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BRIEF REPORT

Self-Reported Parenting Style Is Associated With Children's Inflammation and Immune Activation

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Family environments and parenting have been associated with inflammation and immune activation in children and adolescents; however, it remains unclear which specific aspects of parenting drive this association. In this study, we cross-sectionally examined the association between 5 discrete parenting styles and inflammation and immune activation in late childhood. Data were drawn from 102 families (55 with female children, mean age 9.50 years, $SD = 0.34$) participating in the Imaging Brain Development in the Childhood to Adolescence Transition Study. Children provided saliva samples from which inflammation (C-reactive protein) and immune competence/activation (secretory immunoglobulin A) were measured. Parents completed the Alabama Parenting Questionnaire, which measures 5 aspects of parenting style—positive parental involvement, positive disciplinary techniques, consistency in disciplinary techniques, corporal punishment, and monitoring and supervision. Results showed that higher scores on the poor parental monitoring scale were associated with higher levels of both inflammation and immune activation in children. This study highlights parental monitoring and supervision as a specific aspect of parenting behavior that may be important for children's physical and mental health.

Keywords: parenting behavior, inflammation, immune activation, poor monitoring, physical health

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Recent research has shown that early adverse environments can have an effect on inflammatory profiles and immune activation in children and adolescents (Miller & Chen, 2013). Inflammation in particular appears to be a key mechanism associated with both mental and physical health. Inflammatory processes are known to respond to stress regardless of whether the stressor is physical or psychosocial (Hennessy et al., 2004), suggesting that stressful social environments may be associated with differences in levels of inflammation. Indeed, conflictual relationships are a form of psychosocial stress that has been linked with elevated inflammation in adults (Kiecolt-Glaser et al., 2005), while supportive interpersonal relationships have been linked with lower levels of inflammatory markers (e.g., Lutgendorf, Anderson, Sorosky, Buller, & Lubaroff, 2000).

Given that the family environment is an important source of both psychosocial support and stress in childhood, we might expect variations in family functioning to be associated with children's levels of inflammation. For example, Bronfenbrenner's ecological systems theory of development (i.e., development in context) states that the family is a group in the child's microsystem that most directly influences the child's psychological development (Bronfenbrenner, 1992). As personal relationships have been shown to influence physical and immune health as well (discussed above), we posit that stressful family environments in particular may also play a significant role in the child's immune health. Consistent with this, harsh family climates (i.e., how often parents insulted, threatened, or swore at children or behaved violently, as reported by adolescent girls) have been associated with proinflammatory phenotypes (Miller & Chen, 2010). Although there is consistent research showing that broad childhood maltreatment is associated with elevated inflammation later in life, these studies have not identified parenting styles that are important for children's inflammatory processes. Although a recent observational study (O'Connor et al., 2015) has provided important objective data on the relationship between parental emotional behavior and offspring immune activation, research still lacks detailed measurement of parenting styles (i.e., different types of negative and positive parenting practices) that might be associated with systemic inflammation and immune activation, such as supervision, punishment, reward, and inconsistency. Identifying specific parenting behaviors may inform interventions that aim to improve parenting skills and consequent outcomes for children.

A promising and feasible way to explore this issue in children is to combine established self-report measures of parenting behavior with measures of inflammation and immune activation in saliva. Compared with blood, saliva is safer and easier to collect in research studies (Granger et al., 2007). In particular, saliva has been found to be correlated with systemic or major sources of the general inflammatory marker, C-reactive protein (CRP; Byrne et al., 2013; Ouellet-Morin, Danese, Williams, & Arseneault, 2011; Out, Hall, Granger, Page, & Woods, 2012). Those studies showed medium to large effect sizes for correlations between serum or plasma and salivary CRP, although one study found that salivary and plasma CRP were not significantly correlated (Dillon et al., 2010). Higher levels of CRP have been shown to be related to retrospective reports of risky early family environments (Taylor, Lehman, Kiefe, & Seeman, 2006). In particular, studies with children have shown that salivary levels of CRP are associated with various measures of physical health (Goodson et al., 2014; Naidoo, Konkol, Biccard, Dudose, & McKune, 2012).

