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Full-length Article

Socioeconomic status, financial stress, and glucocorticoid resistance among youth with asthma: Testing the moderation effects of maternal involvement and warmth

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ABSTRACT

Objectives: Children who grow up in more socioeconomically disadvantaged homes experience greater levels of inflammation and worse asthma symptoms than children from more advantaged families. However, recent evidence suggests that certain family-level factors can mitigate health disparities associated with socioeconomic status (SES). In a sample of youth with asthma, we investigated the potential buffering effects of maternal involvement and warmth on SES disparities in asthma-related immune responses, assessed via glucocorticoid resistance (GR) of immune cells.

Methods: One hundred and forty-three youth (10–16 years of age) with asthma completed measures of maternal involvement and warmth, and their primary caregivers reported their levels of education, income, and financial stress. Peripheral blood mononuclear cells from youth's blood were isolated, cultured, and assayed to determine mitogen-stimulated (PMA/INO + Etho) and mitogen/hydrocortisone-stimulated (PMA/INO + Cort) levels of two Th-2 cytokines (i.e., interleukin-5, interleukin-13) and one Th-1 cytokine (i.e., interferon-γ). GR was calculated by subtracting log-transformed cytokine concentration in the PMA/INO + Etho samples from log-transformed cytokine concentration in the PMA/INO + Cort samples.

Results: Both maternal involvement and warmth moderated the indirect pathway from family SES to GR of Th-2 cytokines via financial stress. Specifically, we found that low family SES was associated with elevated GR of Th-2 cytokines via increased financial stress among youth reporting low levels of maternal involvement and warmth, but not among those reporting high levels of maternal involvement or warmth.

Conclusions: These results highlight the protective role of maternal involvement and warmth in health-related biological processes modulated by family SES among youth with asthma.

1. Introduction

Social stratification of wealth and prestige is a robust predictor of health disparities (Adler et al., 1994; Chen and Miller, 2013). Research suggests that the effect of family socioeconomic status (SES) on youth health is mediated by multiple ecological (e.g., family conflict) and psychological factors (e.g., stress), which cumulatively lead to dysregulated health-related biological processes (Gallo and Matthews, 2003; Miller and Chen, 2013). However, less often examined are the protective factors that may mitigate the adverse impact of growing up in a low SES

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environment. The present study tested whether two key aspects of parenting—maternal involvement and warmth—modulated the previously identified link between family SES and immune responses implicated in asthma pathogenesis in a sample of youth with asthma.

Psychological stress is often proposed as a key mediator linking SES to health (Baum et al., 1999; Gallo and Matthews, 2003; McEwen and Gianaros, 2010). For example, the reserved capacity model (Gallo and Matthews, 2003) proposes that individuals from lower SES backgrounds are more likely to experience stressors and less able to cope with these stressors than their higher SES counterparts, partially due to their limited resources. Although stress exposure and response are often invoked as critical mediators linking SES to health, very few studies have formally tested this hypothesis, and no clear evidence has emerged supporting the mediating role of stress exposure in the SES-health link from the few studies that explicitly tested this hypothesis (Cundiff et al., 2020). Cundiff and colleagues (2020) suggest that adopting measures of psychological stress that are theoretically specific to SES (vs. broad measures of stress) may increase the theoretical value and predictive utility of stress when testing the SES-health association. Thus, we examined the mediating role of perceived financial stress in the association between family SES and health in this study.

Chronic stress has been shown to exacerbate airway inflammation and, ultimately, to worsen clinical symptoms of asthma (Chen and Miller, 2007; Haczku and Panettieri, 2010; Landeo-Gutierrez and Celedón, 2020; Rosenberg et al., 2014). Airway inflammation involves a variety of immune cell types and mediators including T-helper (Th) cells, such as Th-1 and Th-2 cells (Busse et al., 1993; Umetsu et al., 2002). Th-1 cells perpetuate cellular immune responses through the production of interferon-γ (IFN-γ) and interleukin-2 (IL-2), while Th-2 cells promote humoral responses through releasing cytokines such as IL-5 and IL-13 (Berger, 2000; Chen and Miller, 2007). One way chronic stress amplifies airway inflammation in asthma is by altering the glucocorticoid sensitivity of immune cells (Chen and Miller, 2007; Haczku and Panettieri, 2010). In the context of asthma, glucocorticoids are a class of steroid hormones that decrease the production of Th-1 (e.g., IFN-γ) and Th-2 (e.g., IL-5, IL-13) cytokines, thereby lowering the magnitude of airway inflammation (Banuelos and Lu, 2016). In humans, cortisol is the major glucocorticoid, and its release is regulated by the activation of the hypothalamic–pituitaryadrenal axis. Persistent secretion of cortisol associated with repeated or chronic exposure to stressors may lead to reduced expression and functioning of glucocorticoid receptors (Miller et al., 2009). Dysfunctional glucocorticoid receptors are, thus, less sensitive to the immunosuppressive action of glucocorticoids (i.e., glucocorticoid resistance [GR]), contributing to the amplification of airway inflammation (Chen and Miller, 2007).

