Family Chaos and Adolescent Inflammatory Profiles: The Moderating Role of Socioeconomic Status

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Objective: To test whether family chaos influences adolescents' inflammatory profiles and whether adolescents from low socioeconomic status (SES) environments are at higher risk for experiencing adverse inflammatory profiles from living in chaotic family environments. **Methods:** A total of 244 families with an adolescent aged 13 to 16 years participated. Parents completed measures of family SES and family chaos. Both systemic inflammation and stimulated proinflammatory cytokine production in response to bacterial challenge were assessed in adolescents. **Results:** Our results suggest that SES moderates the detrimental effect of family chaos on systemic inflammation and interleukin-6 ($B = -0.010$, standard error [SE] = 0.004, $p = .026$), but not C-reactive protein ($B = 0.009$, SE = 0.006, $p = .11$), and on stimulated proinflammatory cytokine production ($B = -0.098$, SE = 0.044, $p = .026$) in adolescents, such that a chaotic family environment is positively associated with greater systemic inflammation and greater stimulated proinflammatory cytokine production in adolescents as family SES declines. **Conclusions:** These findings indicate that living in chaotic family environments places youth who may be vulnerable based on socioeconomic factors at a potentially higher risk for inflammation-related diseases. **Key words:** inflammation, family chaos, adolescents, socioeconomic status.

SES = socioeconomic status; **CRP** = C-reactive protein; **BMI** = body mass index; **IL-1β** = interleukin-1β; **IL-6** = interleukin-6; **IL-8** = interleukin-8; **IL-10** = interleukin-10.

INTRODUCTION

The family environment has been recognized as an important contributor to the physical and emotional well-being of youth. One aspect of the family environment that shapes the lives of adolescents is family chaos. Chaotic families are ones that are marked by greater disorganization, noise, and crowding and by fewer structures in everyday family life (1,2). Dumas and colleagues (3) have shown that family chaos is a concept of interest in its own right, not merely representing undesirable family psychosocial traits (e.g., conflict and poor relationship quality), but rather reflecting order and regularity, or lack thereof, in day-to-day family life. For the purpose of the present study, we refer to family chaos as reflecting various aspects of environmental confusion in daily family life, specifically disorganization/lack of planning, noise, and a general lack of structure. Family chaos seems to be more pronounced among families from lower, rather than higher, socioeconomic status (SES) backgrounds (1,3,4), possibly because of a higher percentage of single-parent families, greater exposure to life stressors, and reduced access to resources that could support the maintenance of everyday routines among these families.

Numerous studies have investigated how a disorganized family environment can negatively affect children and adolescents; however, these studies have focused almost exclusively on outcomes relevant to youth's socioemotional well-being, behavior problems, and academic achievements. This body of research has linked greater family chaos to greater rates of behavior problems (3,5–7), poorer school performance (8), and poorer overall socioemotional development (1,3) among children.

Although convincing evidence linking family chaos to the above outcomes in social domains exists, very few studies have investigated the influence of chaotic family environments on physical health. A small number of studies provide tentative evidence that family chaos is also related to health-relevant outcomes. First, one study found levels of family chaos to partially mediate the relationship between SES and daily salivary cortisol output among a sample of adolescents followed up over the course of 2 years (9). Greater family chaos predicted increasing trajectories of daily cortisol output over the study period and partially explained the relationship between lower SES and greater increases in salivary cortisol output over time. This study suggests that exposure to family chaos may take a physiological toll over time among adolescents.

Second, another study showed that among children and adolescents diagnosed as having asthma, those who lived in homes marked by less stable family routines experienced increases in stimulated interleukin-13 levels over a 2-year period (10). Interleukin-13 is a type of cytokine (Th-2) that has been implicated in the pathophysiology of asthma exacerbations. Together, these studies point to the potential for chaotic family environments to alter physiological systems that have implications for health.

