

Parental Accuracy Regarding Adolescent Daily Experiences: Relationships With Adolescent Psychological Adjustment and Inflammatory Regulation

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Objective: There is evidence that parents play an important role in their adolescent's health and well-being, but the links between specific daily processes and biological mechanisms relevant to health remain to be determined. In this study, we examined the role of parental accuracy—that is, whether parents who are more accurate about their adolescents' daily experiences have adolescents with better psychological functioning and inflammatory regulation. **Methods:** In a 2-week daily diary study of 116 parent-adolescent dyads, we examined whether parental accuracy about their adolescent's daily demands and the positivity of their day together were associated with markers of psychological functioning and with regulation of the inflammatory response in terms of glucocorticoid sensitivity (the extent to which cortisol is able to dampen the production of inflammatory proteins) in adolescents. **Results:** Adolescents whose daily experiences were perceived more accurately by their parents reported better psychological adjustment (lower stress and depression) and a greater sensitivity of their immune cells to anti-inflammatory signals from cortisol (i.e., diminished production of inflammatory proteins when cells were stimulated with the combination of a bacterial product [lipopolysaccharide] and cortisol; $|\beta|$ range, 0.38–0.53, all p values $<.041$). **Conclusions:** Greater parental accuracy regarding adolescents' daily experiences is associated with better adolescent psychological adjustment and a more sensitive anti-inflammatory response to cortisol. These results provide preliminary evidence that parental accuracy regarding their adolescent's daily experiences may be one specific daily parent factor that plays a role in adolescent health and well-being. **Key words:** parental accuracy, adolescent well-being, inflammatory processes, glucocorticoid sensitivity.

LPS = lipopolysaccharide; IL = interleukin; CRP = C-reactive protein; TNF- α = tumor necrosis factor α .

INTRODUCTION

The family environment has robust associations with adolescent psychological and physical health (e.g., Ref. (1)). There is evidence that an adverse family environment and parental distress predict the development and progression of mental health difficulties and chronic health conditions in adolescence and later in life (1–5). However, research that seeks to understand the more specific, day-to-day parenting factors that may play a role in adolescent health and well-being has been less common. The current study focuses on one parent factor—parental accuracy about adolescents' daily life experiences—and examines associations with both adolescent psychological adjustment and inflammatory regulation.

Most previous research has focused on links between broad family or parental factors and childhood health and well-being. For example, both hostile and neglectful family environments are associated with greater child psychopathology, such as anxiety and depression (e.g., Refs. (6–11)). There is also evidence that hostile family environments are associated with a greater number of adolescent-reported physical health complaints (12); a variety of potentially health-relevant biological indicators, including indicators of endocrine and metabolic functioning (13,14); and inflammatory dysregulation in adolescents (15–17). Furthermore, parental stress and depression have been linked to

the development and expression of chronic conditions, such as asthma, in children (18–21) and to inflammatory processes likely to be involved in the worsening of those conditions in adolescents (22), young children (23), and newborns (24). Risky family environments in early life have also been linked to elevated C-reactive protein (CRP), a marker of chronic, low-grade inflammation, in young adults (25).

Overall, there is a good deal of evidence linking broad family and parent factors to biological processes relevant to adolescent health, but it is also important to examine the role of more microlevel parent-adolescent factors to better understand what specific factors of an adolescent's daily life may contribute to psychological and physiological functioning. Previous research has found that negative parenting behaviors in specific interactions, such as coldness, are associated with worse parent-reported child health (26), whereas positive parenting behaviors are associated with fewer adolescent-reported health complaints (27) and better metabolic control over diabetes in adolescents (e.g., Ref. (28)). Furthermore, greater daily interpersonal stress, including parent-adolescent conflict, is associated with higher levels of CRP in adolescents (16), and greater daily family conflict at home is associated with less favorable cortisol profiles in children (29).

One daily parent-adolescent factor that has yet to be examined is parental accuracy about adolescent daily experiences, such as the kinds of demands their adolescent is experiencing and how positive their day was. Experiencing stress and having bad days are a normative part of adolescent experiences that have implications for adolescent health (16,30). However, having these daily experiences, good and bad, be accurately perceived by one's parent may have additional implications for adolescent health and well-being. Indeed, interpersonal accuracy is emerging as an important predictor of psychological and social well-being in other relationship contexts (e.g., Refs. (31,32)), and therefore, we sought to undertake a novel extension of this research to examine the implications of accuracy for health-relevant outcomes. We chose to focus on parent-adolescent accuracy, given the importance of family relationships in the lives of adolescents (1) and preliminary evidence that parental accuracy is relevant

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to adolescent psychological and relationship well-being (33), as will be discussed further below.

Specifically, parental accuracy about their adolescent's daily experiences may benefit the adolescent, perhaps representing a more concrete manifestation of several positive relationship processes, including better communication, the provision and perception of social support, and closeness with the parent. Indeed, being perceived in line with one's self-perceptions is associated with closer relationships (e.g., Ref. (34)), and perceptions of greater parental support are associated with higher self-esteem in adolescents (35), fewer adolescent-reported physical health complaints (e.g., Ref. (27)), and better inflammatory regulation in children with asthma (36). Thus, parental accuracy may be a specific parent-adolescent process that plays a direct role in adolescent psychological adjustment and inflammatory regulation.

There is evidence that being accurately perceived by others is associated with better psychological well-being. For example, accurate perceptions of stable traits within new and close relationships are associated with better relationship and individual psychological adjustment (e.g., Refs. (31,32,37,38)). Most of the close relationships literature has examined romantic relationships, but greater parental accuracy about their adolescent's self-views is linked to greater adolescent psychological adjustment and parent-adolescent relationship satisfaction (33). Furthermore, greater parental knowledge of adolescents' daily activities (often termed *parental monitoring*) is associated with better adolescent adjustment (39) and engaging in fewer risky health behaviors (40). The current study will extend previous research by examining if parental accuracy regarding their adolescent's daily experiences is associated with health-relevant immunological processes in adolescents.