A few studies have linked low SES, as well as other chronic psychosocial stressors, to increased Th-1 and Th-2 cell resistance to glucocorticoids, which has been indexed by the capacity of cortisol to suppress Th-1 and Th-2 cytokine production by stimulated peripheral blood mononuclear cells (PBMCs) *in vitro* (Chen et al., 2016; Miller et al., 2009). For example, in a sample of children with asthma, Chen et al. (2016) found that lower family SES was associated with higher GR of Th-1 cytokines (e.g., IFN-γ) and Th-2 (e.g., IL-5, IL-13) cytokines *in vitro*. GR related to chronic stress may be particularly problematic for children with asthma, who, as a result, can exhibit resistance to corticosteroid medications, the most common therapy for asthma control (Barnes and Adcock, 2009). Identifying protective factors against the pernicious health consequences of stress-related GR is thus critical.

A promising factor that may exert protective effects is positive parenting, which has been linked to multiple beneficial behavioral and psychological outcomes (Hoeve et al., 2009; McLeod et al., 2007) and more favorable health-related biological processes (Chen et al., 2011). Positive parenting is theorized to buffer the effects of SES primarily through its stress-buffering role that mitigates the toxic consequences of stress on health (Cohen and Wills, 1985). We investigated two forms of positive parenting, maternal involvement and warmth. Maternal involvement is a multifaceted construct that includes a broad range of behavioral, cognitive, and affective practices (e.g., monitoring, affective support) that mothers adopt to engage with children's daily lives (Finzi-Dottan et al., 2016; Pleck, 2010). Maternal warmth refers to the acceptance, love, affection, comfort, nurturance, and care that mothers display towards their children (Khaleque and Rohner, 2012). Empirical evidence suggests that maternal involvement and warmth can act as stress-buffering factors in health outcomes (Chen et al., 2011; Cohen et al., 2020; Farrell et al., 2017; Figge et al., 2020). For example, maternal involvement reduced the risk of parent cultural stress on depressive and anxiety symptoms among children from low SES families (Figge et al., 2020). Similarly, maternal warmth was reported to buffer the risk of early socioeconomic disadvantage on adult health via reduced proinflammatory signaling (Chen et al., 2011). These converging reports provide support for the hypothesis that positive parenting may buffer the detrimental effects of growing up in low SES families on health.

The aim of the current study was to investigate the buffering effects of maternal involvement and warmth on the associations among family SES, financial stress, and GR in immune cells in a sample of children with asthma. Given the reported moderation effects of positive parenting on the associations between SES and health and between psychological stress and health in previous studies (Chen et al., 2011; Cohen et al., 2020; Farrell et al., 2017; Figge et al., 2020), we proposed a moderated mediation model (see Fig. 1), in which low family SES would be associated with elevated GR via increased financial stress, and maternal involvement and warmth would moderate the associations between SES and GR and between financial stress and GR. We hypothesized that there would be stronger relationships between SES and GR and between financial stress and GR in youth who reported lower levels of maternal involvement and warmth.

2. Methods

2.1. Participants and procedure

One hundred and ninety-four youth with asthma and their caregivers took part in the Asthma in the Lives of Families Today (ALOFT) project. Data on GR was available for 145 children (10–16 years of age). Among the 145 children, two of them reported using oral steroid medications to manage their asthma. These two individuals were excluded from the analyses, resulting in a final sample of 143 children (M_{age} = 12.65 ± 1.66 years, 39.2% female, 76.2% African Americans). Participants who were excluded from the current data analyses and those included in the current study were not different on any of the variables examined in this study (lowest p = .090).

Participants were recruited from area hospitals and asthma clinics in metropolitan Detroit. Families were informed that the purpose of the study was to better understand the relationship between family daily interactions and asthma. Families were eligible for the study if they had

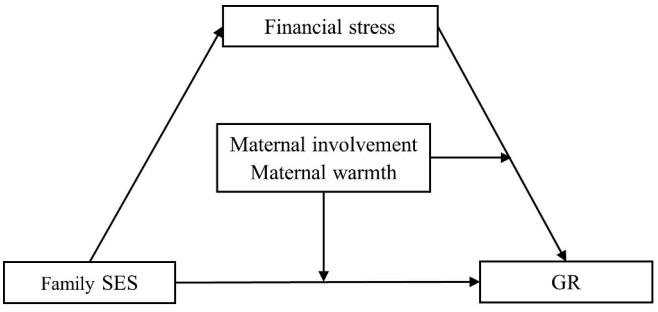

Fig. 1. The hypothesized moderated mediation model in which maternal involvement (maternal warmth) moderated the indirect pathway from family socioeconomic status (SES) to glucocorticoid resistance (GR) via financial stress.

a child aged 10–17 with a diagnosis of mild to severe asthma. Exclusion criteria included children being diagnosed with a chronic condition other than asthma or a medical condition that may interfere with immune system functioning and using oral steroid medication(s). Each child and his/her caregiver who agreed to participate in the study were invited to visit the laboratory, where they completed self-report questionnaires and a stress interview. Afterward, children were asked to complete four days of daily diaries and wore an electronic naturalistic sampling device prior to a blood draw. Participants were compensated for their time. The project was approved by the Institutional Review Board at Wayne State University. Written assent and consent were obtained from all children and their caregivers.