Furthermore, SES may influence the effect that family chaos has on adolescent health-relevant outcomes. Lower SES has itself been linked to increased inflammation among adolescents (11). To our knowledge, no previous research has examined the moderating role of SES on family chaos. Nonetheless, the notion that SES may moderate the effects of psychosocial factors on health has been suggested previously, and below, we review support for this notion more generally. Pulkki et al. (12) followed up children throughout childhood and examined the influence of maternal child rearing on insulin resistance. They found that hostile child-rearing attitudes were associated with an increased risk of insulin resistance, but this was only the case among girls from low, not high, SES families, suggesting that low family SES compounded the negative effect of hostile mothering. This converges with findings from other studies.

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Evans et al. (13) found that among a sample of young adolescents, those who had mothers low in responsiveness experienced higher levels of allostatic load, a physiological marker of chronic wear and tear on the body, but only if they also had experienced multiple psychosocial and physical stressors such as poverty. Similarly, Miller et al. (14) found that adults who grew up with more nurturing mothers were less likely to experience metabolic syndrome later in life, but only if they were from low SES families. Finally, other studies have also found that only low, but not high, SES adolescents benefited from a psychosocial intervention during laboratory stressor tasks, consequently experiencing less physiological reactivity (15) and that among adults, factors such as perceived control and social support were more beneficial among low SES compared with high SES individuals with respect to various health-related outcomes (16,17).

Finally, although only few studies have investigated inflammatory markers among adolescents, evidence suggesting a potentially important role of psychosocial factors on inflammation among adolescents is beginning to accumulate (18). For example, Fuligni et al. (19) showed that adolescents who experienced greater interpersonal stress (assessed using daily diaries over a 14-day period) had higher levels of C-reactive protein (CRP), indicative of systemic inflammation, several months later. In addition, adolescents' ways of coping with stressors may also influence their inflammatory profiles. Among adolescents exposed to significant life stressors, those who engaged in positive engagement coping (i.e., trying to actively change their situation) had lower levels of CRP (20). These studies indicate that stressors in adolescents' social environment and adolescents' ways of coping with the same may represent important influences on adolescents' inflammatory status.

The purpose of the present study was to extend existing research on the effects that growing up in a disorganized home can have on adolescent development by turning from socioemotional and behavioral outcomes to inflammatory outcomes. In this study, we focus on inflammation because of its links to many chronic health conditions. Although acute inflammation represents a necessary defensive response to infection and injury, ongoing or chronic inflammation is increasingly being linked to some of the most common chronic diseases today, including cardiovascular disease, cancer, and diabetes, among others (21–23), as well as allostatic load (24). Indicators of systemic inflammation such as CRP and interleukin-6 (IL-6) are two measures that have been linked to cardiovascular disease risk (25–28). In addition to markers indicating basal levels of inflammation, there may also be value in investigating the functional properties of immune cells, for example, by assessing in vitro expression of inflammatory cytokines after bacterial stimulation to gauge the magnitude of the immune system's responses to challenges (29).

In this study, we focus on adolescents because health disparities are already evident among this age group (30,31), as are individual differences in inflammatory profiles (19,32). Furthermore, the early stages of some disease processes, for example, of atherosclerosis, emerge early and are already noticeable among adolescents (33), making adolescence an important period to study for prevention purposes.

Thus, we focus on a relatively understudied aspect of the family environment, family chaos, as an indicator of structural rather than socioemotional aspects of the family environment, and we also examine a potential moderating factor, SES. Specifically, we hypothesize that living in a chaotic home will be associated with greater inflammatory profiles (higher levels of systemic inflammation and heightened responses of immune cells to bacterial challenge) among adolescents. We further hypothesize that SES will moderate this relationship, such that the effect of family chaos will be stronger among youth who come from lower SES backgrounds.

