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BRIEF REPORT

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Maternal distress across the postnatal period is associated with infant secretory immunoglobulin A

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Abstract

Employing a longitudinal design, relationships between maternal distress (i.e., perceived stress, negative affect, depressive symptomology), and infant secretory immunoglobulin A (sIgA) across the peripartum period were examined in 51 mother–infant dyads. Indices of maternal distress were assessed at four time periods: third trimester of pregnancy and 1, 3, and 6 months postpartum. Infant saliva samples were collected at each of the three time points in the postpartum period to assess sIgA levels. No relationships were found between *prenatal* maternal distress and infant sIgA. Results indicated that during the *postnatal* period, higher concurrent maternal distress was associated with reduced infant sIgA. Maternal distress did not prospectively predict infant sIgA. These findings advance our understanding of the social-context of infant development, highlighting the significance of maternal regulation of infant immunity.

KEYWORDS

infant immunity, maternal depression, maternal mood, maternal stress, salivary secretory immunoglobulin A

1 | INTRODUCTION

Pregnancy and the first several months after childbirth are characterized by joy and excitement, yet this period has also been recognized as a time of stress and mood disturbance as mothers experience major life changes (Bondas & Eriksson, 2001). A growing literature reveals individual differences in maternal experiences regulate infant behavior and biology (Ostlund, Measelle, Laurent, Conradt, & Ablow, 2017). Specifically, prenatal stress, anxiety, and mood disturbances (e.g., depression) are associated with child outcomes such as low infant birth weight and preterm birth (Dunkel Schetter, 2011), aberrations in neurodevelopment (van den Bergh et al., 2017), behavioral and emotional problems (Bush et al., 2017; MacKinnon, Kingsbury, Mahedy, Evans, & Colman, 2018; Nolvi et al., 2016), and poor cognitive development (Bergman, Sarkar, O'Connor, Modi, & Glover, 2007; Schechter et al., 2017). Alterations to the in utero environment can even have long-term implications for the child's behavior and physiology (Hocher, 2014; Rakers et al., 2016). Collectively, these findings support the "fetal programming hypothesis," which poses that the developing fetal system is sensitive to characteristics of its uterine environment (Barker, 1998). Similarly, studies have demonstrated that postnatal maternal stress, anxiety, and mood disturbances are also associated with adverse child outcomes such as behavior problems (Netsi et al., 2018), cognitive and socio-emotional deficits (Field, 2010; Goodman et al., 2011), and heightened physiological reactivity to stress (Feldman et al., 2009). Though mothers influence a host of infant outcomes, questions remain as to how maternal experiences may alter the developing infant immune system. Our previous work has shown levels of salivary secretory immunoglobulin A (sIgA) undergo significant changes across the first three months postpartum (Hibel & Schiltz, 2016). We aim to extend this work by examining the influence of maternal distress (i.e., perceived stress, negative affect, depressive symptomology) during the peripartum period on infants' salivary sIgA.

SIgA, the predominant mucosal immunoglobulin, is often regarded as the first line of defense against invading pathogens

(Bridgman et al., 2016). Antibodies produced in the salivary glands are thought to be indicative of mucosal immune competence (Gleeson & Cripps, 2004) and children's mucosal immune development (Seidel, Schulze, Kiess, Vogtmann, & Borte, 2000). Specifically, lower salivary sIgA levels have been associated with upper respiratory tract infections, periodontal disease, caries, wheezing, and allergic symptoms (Fagerås, Tomičić, Voor, Björkstén, & Jenmalm, 2011; Laurent, Stroud, Brush, D'Angelo, & Granger, 2015; Sandin et al., 2011).

Psychological factors, such as stress and negative emotions, have an effect on salivary sIgA levels (Rein, Atkinson, & McCraty, 1995), which can place individuals at risk for illness (Gleeson & Cripps, 2004; Jemmott & McClelland, 1989). Children's developing immune systems also seem to be sensitive to biological and psychological experiences of stress. Specifically, reduced sIgA has been associated with heightened cortisol and exposure to stressful experiences (Drummond & Hewson-Bower, 1997; Nozaki et al., 2007; Watamura, Coe, Laudenslager, & Robertson, 2010). Thus, studies suggest that sIgA may be a stress responsive biological marker, albeit one that is relatively underexplored in infants and children.