2.2. Measures

2.2.1. Family socioeconomic status (SES)

Two indicators of SES reported by the primary caregiver were used to derive a composite of objective family SES, their levels of education (1 = no schooling to 16 = doctoral degree) and yearly income (1 = $0-$7,825 to 6 = Over $174, 850). These two indicators were standardized and summed ($r = 0.58$) to create a composite measure of SES, with a higher score indicating higher family SES (for a similar approach, see Farrell et al., 2018)

2.2.2. Financial stress

Financial stress was assessed using the UCLA Life Stress Interview (Hammen et al., 1987, 1985), a semi-structured interview developed to evaluate chronic and acute stress across major life domains. For financial stress, children's primary caregivers were interviewed by a trained interviewer to describe their family financial state using a set of standardized questions (e.g., "have enough money to pay bills", "have outstanding loans") and follow-up prompts. The responses were then scored on a scale from 1 to 5, with increments of 0.5, where 1 indicated more than enough money for everything needed and wanted and 5 indicated hardships, poverty, lack of house, food, and health care. Therefore, a higher score reflected a higher level of chronic financial stress. Reliabilities for chronic stress ratings across the semi-structured interviews were based on independent judges' ratings of audiotaped interviews ($n = 34$). Intraclass correlations across all domains assessed in the interview for primary caregivers ranged from 0.73 − 0.98 ($M = 0.86$).

2.2.3. Maternal involvement

Our measure of maternal involvement did not focus on asthma specifically (i.e., maternal monitoring of youth's asthma management) but on two broad aspects of involvement: maternal support and monitoring. Both support (i.e., receiving help and assistance from mothers) and monitoring (i.e., open communication and self-disclosure between mother and child) are critical in buffering the deleterious effect of stress on health (Cohen and Wills, 1985; Miller et al., 2009). The Parental Environment Questionnaire (Elkins et al., 1997) was utilized to assess youth's perceptions of maternal involvement. Twelve items were used to assess maternal involvement with youth on a 4-point scale (1 = *definitely false*, 4 = *definitely true*), which focused on both maternal monitoring (e.g., "my mother [or female guardian] tries to keep up with my performance") and maternal support (e.g., "my mother [or female guardian] praises me when I do well"). Responses on the 12 items were averaged to derive a composite score of maternal involvement, with a higher score reflecting a higher level of maternal involvement. The Cronbach's alpha was 0.79 in the current study.

2.2.4. Maternal warmth

The Parental Behavior Inventory was used to assess youth's perceptions of maternal warmth (Schaefer, 1965). Specifically, 23 items were used to assess maternal warmth on a 3-point scale, ranging from 1 = agree to 3 = disagree. Sample items were "my mother (or female guardian) says I make her happy", "my mother (or female guardian) is happy to see me when I come home". Responses on the 23 items were reverse coded and averaged to create a composite score of maternal warmth, with a higher score reflecting a higher level of maternal warmth. The Cronbach's alpha was 0.95 in the current study.

2.2.5. Glucocorticoid resistance (GR)

Following the four-day daily diary period, two 8 ml peripheral blood samples were collected from each youth into Vacutainer Cell Preparation Tubes containing sodium citrate. Following a modified version of the protocol used by Weckle et al. (2015), PBMCs were isolated and resuspended in RPMI-1640 medium with HEPES supplemented with 10% Fetal Bovine Serum. PBMCs were treated with 25 ng/mL phorbol 12-myristate 13-acetate and 1 μg/mL ionomycin calcium salt dissolved in DMSO (PMA/INO) and either 28 nmol/L hydrocortisone (HC condition) or vehicle control (ethanol) (control condition), then incubated at 37 °C and 5% CO_2 for 48 h. Cell suspensions were centrifuged, and supernatants were collected and frozen at −80 °C. Concentrations of interleukin-5 (IL-5), interleukin-13 (IL-13), and interferon-γ (IFN- γ) were quantified by Quantikine ELISA (Catalog # D5000B, D1300B, DIF50, respectively) following the manufacturer's protocol (R&D Systems, Minneapolis, MN). Briefly, a standard curve for each cytokine was generated using 1:2 serial dilutions of the standard cytokine protein solution provided by the manufacturer. The detection range reported by the manufacturer for the assays was 0–250 pg/mL for IL-5, 0–4000 pg/mL for IL-13, and 0–1000 pg/mL for IFN-γ. Standard curve measurements were done in duplicates, and the average value of the two replicates was used to calculate the absolute concentration of the samples. The intra-assay coefficient of variations for IL-5, IL-13, and IFN-γ were 4.1%, 3.2%, and 4.3%, respectively. Samples were rerun if the coefficient of variation between the duplicates was above 15%. Samples in the HC and control conditions from the same participant were assayed in the same plate. The inter-assay coefficient of variation reported by the manufacturer was 5.8% for IL-5, 10.2% for IL-13, and 6.0% for IFN-γ. Levels of each cytokine in the HC and control conditions were log-transformed and then standardized. Next, these measures were used to create GR measures (i.e., IL-5, IL-13, IFN-γ), which were calculated by subtracting the corresponding control condition cytokine concentration from the HC condition cytokine concentration (for a similar approach, see Miller et al., 2009). We created a composite for GR of Th-2 cytokines by averaging values of GR of IL-5 and GR of IL-13 (for a similar approach, see Chen et al., 2016). These cytokines were selected because of their relevance to asthma and use in previous work on the effects of psychosocial stressors on inflammation among youth with asthma (e.g., Miller et al., 2009).