Whole saliva is also a reliable source of a mucosal antibody called secretory immunoglobulin A (SIgA; Brandtzaeg, 2007), a measure of immune competence or activation. Its main function is to provide the first level of defense against pathogens that enter the mucosal lining (by inhibiting or killing bacteria and neutralizing viruses), and it is therefore a measure of resistance to infectious disease (see Tomasi, 1970). Research has shown that in children, higher levels of SIgA interact with puberty to cross-sectionally predict depressive symptoms (Delany et al., 2016).

The aim of the current study was to examine the association between different parenting styles and children's levels of inflammation and immune activation. Consistent with previous research, we predicted that positive parenting practices would be associated with lower levels of inflammation and immune activation in offspring and that negative parenting practices (i.e., poor supervision and monitoring, inconsistent discipline, and the use of corporal punishment) would be associated with higher levels of inflammation and immune activation in offspring. Given previously identified associations between both depression and/or inflammation and body mass index (BMI; e.g., Stewart, Rand, Muldoon, & Kamarck, 2009), sex (e.g., Bouman, Heineman, & Faas, 2005), age (e.g., Jorm, 1987), pubertal group (Delany et al., 2016), and socioeconomic status (SES; e.g., Owen, Poulton, Hay, Mohamed-Ali, & Steptoe, 2003), these variables were also included in analyses as potential confounders.

Method

The data used in this study were collected from the Imaging Brain Development in the Childhood to Adolescence Transition Study (iCATS), conducted through The University of Melbourne and Murdoch Childrens Research Institute. Recruitment for iCATS occurred within the larger Childhood to Adolescence Transition Study (CATS) cohort. Information regarding the recruitment procedure, participants, methods, and broad aims of iCATS (Simmons et al., 2014) are provided elsewhere. Briefly, children were selected for iCATS if their combined salivary dehydroepiandrosterone and testosterone levels during the CATS baseline assessment fell into the upper or lower tertiles of the larger CATS cohort ("pubertal group"). This was conducted separately for boys and girls. Children with high levels of these hormones were considered to be relatively early developers and children with low levels of these hormones were considered to be relatively late developers. Groups (early developing vs. late developing) were intentionally matched for age to maximize our ability to comment on the effects of relative timing of adrenarche. The iCATS subsample comprised of 128 children (10.3% of the 1,239 children in the larger CATS cohort) with 66 children (35 female) participating from the early developing group, and 62 children (33 female) from the late developing group.

One-hundred thirteen children provided saliva samples for immune analysis in this study. Eleven participants were excluded from analysis due to the use of medications that would affect immunological function (e.g., allergy or asthma medicine, flu shot) in the 24 hr before saliva sample collection. Therefore, a sample size of 102 was used for analysis, including 52 early developers (30 female) and 50 late developers (25 female). Participants and

their parents/guardians provided informed consent and were reimbursed for their participation. Ethics approval was granted by the Royal Children's Hospital Human Research Ethics Committee (Reference Number 32171).

Measures

Independent variables. Parenting behavior (81.6% mothers) was measured using the Alabama Parenting Questionnaire (APQ; Shelton, Frick, & Wootton, 1996). The APQ is a parent self-report 42-item questionnaire that measures: positive parental involvement (Involvement), positive disciplinary techniques (Positive Parenting), consistency in disciplinary techniques (Inconsistent Discipline), corporal punishment, and supervision and monitoring (Poor Monitoring). It asks how often each item "typically" occurs in the home on a 5-point Likert scale ranging from 1 (*never*) to 5 (*always*). It has shown good internal consistency, validity, and test–retest reliability in Australian children (Dadds, Maujean, & Fraser, 2003). Cronbach's alpha coefficients of reliability were medium to high consistency: 0.681 (Involvement), 0.745 (Positive Parenting), 0.600 (Poor Monitoring/Supervision), 0.748 (Inconsistent Discipline), and 0.663 (Corporal Punishment), and are similar to or better than those from a larger sample of Australian parents and children in the same age range (Dadds et al., 2003).