There is evidence that maternal experiences alter offspring immunity, though most notably in animal studies. Findings from rat, mouse, and monkey models suggest that prenatal maternal stress compromises immune function (Marques, O'Connor, Roth, Susser, & Bjørke-Monsen, 2013; Merlot, Couret, & Otten, 2008). Findings in human studies are limited, though evidence suggests that even prenatal experiences within the typical range may influence infant immunological functioning. Specifically, mothers with higher nervousness had neonates with higher cord blood sIgE (Lin, Wen, Lee, & Guo, 2004). Studies of high risk mothers are more prevalent, though findings have been mixed. For example, mothers with mild to moderate depressive symptoms during pregnancy had infants who demonstrated higher spontaneous cytokine production (Mattes et al., 2009), though mothers with a prenatal anxiety diagnosis had infants who exhibited a reduction in immunological responses to vaccines (O'Connor et al., 2013). Thus, though the direction of the effect is not consistent, several studies demonstrate links between forms of maternal prenatal distress and offspring immunity.

Though the fetal immune system appears to be sensitive to influences in utero, the immune system continues developing after birth and is also sensitive to contextual stressors (Riis et al., 2016) and supportive caregiving (Vermeer, van IJzendoorn, Groeneveld, & Granger, 2012). Animal studies show that early rearing behavior can influence infant immunity, such that maternal separation is associated with suppressed immunological functioning in monkeys (Hennessy, Deak, & Schiml-Webb, 2001; Laudenslager, Reite, & Harbeck, 1982). Likewise, in humans, disruptions in caregiving and parenting difficulties reduce immunity and resistance to disease (Johnson, Riley, Granger, & Riis, 2013; Mrazek et al., 1999). Riis et al. (2016) found that, among girls, maternal distress (a composite of maternal anxiety, depression, and parenting stress) moderated cytokine–cortisol associations, such that as maternal distress increased, cytokine–cortisol relations became weaker. Similarly, lower caregiver sensitivity in child care settings has been associated with lower levels of salivary sIgA in toddlers (Vermeer et al., 2012). To our knowledge, we are the first to examine the associations between maternal distress (as measured by perceived stress, negative affect, depressive symptomology) across the pre and postnatal period and infant salivary sIgA.

Utilizing a longitudinal design, we examine the influence of pre and postnatal maternal distress (i.e., stress, negative affect, and depressive symptomology) on infant salivary sIgA. Specifically, we will examine (a) the association between prenatal maternal distress and infant salivary sIgA levels and (b) the association between postnatal maternal distress and concurrent and prospective infant salivary sIgA levels. Given the mixed prenatal findings revealing both heightened and dampened immune activation with maternal psychological states, we do not have a directional prediction. Conversely, accumulating evidence suggests that postnatal maternal distress will attenuate infant sIgA.

2 | METHOD

2.1 | Procedure

This study was approved by the Institutional Review Board at Purdue University. Prior to participating in the study, mothers gave informed consent for themselves and assent for their unborn child. Assessments were scheduled during the third trimester of pregnancy, and then one, three, and six months postpartum on days when mothers were home with their infants (Table 1). Assessments were scheduled around 4:00 p.m. to control for any potential diurnal rhythm in salivary sIgA. Saliva, length, and weight were collected at all visits. Participants were compensated $15 at the prenatal visit, $20 at 1 and 3 months, and $25 at 6 months.