In this study, we focus on immune processes because of the important role they are hypothesized to play in linking harsh family environments to physical health (4,41). Interpersonal stress, including with family members, is associated with elevated CRP in adolescents (16) and also with a more pronounced inflammatory response to microbial challenge in adolescents (42). Healthy adolescents reared in harsh family climates also have lower glucocorticoid sensitivity (less ability of cortisol to regulate and turn off inflammatory responses) (17), as do children with asthma who perceive low support from their parents (36).

Harsh family environments may promote chronic, low-grade inflammation through a variety of mechanisms. Here we focus on two possibilities—that family context accentuates monocyte cytokine responses to a common microbial stimulus, lipopolysaccharide (LPS), and/or that it renders these cells less sensitive to anti-inflammatory signals conveyed through glucocorticoids (43,44). Glucocorticoid resistance may then contribute to chronic inflammation, which has been linked to multiple chronic diseases of aging, including cardiovascular disease, the metabolic syndrome, and autoimmune disorders (45,46). We also assessed circulating levels of inflammatory markers as initial indicators of potential chronic inflammation.

In the present study, we examined whether greater parental accuracy regarding the positivity of the adolescent's day with their parent and their daily demands at home and school was

associated with adolescents' psychological adjustment, and their inflammatory responses and its regulation. We hypothesized that greater parental accuracy about these adolescent daily experiences would be associated with better adolescent psychological adjustment and inflammatory regulation.

METHODS

Participants

A total of 116 parent-adolescent dyads participated in this daily diary study examining family life experiences and cardiovascular risk between January 2010 and March 2012. Families were recruited through public schools, newspaper ads, and community postings. Both adolescents and parents were fluent in English and in good health, defined as being free of acute infections the 2 weeks preceding the study and without a history of chronic medical or psychiatric disorders. In an initial laboratory visit, adolescents completed psychological adjustment measures, and blood was drawn to measure cytokine production and glucocorticoid sensitivity. Adolescents ranged in age from 13 to 16 years and lived with the participating parents, who ranged in age from 35 to 64 years (see the Covariates section for further demographic information). After a research assistant verbally explained the study procedures, informed consent was obtained from the parent and adolescents signed an assent form. This study was approved by the behavioral research ethics board at the University of British Columbia.

Measures

Daily Diary Accuracy Measures

Adolescents and parents then completed daily diaries at the end of each day for 14 days. Participants were given the option of completing the daily questionnaires online or on paper copies sent home with them after the study visit. Participants completing paper questionnaires were asked to stamp each diary entry. Our stampers are electronic devices that record the date and time and cannot be reset without a password.

Daily Demands

Adolescents completed a checklist each day (0 = *no*; 1 = *yes*), rating whether they had "a lot of work at home" (mean proportion [$M_{\text{proportion}}$] = 0.33) and "a lot of work at school" ($M_{\text{proportion}}$ = 0.40). These items were selected from the Daily Stress Scale (47). Parents completed the same checklist for the adolescent across the 14 days, indicating whether or not they thought the adolescent had a lot of work at home ($M_{\text{proportion}}$ = 0.31) and school each day ($M_{\text{proportion}}$ = 0.42). Parent and adolescent average ratings were significantly correlated for demands at both home ($r = 0.56, p < .001$) and school ($r = 0.47, p < .001$). In line with much previous work on interpersonal perception (48,49), we assessed parental accuracy about daily demands for each domain by creating a variable that indicated whether the parent's perception matched their adolescent's rating (0 = *no match*; 1 = *match*), and then averaging across all available days for each dyad to obtain an average proportion correct score regarding demands at home ($M = 0.72$; Kuder-Richardson formula, 20:0.84) and school ($M = 0.75$; Kuder-Richardson formula, 20:0.62). Prior to analyses, the proportion correct scores were arcsine transformed to better approximate a normal distribution for each variable.

Positivity of the Day Together

At the end of each day, adolescents rated the positivity of their day with their parent on a scale ranging from 0 (*negative*) to 2 (*positive*; M [standard deviation {SD}] = 1.45 [0.39]). The parent also provided a rating of the positivity of their day with their adolescent on the same rating scale (M [SD] = 1.72 [0.28]), which was significantly correlated with adolescent ratings ($r = 0.39, p < .001$). Because a proportion correct score may not fully capture the degree to which a parent was inaccurate on these items, we assessed parental accuracy on these items by subtracting the parent's rating from the adolescent's rating, and then averaging across all available ratings for each day (M [SD] = -0.24 [0.38]; range, -1.5 to 1.14 ; $\alpha = .86$). Given indications that the overall amount of disagreement, rather than the direction, was relevant to adolescent functioning,¹

¹Specifically, there was evidence of curvilinear relationships between raw difference scores and adolescent outcomes. Similar patterns were found when direction was taken into account for accuracy regarding daily demands.

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we took the absolute value of the difference scores to obtain a clearer indicator of overall difference and then reverse scored the variable so that higher scores would reflect greater accuracy ($M [SD] = 1.16 [0.30]$; range, 0–1.5). Here, accuracy reflects the agreement parents had with their adolescent about how positive their day together was.

Adolescent Psychological Adjustment

Adolescents completed the 10-item version of the Center for Epidemiological Studies Depression Scale ($\alpha = .71$) (50) and the widely used four-item version of the Perceived Stress Scale ($\alpha = .66$) (51).

Inflammatory Parameters

Peripheral blood was drawn from adolescents through antecubital venipuncture into lithium-heparin Vacutainers (Becton-Dickinson, Oakville, ON, Canada) to assess three aspects of inflammation: circulating inflammation, inflammatory responses to bacterial stimulus, and sensitivity to anti-inflammatory signals from cortisol. First, the extent of systemic inflammatory activity was quantified via serum levels of CRP and interleukin (IL)-6. Blood was drawn into Serum-Separator Tubes (Becton-Dickinson). Samples were left to clot for 60 minutes at room temperature and then centrifuged at $1200 \times g$ for 10 minutes. The serum was then aspirated, divided into 1 ml aliquots, and frozen at -30°C until assayed. CRP was measured in the Clinical Chemistry Laboratory at St Paul's Hospital using a high-sensitivity, chemiluminescent technique on an IMMULITE 2000 (Diagnostic Products Corporation, Los Angeles, CA), which has a detection threshold of 20 mg/l and interassay variability of 2.2%. Levels of IL-6 were measured in duplicate with commercially available enzyme-linked immunosorbent assay (ELISA) kits (HS600B; R&D Systems, Minneapolis, MN), which have a minimum detection threshold of 0.039 pg/ml and interassay and intra-assay variability of less than 10%.