Dependent variables. Immune markers (CRP—inflammation, and SIgA—immune activation) were collected and assayed from unstimulated, whole saliva, collected at waking, using the "passive drool" method. Specific information regarding the collection and processing of saliva can be found elsewhere (Delany et al., 2016). Immunological assays were conducted at Murdoch Childrens Research Institute using Salimetrics enzyme-linked immunosorbent assay kits (Stratech Scientific APAC Pty Ltd., Sydney, Australia). Kits from the same lot numbers were used, as were in-house controls. The interassay coefficients of variation were SIgA = 5.00% and CRP = 1.05%; the intraassay coefficients of variation were SIgA = 5.08% and CRP = 3.48%.

Potential confounding factors. BMI (kg/m^2) was calculated using weight (kg) and height (m^2) using Tanita HD 382 digital scales (Tanita Australia; Kewdale, WA, Australia) and a portable rigid Invicta stadiometer (QuickMedical; Issaquah, WA, USA). A measure of SES by area was calculated from participants' postcodes by using the Socioeconomic Indexes for areas codes ("Index of Relative Socioeconomic Advantage and Disadvantage") by the Australian Bureau of Statistics (Pink, 2006). These indices determine the level of social and economic well-being in each region (higher scores represent higher SES). They include 31 variables pertaining to neighborhood disadvantage, including income, unemployment, occupation, and education.

Statistical Analysis

Some data were missing from the APQ Involvement (1.9%), Positive Parenting (1.9%), Poor Monitoring (3.9%), and Corporal Punishment (1.0%) scales, and two saliva samples were missing CRP values due to assay error (1.9%). Little's test indicated that data was missing completely at random, $\chi^2 = 32.465$ ($df = 706$; $p = 1.000$). To preserve statistical power lost through deletion methods, we used single imputation (Expectation Maximization) using 100 iterations. Due to the individual variability of saliva flow and the speed of analytes transported through cell membranes, CRP and SIgA were adjusted for flow rate (mL/s). These variables were also log transformed and outliers (>3 *SD*) were winsorized to normalize data. Scale variables were treated as continuous data. Independent samples *t* tests were used to detect significant sex differences across all variables.

Hierarchical multiple linear regression analyses were conducted with CRP and SIgA as respective dependent variables (DVs). The potential covariates of age, sex, socioeconomic status, BMI, and pubertal group were entered in the first step, and the APQ parenting scales were entered as independent variables in the second step. Given the likelihood of multicollinearity, the two respective immune variables ($r = .533$) were analyzed in separate regression models (Model 1: DV = CRP; Model 2: DV = SIgA). Statistical analyses were performed using SPSS for Windows, Version 22.

Results

Table 1 presents means and standard deviations for all continuous variables, both for the total sample and for females and males

Table 1
Descriptive Statistics for Parenting Behavior, Immune Markers, and Demographic Characteristics