2.2.6. Covariates

Youth self-reported age, sex (0 = male, 1 = female), and race (0 = White, 1 = non-White). The number of parents in the home (0 = one parent, 1 = both parents) was reported by youth and their primary caregivers. Asthma medication use was reported by youth on daily logs completed across the four days prior to the blood draw. On each day, youth reported the use of (1) inhaled beta-agonist, (2) inhaled corticosteroid, (3) inhaled combination of corticosteroid and beta-agonist, and (4) leukotriene-modifying agent. In line with previous research (Chen et al., 2016), we controlled for inhaled corticosteroid use (0 = no use, 1 = use), inhaled beta-agonistic use (0 = no use, 1 = use), and leukotriene-modifying agent use (0 = no use, 1 = use) in our analyses. Participants who reported using an inhaled combination of corticosteroid and beta-agonist were coded as one on both inhaled corticosteroids use and inhaled beta-agonist use.

2.3. Statistical analyses

Path analyses were performed to test the mediation model in which family SES affected GR of Th-2 cytokines and IFN-γ via financial stress

(Hayes, 2017). Also, path analyses were used to test the moderated mediation model in which maternal involvement and warmth moderated the pathways from SES to GR and from financial stress to GR in the above mediation model. Given the moderate correlation between maternal involvement and maternal warmth ($r = 0.64$), we separately tested the moderation effects of maternal involvement and maternal warmth. The interaction terms in the moderated mediation model were calculated after mean-centering the predictor and moderators. Simple slope analyses were performed to further interpret the significant interactive effect, and the slopes were graphed using an open-source interActive tool based on ordinary least squares regression (McCabe et al., 2018). The indirect effect in the mediation model and the conditional indirect effect in the moderated mediation model were tested using the bootstrapping method (1,000 resamples). Two measures of GR (i.e., Th-2, IFN- γ) were tested in the same model to protect against the potential inflation of Type I error and were allowed to correlate with each other (Leary and Altmaier, 1980). The models were first run without covariates and then controlled for covariates. Given that medication use might not have been relevant when testing the pathway from SES to financial stress, variables for medication use were only included as covariates for GR. The incidence of missing data was 4.4%, and multiple imputation (i.e., 20 imputed datasets) was employed to deal with the missing data (Asparouhov and Muthén, 2010). The models were carried out using maximum likelihood with robust standard errors in Mplus 7.0 (Muthén and Muthén, 2012).

3. Results

3.1. Descriptive results

Table 1 displays the means, standard deviations, and correlation coefficients between study variables. Family SES and financial stress were negatively correlated with each other (r = -0.52, $p < 0.001$); however, neither of them was significantly correlated with GR of Th-2 cytokines or IFN-γ ($ps > 0.10$). Maternal involvement was positively correlated with SES ($r = 0.19$, $p = 0.030$) and negatively correlated with financial stress (r = -0.27, $p = 0.002$), but not with GR of Th-2 cytokines or IFN-γ ($ps > 0.50$). Maternal warmth was negatively correlated with financial stress (r = -0.20, $p = 0.018$), but not with SES or GR outcomes ($ps > 0.05$). A moderate positive correlation was found between maternal involvement and maternal warmth ($r = 0.64$, $p < 0.001$). GR of Th-2 cytokines was positively correlated with GR of IFN-γ ($r = 0.28$, $p = 0.001$).

3.2. The indirect effect of financial stress

The mediation model indicated a negative association between family SES and financial stress (β = -0.49, SE = 0.07, $p < 0.001$). Financial stress, however, was not associated with GR of IFN-γ ($\beta = 0.08$, $SE = 0.09$, $p = 0.42$) or GR of Th-2 cytokines ($\beta = 0.13$, $SE = 0.09$, $p = 0.15$). Family SES was also not associated with GR of IFN-γ ($\beta = 0.06$, $SE = 0.10$, $p = 0.58$) or GR of Th-2 cytokines ($\beta = 0.02$, $SE = 0.09$, $p = 0.87$). Given that obtaining confidence intervals (CIs) for the indirect effect using the bootstrapping method with multiple imputation is not available in Mplus 7.0, the first imputed dataset was used to test the indirect effect (results were similar across 20 imputed datasets). We found no evidence for the indirect pathway from family SES to GR of IFN-γ or Th-2 cytokines via financial stress (indirect effect = -0.007, 95% CI [−0.035, 0.017]; indirect effect = -0.021, 95% CI [−0.058, 0.005], respectively). The results remained very similar after adjusting for demographic covariates and medication use (see Table 2).