Second, whole blood was diluted in a 10:1 ratio and cultured with a bacterial stimulus, LPS, to assess capacity to respond to microbial challenge. Production of proinflammatory cytokines, including IL-1 β , IL-6, IL-8, and tumor necrosis factor α (TNF- α) was assessed. Whole blood was drawn into lithium-heparin Vacutainers (Becton-Dickinson), diluted in a 10:1 ratio with saline, and incubated with LPS (50 ng/ml; Sigma, St Louis, MO) for 6 hours at 37°C in 5% carbon dioxide. The supernatants were collected and frozen at -30°C until analysis. All four proteins (IL-1 β , IL-6, IL-8, TNF- α) were measured in duplicate with MSD Meso Scale Discovery Human ProInflammatory 7-Plex Base Kits (MSD, Rockville, MD) on an MSD SECTOR Imager 2400. These kits have a minimum detection threshold of 0.15 pg/ml and an average variability across samples and cytokines of 6%. Previous studies comparing multiplex assays with single ELISA technology document correlation coefficients of above 0.9 (52). Meso Scale Discovery platforms have greater sensitivity as a multiplex technology (53), and MSD assays gave a broader dynamic quantitative range than Pierce Endogen multiplex assays (Pierce Biotechnology, Woburn, MA) and standard ELISA (R&D Systems) (54).

Third, glucocorticoid resistance was measured by quantifying IL-1 β , IL-6, IL-8, and TNF- α production in cells that had been coincubated with LPS and cortisol. Whole blood was diluted in a 10:1 ratio with saline and dispensed into culture plates (Sigma Chemicals) with LPS (50 ng/ml). A dose of hydrocortisone was added to the wells (2.76×10^{-5} M). After 6 hours of incubation at 37°C in 5% carbon dioxide, the supernatants were collected and frozen at -30°C until analysis. All four proteins (IL-1 β , IL-6, IL-8, TNF- α) were measured in duplicate with the same MSD platform as above. Because of positive skew in the distribution of the proinflammatory cytokine variables, values were log transformed before analyses.

Covariates

Demographic and Biobehavioral Variables

We controlled for several demographic and biobehavioral variables (55), including adolescent age ($M [SD] = 14.57 [1.08]$ years), ethnicity (coded as dummy variables reflecting European [50.86%] or Asian descent [34.48%]), waist circumference ($M [SD] = 74.95 [8.94]$), and parent education level, indexed by highest educational degree achieved by the mother or father, ranging from 1 (some high school) to 5 (graduate school education; $M [SD] = 4.14 [0.84]$). Parents were predominately female (81%) with an average (SD) age of 46.45 (5.00) years.

Broader Psychosocial Variables

We also controlled for several broader family and parent factors to examine whether parental accuracy uniquely predicts adolescent functioning. Specifically, we controlled for adolescent-reported risky family environment (56), which includes 13 items rated on a 1 (not at all) to 5 (very often) scale ($\alpha = .83$); adolescent-reported parental nurturance ($\alpha = .88$) (8,57), rated on nine items using a 1 (never) to 4 (always) scale; and parent-reported parental depression and perceived stress on the 10-item Center for Epidemiological Studies Depression ($\alpha = .80$) and 4-item Perceived Stress Scale ($\alpha = .79$), respectively.

Analytic Approach

We examined whether parental accuracy was associated with adolescent psychological adjustment and inflammatory processes with a series of regression analyses. For example, adolescent psychosocial outcomes, such as depression, were regressed on parental accuracy regarding demands at home and each of the demographic and biobehavioral covariates described above. Analyses were repeated for each parental accuracy indicator. Parallel analyses were conducted for adolescent inflammatory outcomes. Finally, parallel analyses for all adolescent outcomes were run with the additional psychosocial control variables included.

RESULTS

Descriptive Statistics

Descriptive statistics for the psychosocial variables and their bivariate correlations with adolescent inflammatory processes are presented in Table 1. Note that the sample of 116 parent-adolescent dyads only includes dyads for which at least 3 (of 14) daily diary reports were available, for at least one of the three accuracy measures.² On average across accuracy indicators, compliance was high: responses were available for 82.59% of adolescents' ratings and for 86.51% of parents' ratings on average across days, resulting in available data to compute accuracy scores for 77.00% of dyads on average across days.

More positive days together and fewer demands at home across the 2 weeks were correlated with significantly higher levels of parental accuracy ($|r|$ values [range], 0.17–0.69; all p values $<.069$). Full details on the associations between mean daily experiences and parental accuracy and adolescent outcomes adjustment are provided in the Supplemental Digital Content, <http://links.lww.com/PSYMED/A161>. Parental accuracy about the positivity of the adolescent's day with the parent and the adolescent's demands at home was significantly correlated ($r = 0.27, p = .017$), but neither indicator was significantly associated with parental accuracy about adolescents' demands at school (r range, -0.03 to 0.18 ; all p values $>.12$).

Psychological Adjustment

Parental accuracy regarding daily demands at home and school was not significantly associated with adolescent depression or perceived stress ($|b|$ values [range], 0.05–0.23; all

²A total of 10 dyads were excluded for completing less than three daily diary entries on all accuracy measures. Furthermore, low or lack of compliance on the accuracy-specific measures resulted in 113 dyads for the positivity of the day accuracy indicator, 84 dyads for the demands at home indicator, and 88 dyads for the demands at school indicator. Results were consistent, though not as strong, using all available data (i.e., accuracy scores based on 1 or 2 diary entries). Results were consistent using multiple imputation to handle missing and excluded data.

