variance in speech sound ability of the children. This parallels the observation that there is a negative association between male sex and speech sound development in young children (Harrison and McLeod, 2010). More importantly, the present study found that prenatal depression during the third trimester of gestation uniquely accounted for 30.8% of the variance in the children's speech sound acquisition in terms of the number of atypical speech errors produced. The findings were consistent with the suggestion that prenatal problems may increase the risk of SSD (Fox *et al.*, 2002). The prenatal problems described in Fox *et al.* were evaluated using a yes/no question which was reported by caregivers in a retrospective manner. In the present study, antenatal depression was measured during pregnancy using a validated questionnaire which can minimize report bias. In addition, the present study controlled for postnatal maternal depression. Maternal depression is reported to be negatively associated with the quality of mother–child

Table 2 Hierarchical multiple regression model regressing of atypical speech errors on prenatal exposure of maternal depression

Predictor variables	df	β	R^2	ΔR^2	F	ΔF
Step1	1, 24		0.276		9.15**	
Sex		−0.525**				
Step 2	2, 23		0.280	0.004	4.48*	0.138
Sex		−0.505*				
Postnatal depression		−0.069				
Step 3	3, 22		0.588	0.308	10.5***	16.41**
Sex		−0.419**				
Postnatal depression		−0.206				
Prenatal depression		0.572**				

***$P < 0.001$, **$P < 0.01$, *$P < 0.05$.

interactions (Korja *et al.*, 2008), which may in turn pose a pervasive adverse influence on a child's speech and language development (Westerlund and Lagerberg, 2008). Controlling for postnatal mental health problems in the present study could strengthen the evidence that prenatal depression may be associated with and offspring's eventual phonological acquisition.

The current study might shed light on one possible etiology of functional SSD, which is regarded as having an unknown origin. Despite a certain degree of genetic influence, no single gene has been identified as the causal factor of functional SSD in the general population (Smits *et al.*, 2006). Other non-genetic factors such as antenatal enviromental alternations may also contribute to the development of functional SSD. These kinds of non-genetic factors are referred to as epigenetic modifications, which can induce changes in the expression of genes. Through this epigenetic reprogramming during the sensitive period of fetal development, the developmental trajectory of an offspring can be altered (Bloomfield, 2011), thereby affecting postnatal outcomes of the offspring. The present findings imply that epigenetic modifications induced by prenatal exposure to depression during late pregnancy may be associated with speech sound problems in the offspring. The potential effect of prenatal depression is also supported by a pas research which has outlined the possible association of depression during pregnancy with various developmental disorders such as symptoms of autism spectrum disorders (Kinney *et al.*, 2008), attention deficit, and hyperactivity disorders (de Bruijn *et al.*, 2009; O'Connor *et al.*, 2002), as well as language and intellectual problems (Laplante *et al.*, 2004, 2008). For example, Laplante *et al.* (2004) conducted a prospective longitudinal study to investigate the impact of prenatal maternal stress induced by natural disasters on toddlers' cognitive and language development. Fifty-eight toddles whose mothers had experienced different levels of prenatal stress in an ice storm were evaluated using the Mental Scale of the Bayley Scales of Infant Development (BSID) (Bayley, 1993) and the MacArthur Communicative Development Inventory (MCDI) (Fenson *et al.*, 1993). Prenatal stress uniquely accounted for 11.4 and 12.1% of variances in the child's intellectual and language functioning, respectively. The findings appear to parallel those in the present study and suggest that prenatal maternal mental health problems may be related to SSD in children whose speech problems showed an unknown origin.

The effects of the physiological processes of prenatal mental health problems on offsprings are not yet clear (Talge *et al.*, 2007). It may be possible that antenatal depression would induce increased activity of maternal hypothalamic-pituitary-adrenal (HPA) which would in turn mediate epigenetic changes (Huizink *et al.*, 2004). The adrenal cortex of the HPA axis produces and releases cortisol into the bloodstream as the final product and passes to the fetus through the placenta (Talge *et al.*, 2007). While normal levels of cortisol are essential to neuronal maturations, chronically high concentration of maternal cortisol induced by mental health problems may pose detrimental influences to the offspring's brain development (Huizink *et al.*, 2004). Under normal circumstances, the fetal brain is protected from maternal cortisol by a homone called, 11-β-hydroxysteroid dehydrogenase (11-β-HSD) which oxidizes cortisol into its biologically non-toxic form (Field, 2011). However, 11-β-HSD is substantially reduced towards the end of pregnancy, thus allowing greater transduction of maternal cortisol from the pregnant mother to the fetus (Huizink *et al.*, 2003). The perisylvian region of the brain which is responsible for speech and language development grows rapidly during the third trimester of pregnancy (Cohen-Sacher *et al.*, 2006). It might also be possible that the excessively high cortisol level during late pregnancy have deleterious impacts on that specific brain area of the fetus. This might lead to the speech problems observed in the offspring after birth. However, these speculations need stronger physiological data in order to be supported.

Limitations and future study

Certain limitations exist in the present study. First, the small sample size used in this study limits the statistical power and the number of potential covariates that can be included in the analysis. Moreover, the present study could not directly address the underlying biomechanisms by which prenatal depression contributes to an offspring's development. Future studies could make use of a pregnant women's biochemistry profile, such as urinary cortisol or dopamine levels across stages of pregnancy as possible measures. Finally, the study only focused on 2-year-old children exposed to different degrees of prenatal mental health problems. Longer term follow-up speech sound assessments of these children are therefore necessary to determine whether the influences of prenatal mental health problems are intensified, maintained, or lessen as they mature.

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