2.2 | Participants

A convenience sample of pregnant women was recruited through infant CPR and breastfeeding classes offered by local hospitals and parenting networks in a small Midwestern town. From these classes, 106 women expressed initial interest, and of that, 17 did not fit inclusion criteria; 38 withdrew before their first assessment, resulting in 51 mothers completing the first assessment. Inclusion criteria included nonhigh-risk pregnancies, no chronic illnesses, and plans of returning to work within three months of delivery. Mothers' ages ranged from 19 to 41 years (M = 29.1 years, SD = 4.39). The majority were married (90.2%), primiparous (92%), and Caucasian (90.2%). The modal household income fell between $30,000 and $49,000 (range: less than $10,000 to more than $110,000). Forty-seven percent (n = 23) of the infants in the study were female. Mothers had on average 7.0 weeks of maternity leave. At 6 months, 60% of

TABLE 1 Means (and standard deviations, unless indicated otherwise) of mother and infant demographic, biometric, and psychological data at each of the four assessments

	Assessment 1	Assessment 2	Assessment 3	Assessment 4
Dyads N	51	49	47	45
I female %		47	47	49
I age[a]	35.85 (1.2)	4.38 (0.35)	15.27 (3.96)	26.62 (0.95)
Breastfeeding % (*N*)		85.7 (42)	72.3 (34)	64.4 (29)
Exclusive % (*N*)		60.8 (31)	52.9 (27)	31.4 (16)
I flow rate[b]		0.15 (0.11)	0.42(0.21)	0.77 (0.44)
I hours since slept		0.80 (0.93)	1.57 (1.57)	1.35 (1.18)
I weight[c]		9.58 (1.19)	13.38 (2.13)	16.95 (1.81)
I length[d]		21.53 (0.71)	24.36 (1.22)	26.09 (1.14)
I salivary sIgA[e]		170.87 (181.33)	51.24 (42.22)	41.40 (22.29)
M stress[f]	10.75 (5.62)	8.74 (5.90)	9.51 (6.73)	9.35 (4.89)
M affect[g]	1.73 (0.44)	1.65 (0.42)	1.68 (0.62)	1.67 (0.44)
M depressive symptoms[h]	5.02 (3.22)	4.27 (3.47)	4.27 (3.62)	4.22 (3.62)

Note: M indicates maternal, I indicates infant.
[a]For assessment 1, infant age represents weeks gestation, for assessments 2–4 age represents weeks since birth.
[b]Flow rate is calculated in ml/min.
[c]Infant weight in pounds.
[d]Infant length in inches.
[e]All sIgA values are in mg/ml.
[f]Stress was measured with the Perceived Stress Scale.
[g]Affect was measured with adjectives from the Positive and Negative Affect Schedule.
[h]Depressive symptoms were measured using the Edinburgh Postnatal Depression Scale.

the infants were cared for outside of the child's home, 39.5% were cared for in their home by a relative or babysitter, and one child was cared for in their home by their mother in the mother's in-home daycare.

2.3 | Measures

2.3.1 | Maternal psychological distress

Maternal psychological distress was measured using three commonly-used, validated instruments. Perceived Stress was assessed using the 10-item Perceived Stress Scale (PSS; Cohen, Kamarck, & Mermelstein, 1983). This scale has been used with samples of nonpregnant and pregnant women (Culhane et al., 2001; Glynn, Schetter, Hobel, & Sandman, 2008) and been shown to have both good internal consistency (α = 0.80) and validity (Cohen et al., 1983). Alpha reliability was acceptable across all four visits (α = 0.88, 0.87, 0.92, 0.83). Negative Affect was measured using a selection of adjectives from the Positive and Negative Affect Schedule (PANAS; Friedman & Ryff, 2012). This scale measured the extent to which mothers felt a range of emotions across the last 30 days. Negative adjectives were "afraid," "jittery," "irritable," "ashamed," "upset," "angry," and "frustrated." Alpha reliability was acceptable across all four visits (α = 0.76, 0.77, 0.91, 0.74). Depressive symptomology was measured using the Edinburgh Postnatal Depression Scale (EPDS), which has been used during pregnancy and postpartum (Hahn et al., 2019; Matthey, Souter, Mortimer, Stephens, & Sheridan-Magro, 2016). This measures the range of self-reported depressive symptoms over the past week. Alpha reliability was acceptable (α = 0.76, 0.82, 0.79, 0.79) across all four visits. A principal axis factor analysis confirmed that the three subscales load on a single factor at each of the four assessments. Alpha reliability was mostly acceptable (α = 0.76, 0.84, 0.88, 0.67; see correlations Table 2). Higher scores on the maternal distress composite indicate greater distress.