Variable	Total ($N = 102$) $M \pm SD$	Females ($n = 55$) $M \pm SD$	Males ($n = 47$) $M \pm SD$	t, p
APQ Involvement	40.81 ± 3.61	40.70 ± 3.92	40.94 ± 3.25	.332, .740
APQ Positive Parenting	25.33 ± 2.41	25.27 ± 2.56	25.41 ± 2.25	.290, .773
APQ Poor Monitoring	12.05 ± 2.46	12.01 ± 2.89	12.09 ± 1.86	.176, .861
APQ Inconsistent Discipline	13.33 ± 3.32	13.31 ± 3.08	13.36 ± 3.62	.079, .937
APQ Corporal Punishment	5.77 ± 1.59	5.62 ± 1.60	5.96 ± 1.56	1.078, .284
CRP (pg/ml)[a]	193.52 ± 656.63	263.35 ± 856.29	111.79 ± 270.70	−1.164, .247
Log(CRP)[b]	1.52 ± .66	1.55 ± .69	1.48 ± .61	−.524, .601
SIgA (μg/ml)[a]	4.79 ± 11.06	4.82 ± 7.24	4.75 ± 14.38	−.032, .974
Log(SIgA)[b]	.33 ± .46	.39 ± .48	.27 ± .42	−1.294, .199
Age (years)	9.50 ± .34	9.47 ± .32	9.53 ± .36	.911, .365
SES	1,039.89 ± 40.82	1,044.69 ± 42.10	1,034.28 ± 38.96	−1.289, .201
BMI	17.74 ± 2.36	18.11 ± 2.70	17.31 ± 1.82	−1.725, .088

Note. APQ = Alabama Parenting Questionnaire; SES = socioeconomic status; BMI = body mass index.
[a] Adjusted for flow rate (multiplied by weight/seconds). [b] Additionally log transformed and winsorized.

separately. Independent samples *t* tests showed no significant sex differences for any of these variables, although differences for BMI approached significance.

Two-tailed correlations between all continuous variables are presented in supplemental material. Results of hierarchical multiple regression analyses are displayed in Table 2. For Model 1 (DV = CRP), the covariates accounted for only 2.2% (0% adjusted) of the variance in CRP, $F(5, 96) = 0.435$, $p = .823$. Following the inclusion of the APQ scales in the second step, the full model with all predictors accounted for 17.2% (8.1% adjusted) of the variance in CRP, $F(10, 91) = 1.885$, $p = .057$, indicating that APQ scores significantly predicted CRP over and above the effects of age, sex, socioeconomic status, BMI, and pubertal group. However, Poor Monitoring was the only significant, independent predictor of CRP, uniquely accounting for 8.9% of the variance in the DV. For Model 2 (DV = SIgA), the covariates significantly accounted for 13.0% (8.5% adjusted) of the variance in SIgA, $F(5, 96) = 2.881$, $p = .018$, but the full model including the APQ scales predicted 22.5% (14% adjusted) of the variance in the DV, $F(10, 91) = 2.639$, $p = .007$. Both BMI and APQ Poor Monitoring independently and significantly predicted SIgA, accounting for 7.1% and 5.2% of the variance in this variable, respectively. As expected, Poor Monitoring was significantly related to immune markers in both models, such that higher scores on Poor Monitoring were associated with higher levels of inflammation and immune activation in children.

Discussion

In this study of community-based families, self-reported poor monitoring and supervision by parents was associated both with higher levels of inflammation in their children, as measured by salivary CRP, and higher levels of immune competence/activation, as measured by salivary SIgA. Higher BMI was also associated with higher SIgA. Notably, poor monitoring still significantly predicted inflammation and immune competence after controlling for age, sex, SES, and pubertal group. No other parenting scale significantly predicted child immune markers.

This study aligns with other research showing that negative emotional parental behavior (O'Connor et al., 2015) and risky family environments (Miller & Chen, 2010) are associated with adolescent immune activation and inflammatory profiles, respectively. However, our results also suggest that there are specific aspects of stressful early environments, particularly a lack of

Table 2
Hierarchical Regression Models for C-Reactive Protein (CRP) and Secretory Immunoglobulin A (SIgA; Transformed)