METHODS

Participants

Participants were 244 adolescents aged 13 to 16 years (14.57 [1.05] years; 49% male) who visited the laboratory together with one of their parents (76% mothers). All participants were recruited through advertisements in local media from the larger Vancouver, BC, area between January 2010 and March 2012, and healthy and fluent in English. Upon contacting our study staff, interested participants were screened over the telephone. Inclusion criteria included the absence of chronic illnesses and fluency in English for both parents and their children, and adolescents were required to be between 13 and 16 years of age. Eligible parent-adolescent dyads interested in participating were subsequently scheduled for late afternoon (after school) visits. If participants (either child or parent) reported an acute illness, they were rescheduled for 4 weeks after the end of symptoms (in addition, complete blood counts were obtained and blood draws rescheduled if there was evidence of elevated white blood cell counts indicative of infectious disease.) Most adolescents were of European descent (49%), the others of Asian (37%) or "other" descent (14%). Participants represented a wide range of socioeconomic backgrounds. See Table 1 for participant characteristics.

Procedure

Adolescents and their parents were scheduled for a late afternoon (after school) appointment, visited the laboratory together, and provided written assent and consent, respectively. Parents completed a computer questionnaire regarding levels of chaos in the family home along with other measures of family relationships, including parenting behaviors. Parent demographic information and total family income were assessed in an interview by a trained research assistant. Adolescents provided demographic information and underwent a peripheral blood draw through antecubital venipuncture, performed by a trained phlebotomist. Participants were reimbursed for their time and effort. This study was approved by the research ethics board of the University of British Columbia.

Measures

Socioeconomic Status

Parents were asked to indicate their family's total gross income over the past 12 months by selecting an income category on a 9-point scale ranging from "less than $5000" to "$200,000 and higher." See Table 1 for the income distribution in this sample. The range of income represented in our sample is typical of Canadian households. On average, families participating in this study were four-person households, the Canadian median income for which was $79,530 in 2011 (www.statcan.gc.ca). For comparison, the median family income for four-person households in the United States was $74,130 in 2011 (www.census.gov). In the present sample, annual family income ranged from less than $5000 to greater than $200,000, hence representing a wide range of incomes. On average, families in our sample fell into the $50,000 to $74,000 income category.

TABLE 1. Sample Descriptives

	Total Sample (n = 244)		Adolescents Subsampled for Blood Culture (n = 143)	
	n (%)	M (SD)	n (%)	M (SD)
Male	119 (49)		68 (45)	
Female	125 (51)		83 (55)	
Age, y		14.57 (1.05)		14.56 (1.06)
Ethnicity				
European	119 (49)		75 (52)	
Asian	91 (37)		48 (34)	
Other	34 (14)		20 (14)	
BMI, kg/m^2		21.32 (3.64)		21.25 (3.64)
Total family income				
<$5000	3 (1)		2 (1.3)	
$5000–$19,999	12 (5)		7 (4.6)	
$20,000–$34,999	19 (8)		14 (9.3)	
$35,000–$49,999	29 (12)		16 (10.6)	
$50,000–$74,999	59 (24)		35 (23.2)	
$75,000–$99,999	34 (14)		17 (11.3)	
$100,000–$149,999	49 (20)		29 (19.2)	
$150,000–$199,999	26 (11)		19 (12.6)	
>$200,000	13 (5)		11 (7.3)	
Family chaos[a]		2.84 (2.66)		2.62 (2.60)
Inflammatory markers (raw values)				
IL-6β, pg/ml		0.96 (1.10)		0.98 (1.17)
CRP, mg/l		1.03 (3.53)		1.13 (4.34)
Stimulated IL-1β, pg/ml				7668.15 (6862.7)
Stimulated IL-6β, pg/ml				34,119.34 (10,560.0)
Stimulated IL-8β, pg/ml				19,366.51 (11,753.2)
Stimulated IL-10β, pg/ml				223.22 (128.5)

BMI = body mass index; IL-6 = interleukin-6; CRP = C-reactive protein; IL-1β = interleukin-1β; IL-8 = interleukin-8; IL-10 = interleukin-10.
[a] This mean is comparable to those found in other studies using the Confusion, Hubbub, and Order Scale; for example, the original validation study reported a mean of 3.3 (34).