3.3. The moderation effect of maternal involvement

The moderated mediation model for maternal involvement showed that there were no interactive effects between maternal involvement and SES on GR of Th-2 cytokines (β = -0.19, $SE = 0.10$, $p = 0.056$) or IFN- γ (β = -0.04, $SE = 0.11$, $p = 0.75$). However, there was an interactive effect between maternal involvement and financial stress on GR of Th-2 cytokines (β = -0.40, $SE = 0.10$, $p < 0.001$), but not on GR of IFN-γ (β = -0.12, SE = 0.08, p = 0.15). The results remained similar when controlling for demographic covariates and medication use (see Fig. 2). Simple slope analyses indicated that financial stress was significantly associated with GR of Th-2 cytokines when maternal involvement was low (-1 SD), but not when maternal involvement was high (+1 SD, see Fig. 3). We also tested the conditional indirect effect of family SES on GR variables via financial stress at different levels of maternal involvement. Results showed that there was a conditional indirect effect of family SES on GR of Th-2 cytokines via financial stress at low (effect = -0.056, 95% CI [−0.111, −0.019]) but not at high levels of maternal involvement (effect = 0.025, 95% CI [−0.003, 0.070]). No evidence for significant conditional indirect effects from family SES to GR of IFN-γ via financial stress emerged as a function of maternal involvement (at −1 SD, effect = -0.014, 95% CI [−0.047, 0.016]; at + 1 SD, effect = 0.013, 95% CI [−0.018, 0.050]).

3.4. The moderation effect of maternal warmth

Similar to the moderation effect of maternal involvement reported above, maternal warmth did not moderate the pathway from SES to GR of Th-2 cytokines (β = -0.14, $SE = 0.09$, $p = 0.095$) or IFN-γ (β = -0.02, SE = 0.09, p = 0.86). Maternal warmth, however, moderated the pathway from financial stress to GR of Th-2 cytokines (β = -0.30, SE = 0.10, p = 0.002) but not IFN-γ (β = -0.09, SE = 0.10, p = 0.40). The results remained similar after controlling for demographic covariates and medication use (see Fig. 4). Simple slope analyses indicated that financial stress was significantly associated with GR of Th-2 cytokines when maternal warmth was low, but not when maternal involvement was high (see Fig. 5). In addition, results for the conditional indirect effect showed that there was a conditional indirect effect of family SES on GR of Th-2 cytokines via financial stress at low (effect = -0.082, 95% CI [−0.169, −0.030]) but not at high levels of maternal warmth (effect = 0.027, 95% CI [−0.008, 0.083])[1]. No evidence for significant conditional indirect effects from family SES to GR of IFN-γ via financial stress emerged as a function of maternal warmth (at −1 SD, effect = -0.010, 95% CI [−0.064, 0.037]; at + 1 SD, effect = 0.003, 95% CI [−0.032, 0.043]).

4. Discussion

This study tested the associations among family SES, financial stress, maternal involvement and warmth, and GR in a sample of youth with asthma. We found that family SES was associated with financial stress. Family SES and financial stress were not associated with GR of Th-2 cytokines or IFN-γ. However, financial stress–but not family SES–interacted with both maternal involvement and warmth in influencing GR of Th-2 cytokines. Specifically, greater financial stress was associated with higher GR of Th-2 cytokines in youth reporting low levels of maternal involvement and warmth but not in those with high levels of maternal involvement or warmth. Overall, an indirect pathway from low

[1] Sensitivity analyses were performed to test the mediation model and the moderated mediation model for two individual Th-2 cytokines (i.e., IL-5, IL-13) that were significantly correlated with each other ($r = 0.51$, $p < 0.001$). A similar pattern of results was found for GR of IL-5 and IL-13. That is, family SES and financial stress were not associated with GR of IL-5 or IL-13 ($ps > 0.10$). However, maternal involvement and warmth moderated the association between financial stress and GR of IL-5 ($ps < 0.01$) and the association between financial stress and GR of IL-13 ($ps < 0.05$), above and beyond the effects of covariates. Maternal involvement and warmth did not moderate the association between family SES and GR of IL-5 or IL-13 ($ps > 0.10$).

Table 1
The mean, standard deviation (SD), and correlation coefficients between study variables.

Variables	Mean (SD)	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Family SES	0.03(1.76)	–												
2. Financial stress	3.12(0.86)	-0.52***	–											
3. Maternal involvement	3.45(0.49)	0.19*	-0.27**	–										
4. Maternal warmth	2.69(0.37)	0.14	-0.20*	0.64***	–									
5. GR of Th-2 cytokines	−0.00 (0.58)	-0.05	0.12	0.05	0.04	–								
6. GR of IFN-γ	0.00(0.53)	0.02	0.07	0.07	0.15	0.28**	–							
7. Female	56(39.2)[a]	-0.12	0.02	0.04	-0.10	-0.15	-0.24**	–						
8. Non-White	113(79.0)[a]	-0.48***	0.43***	-0.17*	-0.07	-0.06	-0.04	0.03	–					
9. Number of parents in the home	78(54.9)[a]	0.15	-0.26**	0.16	0.08	-0.11	-0.08	-0.05	-0.33***	–				
10. Age	12.65 (1.66)	0.14	-0.18*	-0.04	-0.16	-0.04	0.02	0.15	-0.27**	0.01	–			
11. Leukotriene-modifying agent MU	15(12.6)[a]	0.13	-0.27**	0.13	0.04	0.04	-0.16	-0.01	-0.27**	0.03	0.15	–		
12. Beta-agonist MU	36(30.3)[a]	0.19*	-0.09	0.13	0.06	0.15	0.03	-0.07	-0.33***	-0.02	0.04	0.14	–	
13. Corticosteroid MU	53(44.5)[a]	0.05	0.00	-0.02	0.01	0.26**	0.11	-0.13	-0.14	-0.00	0.11	0.12	0.51***	–

Note. SES = socioeconomic status; GR = glucocorticoid resistance; Th-2 = T-helper 2; IFN-γ = interferon-γ; MU = medication use.
[a] display as *N* (%).
* $p < .05$, ** $p < .01$, *** $p < .001$.