TABLE 1. Descriptive Statistics for Psychosocial Variables and their Correlation with Inflammatory Measures

Psychosocial Variable	Descriptive Statistics		IL-1 β		IL-6		IL-8		TNF- α	
	Mean	SD	LPS	Cort	LPS	Cort	LPS	Cort	LPS	Cort
Positivity of day together accuracy	1.16	0.30	-0.09	-0.16 [†]	-0.15	-0.17 [†]	-0.01	-0.13	-0.15	-0.14 [†]
Demands at home accuracy	0.72	0.27	0.03	-0.22*	0.002	-0.25*	0.10	-0.17 [†]	0.001	-0.22*
Demands at school accuracy	0.75	0.29	-0.13	-0.04	-0.21*	-0.10	-0.11	-0.02	-0.15	-0.04
Adolescent stress	5.07	2.68	-0.05	-0.05	-0.06	-0.11	-0.01	-0.06	-0.07	-0.10
Adolescent depression	7.74	4.40	-0.10	0.05	0.07	0.04	0.07	0.07	-0.09	-0.02
Risky family environment	22.60	6.97	0.10	0.15 [†]	0.04	0.14	-0.05	0.23*	-0.04	0.07
Parent stress	4.92	2.85	0.14	0.20*	0.08	0.13	0.04	0.11	0.07	0.13
Parent depression	6.31	4.59	0.06	-0.02	-0.02	-0.12	0.03	0.09	0.004	-0.08
Parental nurturance	27.41	5.36	0.05	-0.01	-0.08	-0.14	-0.14	-0.15	0.01	-0.06

IL = interleukin; TNF- α = tumor necrosis factor α ; SD = standard deviation; LPS = cytokine production after incubation with lipopolysaccharide; Cort = cytokine production after incubation with cortisol and LPS.

Depression was measured on a 0–3 scale, perceived stress was measured on a 0–4 scale, risky family environment was measured on a 1–5 scale, and parental nurturance was measured on a 1–4 scale; scores on all scales were summed.

* $p < .05$.

[†] $p < .10$.

p values $>.28$). However, adolescents whose parents more accurately perceived the positivity of their day together reported lower depression and perceived stress (β values [range], -0.38 to -0.46 ; all p values $<.036$; see Table 2).

Circulating Inflammation Markers

Adolescents whose parents more accurately perceived their demands at school had significantly greater levels of circulating IL-6 ($\beta = 0.53$, $t(80) = 2.33$, $p = .022$), but parental accuracy regarding positivity of the day and demands at home was not significantly associated with IL-6 levels ($|\beta|$ values [range], 0.01 – 0.04 ; all p values $>.84$). Parental accuracy was not significantly associated with circulating CRP levels ($|\beta|$ values [range], 0.07 – 0.12 ; all p values $>.39$).

LPS-Stimulated Immune Cell Protein Production

Adolescents whose parents more accurately perceived the positivity of their day together and their demands at school produced marginally lower levels of IL-6 in response to LPS (β values [range], -0.33 to -0.44 ; all p values $<.091$; see Table 2).

Glucocorticoid Sensitivity

Adolescents whose demands at home were perceived more accurately by their parents produced significantly lower levels of IL-1 β , IL-6, and TNF- α (β values [range], -0.47 to -0.53 ; all p values $<.041$) when their whole blood was incubated with both LPS and cortisol (see Table 2 and Fig. 1A). Adolescents whose parents more accurately perceived the positivity of their day together were also more sensitive to cortisol inhibition, as evidenced by significantly lower production of IL-1 β and IL-6 and marginally lower production of IL-8 and TNF- α (β values [range], -0.32 to -0.43 ; all p values $<.092$; see Table 2 and Fig. 1B). However, parental accuracy regarding their adolescent's demands at school was not significantly associated with cortisol inhibition (all p values $>.67$).

In line with the moderate correlation between parental accuracy about the adolescent's demands at home and the positivity of their day together, when both accuracy indicators were included within the regression model simultaneously, associations generally became nonsignificant, suggesting that neither accuracy indicator necessarily had a stronger relationship with inflammatory regulation. Furthermore, all effects held controlling

TABLE 2. Associations Between Parental Accuracy and Adolescent Psychological Adjustment and Cytokine Production

Accuracy Category	Depression	Perceived Stress	IL-1 β		IL-6		IL-8		TNF- α	
			LPS	Cort	LPS	Cort	LPS	Cort	LPS	Cort
Positivity of day	-0.46*	-0.38*	-0.19	-0.38*	-0.33 [†]	-0.43*	-0.03	-0.32 [†]	-0.30	-0.35 [†]
Demands at home	0.06	-0.06	0.07	-0.47*	0.05	-0.53*	0.26	-0.33	0.03	-0.48*
Demands at school	0.05	-0.23	-0.21	-0.02	-0.40 [†]	-0.10	-0.11	0.05	-0.26	-0.02

IL = interleukin; LPS = cytokine production after incubation with lipopolysaccharide; Cort = cytokine production after incubation with cortisol and LPS.

Values reported are standardized regression coefficients, β , reflecting the change in psychological adjustment or cytokine production in standard deviations for a 2-standard-deviation increase in the respective accuracy indicator (58). All analyses included covariates.

* $p < .05$.

[†] $p < .10$.

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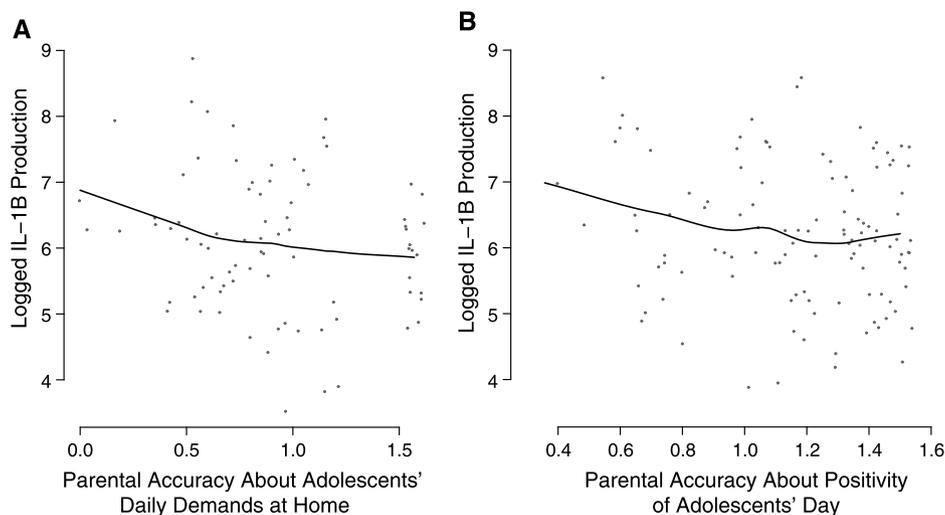