Hierarchical regression step						95% CI for *B*			
	Log(CRP)	*B*	β	*t*	*p*	Lower	Upper	*r*	sr^2
Block 1	Age	.183	.095	.874	.384	−.232	.598	.094	.008
$R^2 = .022$	Sex	.089	.068	.649	.518	−.183	.361	.052	.004
	SES	−.001	−.064	−.623	.535	−.004	.002	−.052	.004
	BMI	−.008	−.027	−.243	.808	−.070	.054	.040	.001
	Pubertal group	.098	.075	.677	.500	−.188	.383	.099	.005
Block 2	Age	.181	.094	.894	.374	−.222	.584	.094	.007
	Sex	.084	.064	.637	.526	−.178	.347	.052	.004
	SES	.000	−.029	−.287	.775	−.004	.003	−.052	.001
	BMI	−.011	−.039	−.356	.723	−.071	.050	.040	.001
	Pubertal group	.101	.077	.729	.468	−.173	.375	.099	.005
	APQ Involvement	−.027	−.147	−1.282	.203	−.068	.015	−.169	.015
	APQ Positive Parenting	.039	.142	1.325	.188	−.019	.097	.039	.016
	APQ Poor Monitoring	.087	.326	3.134	.002	.032	.142	.330	.089
	APQ Inconsistent Discipline	−.031	−.156	−1.443	.152	−.073	.012	−.057	.019
	APQ Corporal Punishment	−.018	−.043	−.394	.695	−.106	.071	−.115	.001
		R^2 change = .149 ($p = .009$)							
	Log(SIgA)								
Block 1	Age	.083	.062	.602	.548	−.191	.357	.143	.003
$R^2 = .130$	Sex	.074	.080	.811	.419	−.106	.254	.128	.006
	SES	.000	−.005	−.053	.958	−.002	.002	−.008	.000
	BMI	.059	.306	2.880	.005	.018	.100	.347	.075
	Pubertal group	.029	.031	.300	.765	−.160	.217	.164	.001
Block 2	Age	.082	.061	.595	.554	−.191	.355	.143	.003
	Sex	.065	.071	.731	.467	−.113	.243	.128	.004
	SES	.000	.023	.236	.814	−.002	.002	−.008	.000
	BMI	.060	.307	2.890	.005	.019	.101	.347	.071
	Pubertal group	.024	.026	.258	.797	−.162	.210	.164	.001
	APQ Involvement	−.015	−.117	−1.058	.293	−.043	.013	−.135	.010
	APQ Positive Parenting	.013	.071	.680	.498	−.026	.053	.012	.004
	APQ Poor Monitoring	.046	.248	2.465	.016	.009	.084	.267	.052
	APQ Inconsistent Discipline	−.019	−.136	−1.302	.196	−.047	.010	−.060	.014
	APQ Corporal Punishment	−.018	−.062	−.593	.555	−.078	.042	−.079	.003
		R^2 change = .094 ($p = .059$)							

Note. CI = confidence interval; APQ = Alabama Parenting Questionnaire; SES = socioeconomic status; BMI = body mass index.

parental supervision, which might be especially deleterious for children's health. One explanation for this finding is that parental supervision represents a marker of the quality of the parent–child relationship, with poor monitoring indicating parental caregiving difficulties, leading to chronic stress that derails normal neurobiological and physiological development. However, other dimensions of parenting behavior examined in the current study are also likely to be markers of the quality of the parent child relationship, so this does not fully explain the specificity of the effect. Another possibility is that the relationship between monitoring and inflammation is mediated by externalizing difficulties. Poor parental monitoring has been consistently linked to externalizing and antisocial behavior in children and adolescents (e.g., Blustein et al., 2015), and, although research is limited, studies have shown that elevated CRP (Slopen, Kubzansky, & Koenen, 2013) and SIgA (Keller, El-Sheikh, Vaughn, & Granger, 2010) are associated with behavior and adjustment problems in children. However, inconsistent discipline has also been associated with externalizing behavior in children (e.g., Ge, Brody, Conger, Simons, & Murry, 2002). Finally, the lack of a significant association between corporal punishment and immune markers in the current study might be due to a floor effect (i.e., this scale had the lowest mean and standard deviation, possibly reflecting both a lack of variability in the sample and/or underreporting due to social desirability bias).