Table 2
Results for the mediation model of SES on GR via financial stress.

	Financial stress			GR of IFN-γ			GR of Th-2 cytokines		
Variables	*β*	*SE*	*p*	*β*	*SE*	*p*	*β*	*SE*	*p*
Family SES	−0.38	0.08	<0.001	−0.03	0.10	0.76	−0.05	0.09	0.57
Financial stress	–	–	–	0.03	0.10	0.79	0.13	0.10	0.19
Female	−0.04	0.15	0.78	−0.52	0.15	0.001	−0.24	0.16	0.13
Non-White	0.45	0.20	0.022	−0.24	0.30	0.43	−0.34	0.22	0.12
Number of parents in the home	−0.27	0.16	0.083	−0.19	0.18	0.29	−0.23	0.19	0.22
Age	−0.08	0.07	0.29	0.05	0.09	0.55	−0.07	0.08	0.39
Leukotriene-modifying agent MU	–	–	–	−0.43	0.30	0.16	0.17	0.24	0.48
Beta-agonist MU	–	–	–	0.01	0.20	0.96	0.03	0.20	0.90
Corticosteroid MU	–	–	–	0.15	0.19	0.44	0.47	0.20	0.021
R^2	0.31***								
				0.10*			0.13*		

Note. SES = socioeconomic status; GR = glucocorticoid resistance; Th-2 = T-helper 2; IFN-γ = interferon-γ; MU = medication use.
* $p < .05$, *** $p < .001$.

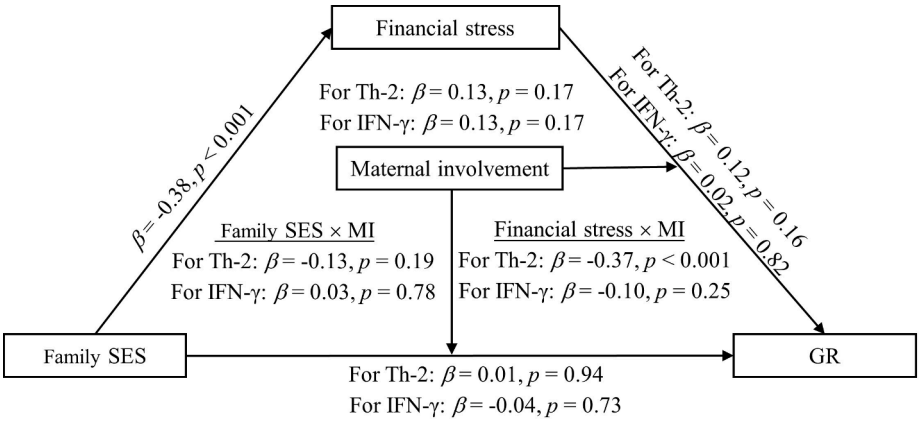

Fig. 2. The moderated mediation model of maternal involvement (MI) on the indirect pathway from family socioeconomic status (SES) to glucocorticoid resistance (GR) via financial stress. Covariates (age, sex, race, number of parents in the home, and medication use) were included in the model but not displayed for simplification. Th-2 = T-helper 2; IFN-γ = interferon-γ.

family SES to elevated GR of Th-2 cytokines via increased financial stress was found among youth who reported low levels, but not high levels, of maternal involvement and warmth.

The moderation effects of maternal involvement and warmth found in the current study align with existing work highlighting the protective role played by positive parenting in health for children from low SES families (Chen and Miller, 2013), as well as previous empirical studies showing the buffering effect of positive parenting on the association between chronic stress and health outcomes among adults from low SES backgrounds (e.g., Cohen et al., 2020; Farrell et al., 2017). These findings add to the existing literature by showing that parenting characterized as high levels of involvement and warmth may have stress-buffering effects on the association between financial stress and decreased glucocorticoid sensitivity of immune cells of youth with