Figure 1. Parental accuracy about the daily demands at home (A) and positivity of the adolescents' day (B) predicting adolescents' logged interleukin- β levels (after incubation with both LPS and cortisol). LPS = lipopolysaccharide.

for adolescent perceived stress and depression, indicating that the associations between parental accuracy and adolescent inflammatory regulation cannot be explained by worse adolescent psychological adjustment. In sum, adolescents who were perceived more accurately by their parents regarding their demands at home and the positivity of their day with their parent were more sensitive to the anti-inflammatory effects of cortisol.

Broader Psychosocial Variables

To determine whether parental accuracy plays a unique role in adolescent inflammation, above and beyond broader family and parent characteristics, we controlled for a variety of additional psychosocial variables. Only risky family environment and parent perceived stress showed significant bivariate associations with adolescent glucocorticoid sensitivity (see Table 1). Furthermore, only two variables showed associations with parental accuracy: parental nurturance predicted significantly higher and risky family environment predicted marginally lower parental accuracy regarding the positivity of the adolescent's day with their parent ($|\beta|$ values [range], 0.36 to 0.71; all p values $<.069$). Nevertheless, parental accuracy regarding the positivity of the adolescent's day with their parent remained a significant or marginal predictor of adolescent depression and perceived stress when controlling for broader family factors (β values [range], -0.30 to -0.31 ; all p values $<.13$). Furthermore, the significant relationships between both parental accuracy indicators and glucocorticoid sensitivity generally remained intact when controlling for these broader factors (β values [range], -0.32 to -0.39 ; all p values $<.094$).

DISCUSSION

The family environment plays a role in adolescent health (e.g., Ref. (1,4)), but less is known about the more specific, daily parenting factors that relate to adolescent health and well-being. This study suggests that parental accuracy about adolescent daily experiences may be one more specific day-to-day

parenting factor that plays a unique role in adolescent psychological adjustment and inflammatory regulation. Specifically, adolescents whose parents more accurately perceived how positive their day was with their parent reported better psychological adjustment. Furthermore, adolescents whose parents more accurately perceived the positivity of their day together and their demands at home exhibited greater glucocorticoid sensitivity—their immune cells were more sensitive to anti-inflammatory signals from cortisol. Parental accuracy about their adolescent's demands at school was generally not associated with psychological adjustment or inflammatory processes—it is unclear whether this is a result of the lower reliability of this accuracy indicator or because parental accuracy in familial domains is more relevant to adolescent functioning. Overall, these findings provide novel evidence that how well parents understand their adolescents' day-to-day experiences may play a unique role in both adolescent psychological functioning and glucocorticoid sensitivity.

The finding that greater parental accuracy about the positivity of the adolescent's day with the parent was associated with lower adolescent stress and depression is in line with previous findings that being perceived accurately is associated with better psychological adjustment in other social contexts (31,37). Previous research has found evidence for this link in parent-adolescent relationships (33), but the current results extend these findings by demonstrating that these links emerge for parental accuracy regarding adolescents' daily experiences and for a different set of adolescent adjustment measures related to depression and stress. This pattern of results suggests that the association between being perceived accurately and psychological adjustment is quite robust across relationship types and accuracy indicators. Note that supplemental analyses revealed that this association may be partly driven by the tendency for adolescents who were seen less accurately to report less positive days with their parents (see Supplemental Digital Content, <http://links.lww.com/PSYMED/A161>). Future research is needed to establish whether these effects are robust within this context. More broadly, it will be important to examine at what

level inaccuracy may become particularly problematic for individuals and whether being chronically perceived inaccurately may have clinical significance.

The current findings are also the first that we are aware of to link the accuracy of others' perceptions about one's daily life to immunological processes potentially relevant to health. Specifically, greater parental accuracy regarding the positivity of the adolescent's day with the parent and demands at home was related to more sensitive responses to the anti-inflammatory effects of cortisol in adolescents. This is consistent with previous work linking broader family and parental characteristics to individual inflammatory processes (17,22,25,36,42), but sheds light on the more specific daily parent-adolescent processes that may play an independent role. These results are also in line with findings that day-to-day parent-adolescent conflict is associated with adolescent systemic inflammation (16), but indicate that parental accuracy about such daily experiences may have additional implications for adolescent inflammatory processes. Importantly, parental accuracy was generally a stronger predictor of inflammatory regulation than main effects of daily experiences (e.g., accumulated daily demands and mean positivity ratings) when examined separately, and the effects of accuracy generally held when controlling for main effects, especially for accuracy regarding demands at home (see Supplemental Digital Content, <http://links.lww.com/PSYMED/A161>). Future work is needed to examine whether the associations between accuracy and inflammatory processes extend to other domains and relationship contexts.

Note that the associations between parental accuracy and adolescent inflammation were stronger for glucocorticoid sensitivity assays (incubation with LPS + cortisol) than for incubation with LPS alone. This suggests that parental accuracy is more strongly linked to the regulation of inflammatory processes (through cortisol) than to inflammatory responsiveness to challenges. It is possible that different types of psychological experiences shape different aspects of immune responses. For example, positive relationship factors may play an important role in mitigating chronic exposures to stress, which can shape the development of glucocorticoid resistance. In contrast, other psychological factors, such as appraisals of threat, may prime vigilant states that affect how the immune system responds to challenges. This would be consistent with previous findings that parent supportiveness is associated with better inflammatory regulation in children with asthma (36) and with findings that threat perceptions are associated with inflammatory responses to mitogen challenge in children with asthma (59,60).

Parental accuracy was also not consistently linked to indicators of adolescent chronic inflammation, such as circulating CRP and IL-6 levels. It is unclear whether these generally null associations are a result of examining a young and healthy sample, with 90% of these adolescents in the low-risk range (CRP ≤ 1 mg/l), or whether parental accuracy does not relate to adolescent chronic inflammation more broadly.