Family Chaos

To assess family chaos in our participants' homes, parents were asked to complete the Confusion, Hubbub, and Order Scale (CHAOS (34)). The CHAOS scale consists of 15 items answered either true or false. Sample items include "At home we can talk to each other without being interrupted," "You can't hear yourself think in our home," and "Our home is a good place to relax." A complete list of items can be found in the original article (34). Items are summed to create a total score between 0 and 15, such that higher scores are indicative of more chaotic and disorganized home environments. The CHAOS questionnaire has been shown to be very stable over a 12-month period (r = 0.74) (34) and hence is representative of chronic conditions in the family environment. It has furthermore been shown to be significantly related to more noise, crowding, and greater "home traffic patterns," as assessed by the Purdue Home Stimulation Inventory, in families with toddlers (34). The CHAOS questionnaire has also been successfully used in families with older children and adolescents (6,9,35). Internal consistency in the present sample was acceptable at α = .75.

Parenting

Parents also completed a questionnaire regarding their parenting behaviors, including 7 questions (e.g., "How often do you give reasons to your child for your decisions?") assessing nurturant/involved parenting and 11 questions (e.g., "When your child does something wrong, how often do you lose your temper and yell at him/her?") assessing harsh/inconsistent parenting (36). All items were answered on a 4-point scale ranging from 1 (always) to 4 (never). Responses to the questions in the respective subscales were summed to create a total score for each subscale. Higher scores indicate more nurturant or harsher parenting.

Inflammatory Markers

Adolescents' peripheral blood was drawn into SST tubes (Becton-Dickinson, Franklin Lakes, NJ), and two measures of systemic inflammation, IL-6 and CRP, were assessed. SST tubes were spun for 10 minutes at 1200 rpm between 60 and 120 minutes after the blood draw, and serum was stored at −30°C until further analysis. Both elevated levels of IL-6 and CRP have been linked to increased cardiovascular disease risk (37–39). Serum IL-6 levels were measured using a high-sensitivity enzyme-linked immunosorbent assay kit (R&D Systems, Minneapolis, MN; intra-assay coefficient of variance [CV] < 10%; detection threshold = 0.04 pg/ml). CRP assays were conducted using a high-sensitivity, chemiluminescent technique (interassay CVs = 2.2%; detection threshold = −0.20 mg/l).

In addition to basal levels of systemic inflammation, we also assessed the ability of participants' white blood cells to respond to microbial challenge. We cultured cells with lipopolysaccharide (LPS), a bacterial stimulus that causes cells to secrete cytokines. This procedure was performed among a subset of 143 adolescents (randomly subsampled irrespective of age, sex, or ethnicity, to limit cost; these adolescents were not significantly different from adolescents for whom stimulated cytokine data are not available with respect to age, sex, ethnicity, body mass index [BMI], total family income, and levels of family chaos;

all p values >.20). Adolescents' whole blood was drawn into sodium-heparin Vacutainers (Becton-Dickinson) and subsequently 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. Supernatants were collected and frozen at −30°C until further analysis. Interleukin-1β (IL-1β), IL-6, interleukin-8 (IL-8), and interleukin-10 (IL-10) were measured in duplicate with MSD Meso Scale Discovery Human ProInflammatory 7-Plex Base Kits (MSD, Rockville, MD), with a minimum detection threshold of 0.15 pg/ml. Interassay and intra-assay CVs were less than 10%. See Table 1 for descriptive information regarding adolescent inflammatory outcomes.

IL-1β, IL-6, and IL-8 are all proinflammatory proteins that are released in response to bacterial products, involved in the acute inflammatory response, and produced primarily by macrophages. As the stimulated production of these cytokines was highly correlated in our sample (see Table 2), we combined the stimulated production of these three cytokines into one composite variable to obtain a more stable estimate of stimulated proinflammatory cytokine production and to reduce the number of analyses required to test our main hypotheses.