2.3.2 | Salivary Secretory Immunoglobulin A

Infant saliva was collected by trained experimenters using a highly absorbent hydrocellulose swab designed specifically for saliva collection in infants (Salimetrics). The swab was placed in the infant's mouth until the lower third of the swab was saturated. The time required to complete the saliva collection was noted to calculate flow rate. Infant intake of breast milk, formula, or other dairy products was restricted for 20 min prior to the saliva collections. Samples were transported on ice and remained frozen at −80°C until assay. All samples were assayed for sIgA using an indirect enzyme immunoassay (Salimetrics). The sIgA assay uses 25 μl of saliva and has a sensitivity of 2.5 μg/ml. Assays were run in duplicate and the average of the duplicates was used in all analyses. To correct for skewed distributions, sIgA levels were log transformed and Winsorized by setting all outliers to three standard deviations beyond the mean (Riis, Granger, Dipietro, Bandeen-Roche, &

TABLE 2 Bivariate correlations among mother and infant biometric and psychological data at each of the four assessments. Subscripts indicate assessment number

Measure	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. $sIgA_2$	1														
2. $sIgA_3$	0.32*	1													
3. $sIgA_4$	0.13	0.39**	1												
4. $Stress_1$[a]	−0.21	−0.42**	−0.20	1											
5. $Stress_2$	0.07	−0.12	−0.10	0.31*	1										
6. $Stress_3$	−0.12	−0.27	−0.03	0.53**	0.58**	1									
7. $Stress_4$	0.05	−0.51**	−0.30*	0.48**	0.59**	0.60**	1								
8. $Affect_1$[b]	−0.20	−0.13	0.05	0.45**	0.26	0.21	0.11	1							
9. $Affect_2$	−0.01	−0.06	−0.23	0.13	0.59**	0.44**	0.46**	0.20	1						
10. $Affect_3$	−0.24	−0.28	−0.25	0.44**	0.33*	0.66**	0.26	0.11	0.55**	1					
11. $Affect_4$	−0.04	−0.20	−0.37*	0.38**	0.33*	0.42**	0.43**	0.13	0.64**	0.52**	1				
12. Dep Sx_1[c]	−0.18	−0.30*	−0.14	0.61**	0.16	0.30*	0.24	0.46**	0.06	0.22	0.28	1			
13. Dep Sx_2	0.14	−0.07	−0.25	0.28	0.68**	0.39**	0.52**	0.22	0.65**	0.35*	0.50**	0.45**	1		
14. Dep Sx_3	−0.07	−0.28	−0.14	0.50**	0.45**	0.78**	0.49**	0.26	0.44**	0.66**	0.45**	0.47**	0.51**	1	
15. Dep Sx_4	0.19	−0.19	−0.07	0.45**	0.27	0.32*	0.45**	−0.09	0.11	−0.02	0.12	0.32*	0.29	0.34*	1

[a]Stress was measured with the Perceived Stress Scale.
[b]Affect was measured with adjectives from the Positive and Negative Affect Schedule.
[c]Depressive symptoms were measured using the Edinburgh Postnatal Depression Scale.
*$p < .05$.
**$p < .01$ level.

Johnson, 2015). Two infant values were Winsorized. All skewness statistics were below 1.0 after the transformations. Out of the 153 potential sIgA samples, 5 infant saliva samples were missing due to insufficient sample for assay, and 12 were missing due to attrition.

2.3.3 | Key covariates

Indices of infant and maternal health (e.g., infant length and weight, breastfeeding status, smoking status, symptoms of current illness), family demographics (marital status, maternal age, household income, infant sex) and infant salivary flow rate were examined as potential control variables. Breastfeeding status was determined by a dichotomous variable asking the mothers "are you currently breastfeeding?", which included mothers who provided any breastmilk, even if not exclusively breastfeeding. Indices were included in analyses as covariates if variables at any time point were associated at $p < .1$ with infant sIgA at any time point.