One limitation of the current study is that it is cross sectional, making causal hypotheses speculative. It is possible that adolescent stress and depression could affect parental accuracy,

rather than the reverse, and that other third variables exist that affect both parental accuracy and adolescent functioning. Furthermore, the antecedents of accuracy, such as broader parent, family, and relationship characteristics, need to be further examined, to better understand process and whether accuracy plays a unique or mediating role. In the current study, parental accuracy was associated with adolescent psychological adjustment and inflammatory regulation quite independently of both risky family and parent factors, indicating that parental accuracy plays a unique role in adolescent health that deserves further examination. It will also be critical to examine the mediating links between parental accuracy and adolescent functioning. We did not find evidence that adolescent depression and stress play a mediating role, but more in depth, preferably longitudinal, examinations of potential mechanisms, such as parent supportiveness and the quality of interactions, are needed.

Another important future direction will be to examine whether being perceived accurately is associated with other types of health-relevant biological processes that have been linked to risky family environments, both in adolescence and/or in adulthood, such as unfavorable cortisol profiles (29,61,62), indicators of cardiovascular risk such as heightened blood pressure (13,25,63), and worse metabolic functioning (64). Furthermore, to begin disentangling the causal relationships among these variables, future research will need to use prospective, longitudinal designs to examine whether parental accuracy in adolescence predicts glucocorticoid sensitivity and other health-relevant processes several months or years later. Finally, it will be critical for future research to follow up samples with clinical health outcomes, to examine the implications of accuracy for longer-term health measures. For instance, one could examine whether being perceived inaccurately in adolescence is associated with the development of both more intermediate-risk factors for chronic illnesses, such as systemic inflammation, as well as with the development of chronic diseases, such as cardiovascular disease and diabetes, later in life.

Another important goal for future research will be to determine the nature and sources of parental accuracy. Parental accuracy was associated with generally more positive days together and fewer accumulated demands at home (see Supplemental Digital Content, <http://links.lww.com/PSYMED/A161>), as well as several broader family environment factors, including greater parental nurturance and a less risky family environment. Thus, parental accuracy is related to generally more positive daily lives for adolescents and more supportive, positive family environments. Within such positive environments, there are several potential sources of parental accuracy: parent perceptivity, adolescent expressivity, or unique relationship characteristics. Greater parental knowledge about their adolescents' whereabouts seems to be driven more by adolescent disclosure than parental vigilance (e.g., Ref. (39)), there tends to be less discussion of feelings in more distressed families (65), and children and young adults from such families tend to be less accurate at expressing their emotions (66,67). Thus, adolescent expressivity may be the primary source of parental accuracy, but this remains to be directly examined. It would also be interesting

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to examine the extent to which parental accuracy is driven by explicit, verbal disclosure (such as by discussing one's day together), or less direct nonverbal cues.

There are several limitations of the current study design that could be improved upon in future research. For example, although daily ratings help to deal with recall and other reporting biases that may be found in broader questionnaires, such ratings still rely on memory and aggregation to some degree—future research could use time or experience sampling techniques to capture accuracy directly in the moment. There are also limitations to the proportion correct and difference score accuracy indicators used in this study. For example, the proportion correct accuracy indicator can be influenced by base rates (68) and may be a less sensitive measure of accuracy than difference score and correlational approaches. Although a simple and often used indicator of discrepancy, difference scores also have important limitations that can influence their interpretability (e.g., Refs. (69,70)). Such limitations would be best overcome by assessing parental accuracy with different rating scales and on a larger set of items, enabling the use of more sophisticated approaches to assessing accuracy and agreement (e.g., Refs. (69,71)). Having demonstrated that these associations emerge across two rather simple and distinct accuracy indicators will hopefully justify more extensive measurement in future research. Finally, future work should also obtain accuracy criteria beyond adolescents' self-reports. Although self-other agreement is a common and generally valid accuracy indicator (72), it would be beneficial to use additional accuracy criteria, such as behavioral measures and observer reports in future studies.

In sum, the current study provides preliminary evidence that parental accuracy about adolescent daily experiences is one more specific parent factor that is associated with better adolescent psychological adjustment and inflammatory regulation, which may have downstream health implications for adolescents. Although questions remain about causality and generalizability, these findings begin to shed light on day-to-day parent-adolescent relationship processes that may affect adolescent psychological and physical health.