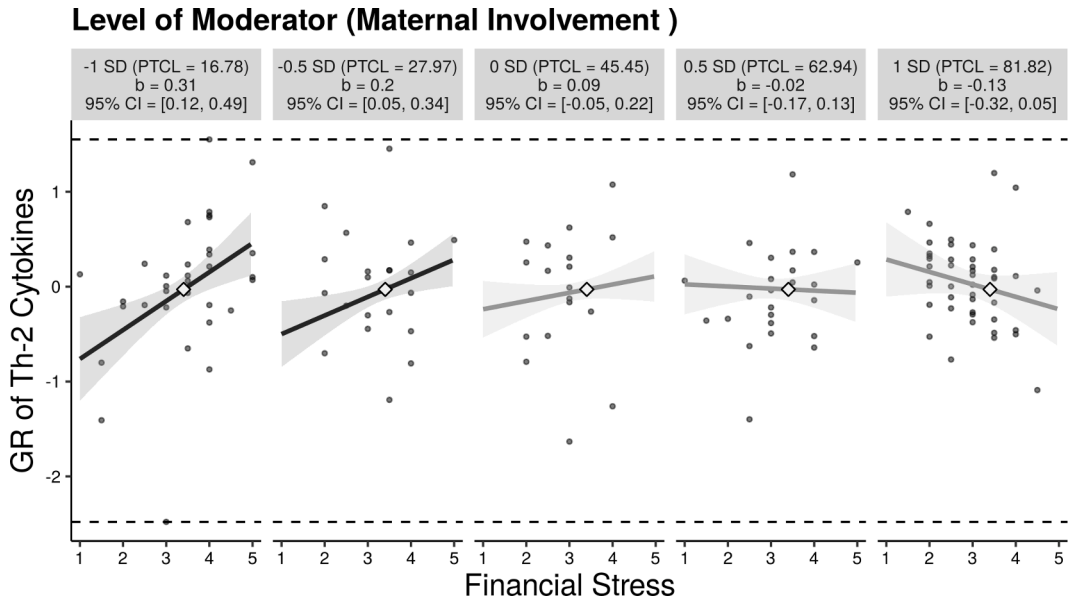

Fig. 3. The simple slopes for the effect of financial stress on GR of T-helper 2 (Th-2) cytokines by levels of maternal involvement. GR = glucocorticoid resistance; PTCL = percentile. Plots were generated using the first imputed dataset given that multiple imputation was not available in the open-source interActive tool developed by McCabe and colleagues (2018). Plots were similar when using the other 19 imputed datasets.

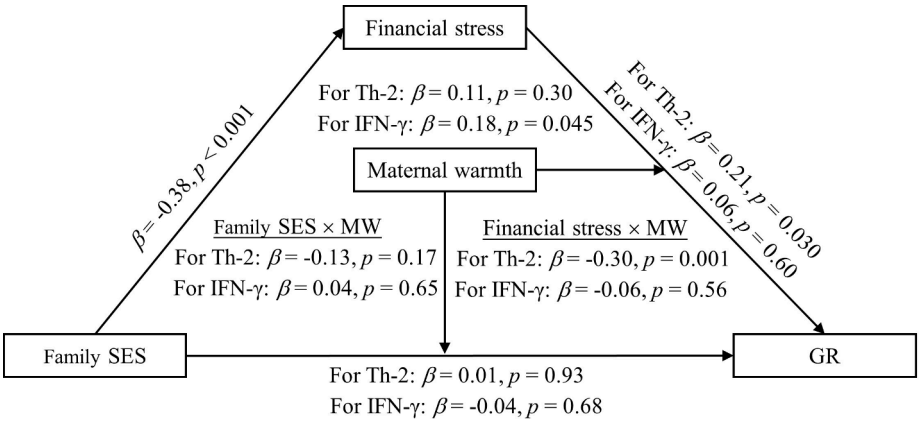

Fig. 4. The moderated mediation model of maternal warmth (MW) on the indirect pathway from family socioeconomic status (SES) to glucocorticoid resistance (GR) via financial stress. Covariates (age, sex, race, number of parents in the home, and medication use) were included in the model but not displayed for simplification. Th-2 = T-helper 2; IFN-γ = interferon-γ.

asthma. More importantly, the consistent moderation effects observed for maternal involvement and warmth highlight that positive parenting practices facilitating a supportive and warm mother–child relationship might be fruitful targets for interventions that aim to improve asthma-related health outcomes for youth living in low SES families. Although positive parenting practices and SES might not be completely independent (e.g., a significant correlation between low SES and low maternal involvement was found in our sample), our study and others (Chen et al., 2011; Farrell et al., 2017; Imami et al., 2015) have found that positive parenting can be found across all levels of the SES spectrum. Mothers can be resilient and remain supportive and responsive despite the challenges posed by low SES environments. Germane to this point, interventions designed to promote positive parenting (e.g., maternal involvement) among low SES samples have been shown to be effective (Shaw et al., 2006) and have been reported to promote health outcomes in youth (Morris et al., 2017).