2.3.4 | Analytical strategy

The main analyses employed a series of mixed model repeated measures ANCOVAs determining the association between maternal distress and infant sIgA across the three assessments (1, 3, and 6 months postpartum). SAS PROC MIXED (SAS v9.2; SAS Institute) was used for all main analyses. To avoid bias in estimates associated with listwise deletion, full-information maximum likelihood was utilized on all mixed models (Schafer & Graham, 2002). Mixed models account for the nesting of longitudinal data and employ standard error adjustments that account for these correlations (Singer & Willet, 2003). The 3-month assessment served as the reference point in all analyses, to capture the nonlinear change in sIgA across this time period (Hibel & Schiltz, 2016). First, for the prenatal analyses, the composite factor of maternal prenatal distress (i.e., perceived stress, negative affect, depressive symptomatology) was used to predict the three infant sIgA samples (1, 3, 6 months), providing the main effect of prenatal distress on infant immunity. We next examined if this association changed across the three assessments by examining assessment time point as a moderator. Second, for the postnatal analyses, the composite factor of maternal postnatal distress at each of the three assessments was used to predict infant sIgA at each of the three assessments. This analysis provided the concurrent associations between maternal distress and infant sIgA. Next, we examined if these associations changed across the three assessments by examining assessment time point as a moderator. Finally, using a time-lagged stacked dataset, maternal distress was prospectively examined as a predictor of subsequent infant sIgA. In other words, maternal distress at one time point predicted infant sIgA at the next time point. In line with previous analyses (Hibel & Schiltz, 2016), only infant length and breastfeeding status were associated with infant sIgA in bivariate correlations, thus these variables were controlled for in all analyses.

2.3.5 | Data availability statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

3 | RESULTS

3.1 | Descriptive statistics

Descriptive statistics of mother and infant demographic, biometric, and main analysis variables are displayed in Table 1. Maternal depressive symptoms were predominantly in the normative range (0–20) with only five scores (out of 192) above the clinical cutoff (≥13). Negative Affect and Perceived Stress scores, with ranges of 1–3.5 and 0–27, respectively, also reflect typical levels. Depressive symptoms, negative affect, and perceived stress were stable across the four assessments (Table 1). As reported previously (Hibel & Schiltz, 2016) infant sIgA decreased from 1 month to 3 months ($b = 0.91$, $SE = 0.07$, $p < .001$) but then remained stable from 3 months to 6 months ($b = 0.05$, $SE = 0.04$, *ns*). Bivariate correlations of main analysis variables are displayed in Table 2.

3.2 | Main analyses

3.2.1 | Does prenatal maternal distress predict infant salivary sIgA?

Employing a mixed model repeated measures ANCOVA, prenatal maternal distress was examined as a predictor of infant sIgA, while controlling for child length, breastfeeding status, and assessment time point (1, 3, or 6 months postpartum). Concurrent maternal distress was also included as a control variable. Maternal distress was not a significant predictor of infant sIgA ($b = -0.06$, SE = 0.02, *ns*; Table 3; Model 1). Further, maternal distress did not interact with assessment to predict infant sIgA across the visits (see table 3; Model 2).

3.2.2 | Do postnatal levels of maternal distress concurrently or prospectively associate with infant salivary sIgA?

Employing a mixed model repeated measures ANCOVA, associations among maternal distress at each of the three postnatal time points and concurrent infant sIgA levels were examined, while controlling for child length, and breastfeeding status. Greater maternal distress was concurrently associated with reduced infant sIgA ($b = -0.14$, $SE = 0.06$, $p = .03$). The associations between maternal distress and infant sIgA did not change across the assessments (see Table 3; Model 4) and maternal distress did not prospectively predict infant sIgA (see Table 3; Models 5 and 6).