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REFERENCES

1. Repetti RL, Taylor SE, Seeman TE. Risky families: family social environments and the mental and physical health of offspring. *Psychol Bull* 2002;128:330–66.
2. Heim C, Nemeroff CB. The role of childhood trauma in the neurobiology of mood and anxiety disorders: preclinical and clinical studies. *Biol Psychiatry* 2001;49:1023–39.
3. Matthews KA. Psychological perspectives on the development of coronary heart disease. *Am Psychol* 2005;60:783–96.
4. Miller GE, Chen E, Parker KJ. Psychological stress in childhood and susceptibility to the chronic diseases of aging: moving toward a model of behavioral and biological mechanisms. *Psychol Bull* 2011;137:959–97.
5. Shonkoff JP, Boyce WT, McEwen BS. Neuroscience, molecular biology, and the childhood roots of health disparities: building a new framework for health promotion and disease prevention. *JAMA* 2009;301:2252–9.
6. Chorpita BF, Barlow DH. The development of anxiety: the role of control in the early environment. *Psychol Bull* 1998;124:3.
7. Cicchetti D, Toth SL. Child maltreatment. *Annu Rev Clin Psychol* 2005;1:409–38.
8. Conger RD, Conger KJ, Elder GH, Lorenz FO, Simons RL, Whitbeck LB. A family process model of economic hardship and adjustment of early adolescent boys. *Child Dev* 1992;63:526–41.
9. Emery RE, Laumann-Billings L. An overview of the nature, causes, and consequences of abusive family relationships: toward differentiating maltreatment and violence. *Am Psychol* 1998;53:121.
10. Kaslow NJ, Deering CG, Racusin GR. Depressed children and their families. *Clin Psychol Rev* 1994;14:39–59.
11. Lempers JD, Clark-Lempers D, Simons RL. Economic hardship, parenting, and distress in adolescence. *Child Dev* 1989;25–39.
12. Mechanic D, Hansell S. Divorce, family conflict, and adolescents' well-being. *J Health Soc Behav* 1989;30:105–16.
13. Evans GW. A multimethodological analysis of cumulative risk and allostatic load among rural children. *Dev Psychol* 2003;39:924.
14. Weidner G, Hutt J, Connor SL, Mendell NR. Family stress and coronary risk in children. *Psychosom Med* 1992;54:471–9.
15. Danese A, Caspi A, Williams B, Ambler A, Sugden K, Mika J, Werts H, Freeman J, Pariante CM, Moffitt TE. Biological embedding of stress through inflammation processes in childhood. *Mol Psychiatry* 2010;16:244–6.
16. Fuligni AJ, Telzer EH, Bower J, Cole SW, Kiang L, Irwin MR. A preliminary study of daily interpersonal stress and C-reactive protein levels among adolescents from Latin American and European backgrounds. *Psychosom Med* 2009;71:329–33.
17. Miller GE, Chen E. Harsh family climate in early life presages the emergence of a proinflammatory phenotype in adolescence. *Psychol Sci* 2010;21:848–56.
18. Klinnert MD, Nelson HS, Price MR, Adinoff AD, Leung DY, Mrazek DA. Onset and persistence of childhood asthma: predictors from infancy. *Pediatrics* 2001;108:e69.
19. Kozyskyj AL, Mai XM, McGrath P, Hayglass KT, Becker AB, Macneil B. Continued exposure to maternal distress in early life is associated with an increased risk of childhood asthma. *Am J Respir Crit Care Med* 2008;177:142–7.
20. Shalowitz MU, Berry CA, Quinn KA, Wolf RL. The relationship of life stressors and maternal depression to pediatric asthma morbidity in a subspecialty practice. *Ambul Pediatr* 2001;1:185–93.
21. Weil CM, Wade SL, Bauman LJ, Lynn H, Mitchell H, Lavigne J. The relationship between psychosocial factors and asthma morbidity in inner-city children with asthma. *Pediatrics* 1999;104:1274–80.
22. Wolf JM, Miller GE, Chen E. Parent psychological states predict changes in inflammatory markers in children with asthma and healthy children. *Brain Behav Immun* 2008;22:433–41.
23. Wright RJ, Finn P, Contreras JP, Cohen S, Wright RO, Staudenmayer J, Wand M, Perkins D, Weiss ST, Gold DR. Chronic caregiver stress and IgE expression, allergen-induced proliferation, and cytokine profiles in a birth cohort predisposed to atopy. *J Allergy Clin Immunol* 2004;113:1051–7.
24. Wright RJ, Visness CM, Calatroni A, Grayson MH, Gold DR, Sandel MT, Lee-Parritz A, Wood RA, Kattan M, Bloomberg GR, Burger M, Toggias A, Witter FR, Sperling RS, Sadosky Y, Gern JE. Prenatal maternal stress and cord blood innate and adaptive cytokine responses in an inner-city cohort. *Am J Respir Crit Care Med* 2010;182:25–33.
25. Taylor SE, Lehman BJ, Kiefe CI, Seeman TE. Relationship of early life stress and psychological functioning to adult C-reactive protein in the coronary artery risk development in young adults study. *Biol Psychiatry* 2006;60:819–24.
26. Gottman JM, Katz LF. Effects of marital discord on young children's peer interaction and health. *Dev Psychol* 1989;25:373.
27. Wickrama KAS, Lorenz FO, Conger RD. Parental support and adolescent physical health status: a latent growth-curve analysis. *J Health Soc Behav* 1997;149–63.
28. Martin MT, Miller-Johnson S, Kitzmann KM, Emery RE. Parent-child relationships and insulin-dependent diabetes mellitus: observational ratings of clinically relevant dimensions. *J Fam Psychol* 1998;12:102.