Covariates

Adolescents and their parents reported their age, sex, and ethnicity. Two dummy variables were created to compare adolescents of Asian and "other" origin to white adolescents. In addition, adolescents' height and weight were measured using a medical-grade scale and height rod during the laboratory visit without shoes and outerwear, and BMI was computed as weight in kilograms divided by height in meters squared.

Analyses

Levels of inflammatory markers were not normally distributed and consequently log transformed to reduce skewness. A composite variable representing proinflammatory responses to in vitro bacterial stimulation was computed by first standardizing and then summing concentrations of LPS-stimulated IL-1β, IL-6, and IL-8. Multiple linear regression analyses were performed to independently assess the main effects of family chaos and SES (family income) on adolescent inflammatory profiles. Next, hierarchical multiple regression analyses were used to investigate the two-way interaction effects of family chaos × SES on adolescent inflammatory profiles. First, covariates, family chaos, and SES scores were centered at zero, and family chaos and family income were multiplied to create the interaction term as recommended by Aiken and West (40). In Step 1 of analyses, we controlled for adolescents' age, sex, and ethnicity and entered family chaos and SES as main effects. In Step 2, the family chaos × SES interaction term was entered. All analyses were performed using SPSS version 18.0 (IBM, New York, NY).

RESULTS

Main Effects of Family Chaos and SES

First, we assessed the independent main effects of family chaos and family SES on systemic and stimulated adolescent cytokine production.

Systemic Inflammation

When entered as predictors independently, there was a trend toward greater family chaos being associated with higher levels of CRP ($B = 0.020$, standard error [SE] = 0.011, $p = .068$; $\Delta R^2 = 0.014$). Family chaos was unrelated to basal levels of IL-6 ($B = 0.005$, SE = 0.008, $p = .51$; $\Delta R^2 = 0.002$). Higher SES was associated with significantly lower levels of CRP ($B = -0.036$, SE = 0.016, $p = .024$; $\Delta R^2 = 0.021$) and significantly lower levels of basal IL-6 ($B = -0.027$, SE = 0.011, $p = .022$; $\Delta R^2 = 0.021$).

Stimulated Cytokine Production

Greater family chaos was associated with significantly higher levels of our composite variable of stimulated proinflammatory cytokine production (reflecting stimulated IL-1β, IL-6, and IL-8 production; $B = 0.204$, SE = 0.081, $p = .013$; $\Delta R^2 = 0.043$) but not associated with levels of stimulated IL-10 production ($B = 0.011$, SE = 0.008, $p = .19$; $\Delta R^2 = 0.012$). There was a trend toward higher SES being associated with lower levels of stimulated proinflammatory cytokine production ($B = -0.218$, SE = 0.115, $p = .059$; $\Delta R^2 = 0.025$). SES was unrelated to stimulated IL-10 production ($B = -0.007$, SE = 0.011, $p = .53$; $\Delta R^2 = 0.003$).

Interaction Effects of Family Chaos × SES

Second, we investigated whether SES moderates the association between family chaos and systemic and stimulated adolescent cytokine production. See Table 3 for a summary of the interaction effects.

Systemic Inflammation

There was no significant effect of the family chaos × SES interaction term on CRP ($B = 0.009$, SE = 0.006, $p = .11$; $\Delta R^2 = 0.010$). However, there was a significant effect of the family chaos × SES interaction term on basal levels of IL-6 ($B = -0.010$, SE = 0.004, $p = .026$; $\Delta R^2 = 0.019$), such that, as SES declined, the detrimental effect of family chaos became more pronounced, and greater family chaos was associated with increased IL-6 levels among adolescents from lower SES households. See Figure 1 for a graphical depiction of the family chaos × SES interaction effect on adolescent IL-6 levels.