TABLE 3 Hierarchical linear models examining maternal distress and infant sIgA

	Model 1		Model 2		Model 3		Model 4		Model 5		Model 6	
Parameter	**Estimate**	***SE***	**Estimate**	***SE***	**Estimate**	***SE***	**Estimate**	***SE***	**Estimate**	***SE***	**Estimate**	***SE***
Intercept	0.89	1.39	1.65	1.26	0.70	1.30	0.60	1.32	0.42	1.36	0.27	1.38
Breastfed	−0.02	0.15	−0.08	0.16	0.01	0.15	−0.03	0.16	−0.03	0.16	−0.03	0.16
Infant length	−0.05	0.05	−0.08	0.05	−0.04	0.05	−0.03	0.05	−0.03	0.05	−0.02	0.06
Assessment 2[a]	0.87**	0.33	0.78**	0.24	0.89***	0.24	0.91***	0.24	0.88**	0.24	0.89***	0.24
Assessment 4[b]	−0.01	0.14	0.08	0.13	−0.04	0.14	−0.05	0.14	−0.07	0.15	−0.09	0.15
Distress	−0.09	0.08	−0.28	0.09	−0.13*	0.06	−0.20*	0.09	−0.02	0.06	−0.01	0.11
Distress* 2			0.01	0.18			0.26	0.19			−0.13	0.20
Distress* 4			0.26	0.10			0.07	0.11			0.01	0.13

Note: Model 1: prenatal maternal distress; Model 2: prenatal maternal distress moderated by assessment time point; Model 3: concurrent postnatal maternal distress; Model 4: concurrent postnatal maternal distress moderated by assessment time point; Model 5: time-lagged maternal distress; Model 6: time-lagged maternal distress moderated by assessment time point.

[a]Assessment time point 3 (3 months postpartum) served as the reference group, thus Assessment 2 (1 month postpartum) indicates the difference in sIgA at assessment time point 2 relative to time point 3.

[b]Assessment time point 3 served as the reference group, thus Assessment 4 (6 months postpartum) indicates the difference in sIgA at assessment time point 4 relative to time point 3.

*$p < .05$.

**$p < .01$ level.

***$p < .001$ level.

4 | DISCUSSION

Infant biobehavioral regulation develops within the context of the mother–child relationship. Specifically, studies have found mothers to influence the development of child behavior, emotions, and stress physiology (Ciciolla, Gerstein, & Crnic, 2014; Sanders, Zeman, Poon, & Miller, 2015; Talge, Neal, & Glover, 2007). Relatively fewer studies have focused on the role of maternal functioning on infant immunological development, particularly in nonclinical community samples. In this study, a maternal distress composite was created from mothers' perceived stress, negative affect, and depressive symptomology. Across the assessments mothers exhibited depression symptoms in the normative range (Dennis & Ross, 2005), and had stress levels comparable to other community samples of pregnant women (Chou, Avant, Kuo, & Fetzer, 2008). The current study provides evidence that even normative experiences of maternal distress may reduce infant secretion of sIgA. These findings demonstrate the significance of maternal mental well-being on infant immunological development during the first six months of life.

Past studies examining prenatal maternal influences on infant immunity have predominantly involved animal models, and suggest maternal prenatal stress hinders offspring immunity (Marques et al., 2013). Human studies, on the other hand, have been more mixed in their findings, and therefore there are debates in the field as to how and why maternal experiences influence fetal and infant outcomes. For example, some evidence suggests that normative levels of stress are important for neonates' optimal psychological and psychological development (DiPietro, 2004). Others suggest that stress, even at nonclinical levels, can be detrimental (Huizink, Robles De Medina, Mulder, Visser, & Buitelaar, 2003; Talge et al., 2007). Further, the timing of the stress may also prove to be important. Specifically, stress early in pregnancy may lead to preterm birth and worse infant outcomes, though there may be an adaptive decline in the transmission of maternal stress to the fetus across gestation (Glynn et al., 2008). Perhaps lending support for the notion that third trimester experiences are less influential, we did not find individual differences in typical levels of maternal distress during the third trimester of pregnancy to up or down regulate infant salivary sIgA.