29. Slatcher RB, Robles TF. Preschoolers' everyday conflict at home and diurnal cortisol patterns. *Health Psychol* 2012;31:834–8.
30. Nishina A, Juvonen J, Witkow MR. Sticks and stones may break my bones, but names will make me feel sick: the psychosocial, somatic, and scholastic consequences of peer harassment. *J Clin Child Adolesc Psychol* 2005;34:37–48.
31. Human LJ, Biesanz JC. Target adjustment and self-other agreement: utilizing trait observability to disentangle judgeability and self-knowledge. *J Pers Soc Psychol* 2011;101:202–16.
32. Neff LA, Karney BR. To know you is to love you: the implications of global adoration and specific accuracy for marital relationships. *J Pers Soc Psychol* 2005;88:480–97.
33. Sillars A, Koerner A, Fitzpatrick MA. Communication and understanding in parent-adolescent relationships. *Hum Commun Res* 2005;31:102–28.
34. Swann WB Jr, De La Ronde C, Hixon JG. Authenticity and positivity strivings in marriage and courtship. *J Pers Soc Psychol* 1994;66:857.
35. Whitbeck LB, Simons RL, Conger RD, Lorenz FO, Huck S, Elder GH Jr. Family economic hardship, parental support, and adolescent self-esteem. *Soc Psychol Q* 1991;353–63.
36. Miller GE, Gaudin A, Zysk E, Chen E. Parental support and cytokine activity in childhood asthma: the role of glucocorticoid sensitivity. *J Allergy Clin Immunol* 2009;123:824–30.
37. Colvin CR. Judgable people: personality, behavior, and competing explanations. *J Pers Soc Psychol* 1993;64:861.
38. Human LJ, Sandstrom GM, Biesanz JC, Dunn EW. Accurate first impressions leave a lasting impression the long-term effects of distinctive self-other agreement on relationship development. *Soc Psychol Pers Sci* 2013;4:395–402.
39. Kerr M, Stattin H. What parents know, how they know it, and several forms of adolescent adjustment: Further support for a reinterpretation of monitoring. *Dev Psychol* 2000;36:366.
40. DiClemente RJ, Wingood GM, Crosby R, Sionean C, Cobb BK, Harrington K, Davies S, Hook EW, Oh MK. Parental monitoring: association with adolescents' risk behaviors. *Pediatrics* 2001;107:1363–8.
41. Taylor SE. Mechanisms linking early life stress to adult health outcomes. *Proc Natl Acad Sci U S A* 2010;107:8507–12.
42. Miller GE, Rohleder N, Cole SW. Chronic interpersonal stress predicts activation of pro- and anti-inflammatory signaling pathways 6 months later. *Psychosom Med* 2009;71:57–62.
43. Marques AH, Silverman MN, Sternberg EM. Glucocorticoid dysregulations and their clinical correlates. From receptors to therapeutics. *Ann NY Acad Sci* 2009;1179:1–18.
44. Raison CL, Miller AH. When not enough is too much: the role of insufficient glucocorticoid signaling in the pathophysiology of stress-related disorders. *Am J Psychiatry* 2003;160:1554–65.
45. Chung HY, Cesari M, Anton S, Marzetti E, Giovannini S, Seo AY, Carter C, Yu BP, Leeuwenburgh C. Molecular inflammation: underpinnings of aging and age-related diseases. *Ageing Res Rev* 2009;8:18–30.
46. Nathan C, Ding A. Nonresolving inflammation. *Cell* 2010;140:871–82.
47. Bolger N, DeLongis A, Kessler RC, Schilling EA. Effects of daily stress on negative mood. *J Pers Soc Psychol* 1989;57:808.
48. Nowicki S Jr, Duke MP. Individual differences in the nonverbal communication of affect: the diagnostic analysis of nonverbal accuracy scale. *J Nonverbal Behav* 1994;18:9–35.
49. Rosenthal R, Hall JA, DiMatteo MR, Rogers PL, Archer D. Sensitivity to Nonverbal Communication: The PONS Test. Baltimore: Johns Hopkins University Press; 1979.
50. Radloff LS. The CES-D scale: a self-report depression scale for research in the general population. *Appl Psychol Meas* 1977;1:385–401.
51. Cohen S, Kamarck T, Mermelstein R. A global measure of perceived stress. *J Health Soc Behav* 1983;385–96.
52. Urbanowska T, Mangialaio S, Zickler C, Cheevaprak S, Hasler P, Regenass S, Legay F. Protein microarray platform for the multiplex analysis of biomarkers in human sera. *J Immunol Methods* 2006;316:1–7.
53. Chowdhury F, Williams A, Johnson P. Validation and comparison of two multiplex technologies, luminex and mesoscale discovery, for human cytokine profiling. *J Immunol Methods* 2009;340:55–64.
54. Toedter G, Hayden K, Wagner C, Brodmerkel C. Simultaneous detection of eight analytes in human serum by two commercially available platforms for multiplex cytokine analysis. *Clin Vaccine Immunol* 2008;15:42–8.
55. O'Connor MF, Bower JE, Cho HJ, Creswell JD, Dimitrov S, Hamby ME, Hoyt MA, Martin JL, Robles TF, Sloan EK, Thomas KS, Irwin MR. To assess, to control, to exclude: effects of biobehavioral factors on circulating inflammatory markers. *Brain Behav Immun* 2009;23:887–97.
56. Taylor SE, Lerner JS, Sage RM, Lehman BJ, Seeman TE. Early environment, emotions, responses to stress, and health. *J Pers* 2004;72:1365–93.
57. Locke LM, Prinz RJ. Measurement of parental discipline and nurturance. *Clin Psychol Rev* 2002;22:895–929.
58. Gelman A. Scaling regression inputs by dividing by two standard deviations. *Statistics in Medicine* 2008;27:2865–2873.
59. Chen E, Fisher EB, Bacharier LB, Strunk RC. Socioeconomic status, stress, and immune markers in adolescents with asthma. *Psychosom Med* 2003;65:984–92.
60. Chen E, Hanson MD, Paterson LQ, Griffin MJ, Walker HA, Miller GE. Socioeconomic status and inflammatory processes in childhood asthma: the role of psychological stress. *J Allergy Clin Immunol* 2006;117:1014–20.
61. Luecken LJ, Appelhans BM. Early parental loss and salivary cortisol in young adulthood: the moderating role of family environment. *Dev Psychopathol* 2006;18:295.
62. Taylor SE, Karlamangla AS, Friedman EM, Seeman TE. Early environment affects neuroendocrine regulation in adulthood. *Soc Cogn Affect Neurosci* 2011;6:244–51.
63. Loucks EB, Almeida ND, Taylor SE, Matthews KA. Childhood family psychosocial environment and coronary heart disease risk. *Psychosom Med* 2011;73:563–71.
64. Lehman BJ, Taylor SE, Kiefe CI, Seeman TE. Relation of childhood socioeconomic status and family environment to adult metabolic functioning in the CARDIA study. *Psychosom Med* 2005;67:846–54.
65. Dunn J, Brown J. Affect expression in the family, children's understanding of emotions, and their interactions with others. *Merrill-Palmer Q* 1994;40:120–37.
66. Camras LA, Ribordy S, Hill J, Martino S, Spaccarelli S, Stefani R. Recognition and posing of emotional expressions by abused children and their mothers. *Dev Psychol* 1988;24:776.
67. Halberstadt AG. Family socialization of emotional expression and nonverbal communication styles and skills. *J Pers Soc Psychol* 1986;51:827.
68. Uebersax JS. Diversity of decision-making models and the measurement of interrater agreement. *Psychol Bull* 1987;101:140.
69. Laird RD, De Los Reyes A. Testing informant discrepancies as predictors of early adolescent psychopathology: why difference scores cannot tell you what you want to know and how polynomial regression may. *J Abnorm Child Psychol* 2013;41:1–14.
70. Griffin D, Murray S, Gonzalez R. Difference score correlations in relationship research: a conceptual primer. *Pers Relationships* 1999;6:505–18.
71. Biesanz JC. The social accuracy model of interpersonal perception: assessing individual differences in perceptive and expressive accuracy. *Multivariate Behav Res* 2010;45:853–85.
72. Funder DC, Colvin CR. Congruence of Others' and Self-Judgments of Personality. San Diego: Academic Press; 1997.