Although speculative, it may be that the disengagement from parenting reflected by poor monitoring represents a form of social stress that is particularly associated with inflammation and immune activation, more than overtly conflictual dimensions of the parent–child relationship. An immune pathway that is particularly sensitive to social stress (such as rejection) has been proposed as a mechanism for depression (Slavich & Irwin, 2014). It is possible that the child's perception of poor monitoring results in a perception of social rejection. Future research with larger sample sizes and greater variability in both parenting and child behavior should (a) examine the way in which various types of parenting behavior are perceived by the child and (b) carefully measure children's externalizing behavior to see if this is associated with both parental monitoring and children's immune health.

The values of salivary CRP in our sample (M = 193.52 pg/ml, median = 22.03 pg/ml) were intermediate with those previously reported in other studies of children and adolescents (Byrne et al., 2013; Goodson et al., 2014; Naidoo et al., 2012), and lower than those in studies of adults (Laurent, Lucas, Pierce, Goetz, & Granger, 2016; Lucas et al., 2016; Mohamed, Campbell, Cooper-White, Dimeski, & Punyadeera, 2012; Ouellet-Morin et al., 2011; Out et al., 2012). The lower concentration of CRP in our sample may be due to the younger (and physically healthier) sample. However, currently, we are unable to strongly interpret the values in our sample compared to samples in other studies because there are no validated norms by age for this measure yet. Therefore, more research on salivary CRP norms, which also account for flow rate, is needed for children.

Furthermore, our values of SIgA (4.79 μg/ml = mg/L) were lower than those from two studies of children and adults (Fagerås, Tomičić, Voor, Björkstén, & Jenmalm, 2011; Kugler, Hess, & Haake, 1992), but comparable to those in a study of 14-year-old adolescents (Sonesson, Hamberg, Wallengren, Matsson, & Ericson, 2011). The effect of pubertal timing on levels of SIgA across childhood has not yet been addressed. Our sample included two groups of children that differed on levels of dehydroepiandrosterone and testosterone, and this could have affected mean values of SIgA.

There are limitations to the current study that should temper our interpretations and be addressed by future research. First, although we excluded participants who were taking medication that could affect immune functioning, we did not measure temperature, other illness, or dental hygiene. Second, this was a cross-sectional study, so further longitudinal research is needed to determine if parenting behavior is a prospective risk factor or simply a correlate of inflammation and immune activation. Relatedly, future longitudinal and intervention research in this area could identify specific mechanisms that help explain the relationship between parenting behavior and children's physical health. Such studies could combine measures of parenting style with methods that capture change in the child's behavior, mood, perceived stress, and brain development over time. As discussed above, if poor parental supervision is an index of externalizing behavior, which is, in turn, associated with inflammation or immune activation, longitudinal research could elucidate the temporal relationships between these variables. More specifically, child behavior may be directly influenced by parental monitoring, potentially impacting future physical health. Therefore, poor monitoring could be a modifiable risk factor that represents a promising target for intervention strategies aiming to improve both behavioral and physical health outcomes in children. Parental monitoring is also likely to be affected by child behavior; children who do not engage in externalizing behaviors, for example, share more information with their parents (Stattin & Kerr, 2000), suggesting that intervention efforts should also be directed toward addressing externalizing childhood behavior. However, only longitudinal research that measures these variables across time, along with intervention studies that seek to directly modify them, can elucidate the mechanisms that underlie the relationships between parenting style and children's immune health.

The findings from this study show that there is a specific aspect of parenting behavior and style, namely poor monitoring and supervision, that is associated with higher levels of inflammation and immune activation in children. Future research should make use of larger samples and complementary, more objective measures of parenting behavior in an attempt to replicate these findings and further explore the influence of more overt negative parenting behaviors (e.g., corporal punishment) on child immune functioning. Researchers should also focus on improving immunological methodology and examining the relationships between parenting behavior, child behavior (especially externalizing behavior), and physical health in children prospectively. Such efforts have the potential to inform prevention and early intervention approaches by identifying modifiable psychosocial risk factors, such as parental monitoring and supervision that could have lifelong implications for the physical and mental health of children.

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