The human immune system is highly complex, with numerous substrates each performing synergistic (Gouwy, Struyf, Proost, & Van Damme, 2005), or even antagonistic actions relative to other substrates (Opal & DePalo, 2000). Further, immune marker action must be interpreted within the context of the sampling source (e.g., blood, fecal, saliva, nasal wash). In other words, the lack of significant association between prenatal distress and salivary sIgA is not necessarily at odds with previous prenatal programming research, and suggests that salivary sIgA may be one aspect of the infant immune system that is not influenced by normative prenatal experiences. B cells produce secretory IgA. Yet at birth, these immature cells lack their full production capabilities (Gregory & Walker, 2013). It is possible that the developmental timing of B cell functionality might protect these cells from

prenatal influence, but leave them vulnerable to postnatal experiences and exposures.

Indeed, we found that higher maternal distress was associated with concurrent reductions in sIgA. These associations were independent of multiple potential confounds, including prenatal psychological states, child length, and the child's breastfeeding status. SIgA serves as a defense against infections, and low salivary sIgA levels are associated with increased likelihood of illness (Jemmott & McClelland, 1989; Volkmann & Weekes, 2006). Numerous behavioral immunology studies in adults have shown that psychological distress and stressful experiences reduce breast milk (Groer, Davis, & Steele, 2004; Kawano & Emori, 2015) and salivary sIgA (Engeland et al., 2016; Graham, Chiron, Bartholomeusz, Taboonpong, & Brooy, 1988; Phillips et al., 2006; Segerstrom & Miller, 2004) while the developmental literature consistently shows maternal well-being is a primary regulator of child development (e.g., Morris, Silk, Steinberg, Myers, & Robinson, 2007). Our findings extend these two fields revealing the importance of maternal well-being for infant immunity. These findings are in line with similar work suggesting nonclinical levels of maternal distress disrupts children's ability to appropriately up and down regulate inflammatory processes (Riis et al., 2016) and extend these associations to include implications for humoral immunity in the oral cavity. Interestingly, the lack of time-lagged associations imply that the biobehavioral associations across this time period are not creating long-term programming. It seems that mother–child biobehavioral transactions are more acutely impacting infant immunity.

Mothers and infants exchange physiology both prenatally and postnatally, in utero via the placenta and later through breastfeeding. However, we did not find relationships among sIgA and prenatal maternal distress, and relationships between postnatal maternal distress and infant sIgA were significant, independent of breastfeeding status. These findings suggest that another mechanism beyond physiological exchange may explain the association between mothers' psychological state and infants' immunity. It is well understood that maternal sensitivity is paramount to optimal child development, and hostile or less sensitive caregiving can compromise infant emotional (Taylor-Colls & Pasco Fearon, 2015) and physiological (e.g., cortisol; Valentino et al., 2015) development. SIgA appears to be related to caregiver quality, too, as lower caregiver sensitivity in a community sample of caregivers has been associated with lower sIgA levels in toddlers (Vermeer et al., 2012). It is therefore possible that maternal psychological states influence infant sIgA through maternal sensitivity, even in nonclinical, community samples.

Limitations of this study should be noted. First, our small and racially homogenous sample limits the generalizability of these findings. This could have affected the lack of significant relationships detected between prenatal influences and infant immunity. Future studies should include a larger, more diverse sample. Second, we lack a measure to determine the method of how maternal distress transmits to influence child physiology. Examining maternal sensitivity within the context of the developing infant immune system could open up the possibility of understanding how these mother–infant biobehavioral associations unfold. Lastly, previous literature has found associations between maternal factors and child outcomes to be sex dependent (Riis et al., 2016). This study was under powered to test sex differences, but future studies should attempt to examine how infant sex influences mother–child biobehavioral associations overtime.

5 | CONCLUSIONS

Our findings highlight the role of mothers' psychological state on the developing infant immune system. The United States is the only wealthy nation that does not guarantee paid family leave surrounding the birth or adoption of a child (Burtle & Bezruchka, 2016). Numerous studies have highlighted the mental and physical health benefits of paid leave. Specifically, studies have shown paid leave is associated with reduced maternal psychological distress and depression, and is strongly associated with lower infant mortality rates (Burtle & Bezruchka, 2016; Tully, Stuebe, & Verbiest, 2017). Policy supporting postnatal maternal mental well-being may contribute to a more robust infant immune system, which may prevent infant vulnerability to infections and disease.

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