We found that the moderation effects of maternal involvement and warmth were observed for GR of Th-2 cytokines but not GR of IFN-γ. This finding is somewhat consistent with a previous study showing that children with asthma reporting higher levels of secure attachment, which is likely nurtured by supportive and responsive parenting (Hong and Park, 2012), produced lower levels of Th-2 cytokines (e.g., IL-5, IL-13) but not Th-1 cytokines (e.g., IFN-γ) in response to PMA/INO stimulation (Ehrlich et al., 2019). Th-2 cells have long been recognized as a key driver for asthma pathogenesis (Akbari et al., 2003; Durrant and Metzger, 2010). For example, IL-5 cytokine production has been found to predict eosinophil activity in children with asthma (Chan et al., 2015). On the other hand, Th-1 cells have been suggested to have a beneficial role in asthma by downregulating the effect of Th-2 cells (Huang et al., 2001). However, the view that Th-1 and Th-2 cells have opposing effects on asthma should be considered with caution, since Th-1 cells, primarily through the action of IFN-γ, have been suggested to contribute to inflammation in asthma (Durrant and Metzger, 2010). Also, we should note that certain psychosocial factors have been linked to both GR of Th-1 and Th-2 cytokines in children with asthma (Miller et al., 2009). For example, Miller et al. (2009) found that parental support was negatively associated with GR of Th-1 (e.g., IFN-γ, lower parental support, greater GR) and Th-2 cytokines (e.g., IL-5) among

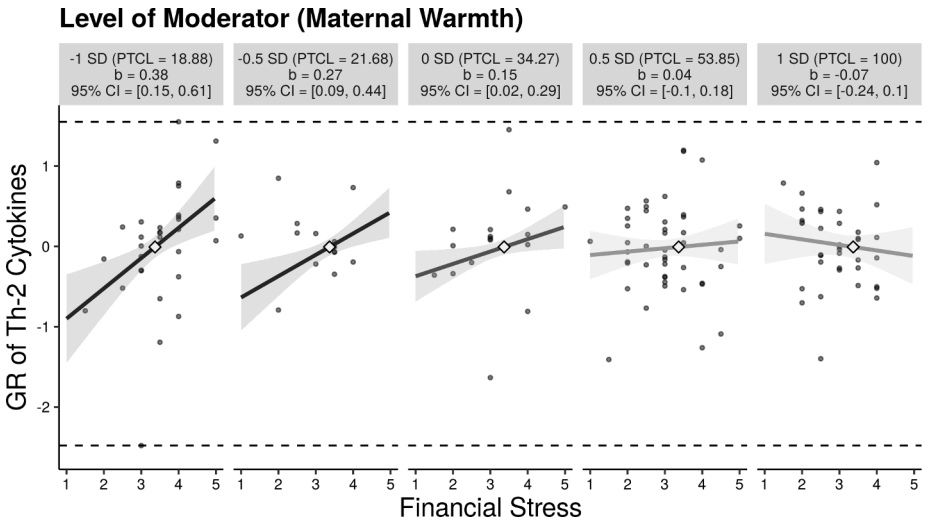

Fig. 5. The simple slopes for the effect of financial stress on GR of T-helper 2 (Th-2) cytokines by levels of maternal warmth. GR = glucocorticoid resistance; PTCL = percentile. Plots were generated using the first imputed dataset given that multiple imputation was not available in the open-source interActive tool developed by McCabe and colleagues (2018). Plots were similar when using the other 19 imputed datasets.

children with asthma but not among children without asthma. Future studies are warranted to corroborate whether the moderation effects of maternal involvement and warmth are specific to GR of Th-2 cytokines.

Unexpectedly, we did not find an indirect effect of family SES on GR of IFN- γ or Th-2 cytokines via financial stress. Neither family SES nor financial stress was directly associated with these two GR measures. These results contradict previous studies reporting a significant association between lower SES and elevated GR of immune cells (e.g., Chen et al., 2016). One potential explanation for these mixed results may be related to the differences in the sample characteristics between previous research (a significant percentage of White youth) and our sample (predominantly African American youth, 76.2% of the sample). SES disparities in health have been found to be more pronounced among Whites than African Americans (e.g., Farmer and Ferraro, 2005), presumably due to the fact that high-SES African Americans do not experience the same health benefits as high-SES Whites (Assari et al., 2018). Assari et al. (2018) have suggested that some social barriers (e.g., discrimination) may limit the potential health gains associated with high SES among African American youth. Unfortunately, because of the small number of children who self-identified as White in our sample, we cannot directly test this explanation in the current study.

Several limitations should be noted for this study. First, the data were cross-sectional. Longitudinal designs are needed to establish the temporal nature of the associations among family SES, financial stress, maternal involvement and warmth, and GR. Second, this study only examined the buffering effects of maternal involvement and warmth. Research on parenting has long focused on the mother–child dyad, given that mothers are usually the primary caregivers of youth. However, recent research has emphasized the importance of examining both maternal and paternal parenting (Cabrera et al., 2018; Volling and Cabrera, 2019). Future studies would benefit from examining the combined and unique buffering effects of maternal and paternal involvement and warmth on the SES-health link. Third, the SES and financial stress data were only collected from the primary caregiver and may not accurately reflect the SES condition of the family as a whole. Fourth, this study only focused on Th-1 and Th-2 cytokines (i.e., IL-5, IL-13, IFN-γ); however, it is possible that other pro-inflammatory cytokines (e.g., IL-6) may also contribute to poor asthma outcomes (Neveu et al., 2010). Future studies are needed to examine whether the buffering effects of positive parenting reported in this study generalize to GR of other immune markers.

Despite these limitations, findings from this study support the idea that warm and supportive parenting has the potential to foster better physical health despite the challenges put forth by socioeconomic disadvantage and financial stress. This work indicates that positive parenting can get under the skin and buffer the adverse effect of financial stress on offspring's immune responses.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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