TABLE 2. Pearson Correlation Matrix of Main Predictor Variables and Inflammatory Outcomes

	Income	CHAOS	IL-6	CRP	Stimulated IL-1β	Stimulated IL-6	Stimulated IL-8	Stimulated IL-10
Income	1	−.003	−.17***	−.15**	−.11	−.10	−.02	−.03
CHAOS	—	1	.06	.12*	.11	.11	.26***	.10
IL-6	—	—	1	.38****	.38****	.34****	.24****	.19**
CRP	—	—	—	1	.23****	.24****	.16*	.12
Stimulated IL-1β	—	—	—	—	1	.61****	.40****	.30****
Stimulated IL-6	—	—	—	—	—	1	.63****	.51****
Stimulated IL-8	—	—	—	—	—	—	1	.53****
Stimulated IL-10	—	—	—	—	—	—	—	1

CHAOS = Confusion, Hubbub, and Order Scale; IL-6 = interleukin-6; CRP = C-reactive protein; IL-1β = interleukin-1β; IL-8 = interleukin-8; IL-10 = interleukin-10.
* $p < .10$, ** $p < .05$, *** $p < .01$, **** $p < .001$.

TABLE 3. Hierarchical Multiple Regression Analyses of Family Chaos × SES Interaction Predicting Adolescent Inflammatory Outcomes

	B	SE	*p*
IL-6			
Step 1			
Intercept	−0.159	0.021	<.001
Age	−0.051	0.020	.011
Sex	0.048	0.042	.26
Whites versus Asians	−0.055	0.046	.23
Whites versus ''other''	0.004	0.064	.95
Family chaos	0.006	0.008	.47
SES	−0.027	0.012	.021
ΔR^2 for Step 1 = 0.069, p = .009			
Step 2			
Intercept	−0.159	0.021	<.001
Age	−0.049	0.020	.013
Sex	0.047	0.042	.26
Whites versus Asians	−0.054	0.045	.24
Whites versus ''other''	0.002	0.063	.98
Family chaos	0.005	0.008	.48
SES	−0.028	0.011	.014
Family chaos × SES	−0.010	0.004	.026
ΔR^2 for Step 2 = 0.019, p = .026			
Overall model: R^2 = 0.088; $F(7,235)$ = 3.25, p = .003			
CRP			
Step 1			
Intercept	−.411	.028	<.001
Age	.009	.027	.74
Sex	.002	.058	.98
Whites versus Asians	−.029	.062	.64
Whites versus ''other''	.054	.087	.53
Family chaos	.020	.011	.061
SES	−.036	.016	.021
ΔR^2 for Step 1 = 0.043, p = .11			
Step 2			
Intercept	−.411	.028	<.001
Age	.008	.027	.76
Sex	.001	.057	.98
Whites versus Asians	−.031	.062	.62
Whites versus ''other''	−.055	.087	.52
Family chaos	.020	.011	.056
SES	−.035	.016	.026
Family chaos × SES	.009	.006	.11
ΔR^2 for Step 2 = 0.010, p = .11			
Overall model: R^2 = 0.053; $F(7,236)$ = 1.88, p = .073			
LPS-stimulated proinflammatory cytokine composite			
Step 1			
Intercept	.143	.210	.50
Age	.262	.202	.20
Sex	−.188	.426	.66
White versus Asian	−.491	.468	.30
White versus ''other''	−1.026	.639	.11
Family chaos	.204	.080	.012
SES	−.219	.112	.054
ΔR^2 for Step 1 = 0.087, p = .051			
Step 2			
Intercept	.138	.207	.51
Age	.254	.199	.21
Sex	−.155	.420	.71
White versus Asian	−.398	.463	.39
White versus ''other''	−.955	.630	.13
Family chaos	.196	.079	.015
SES	−.257	.112	.023
Family chaos × SES	−.098	.044	.026
ΔR^2 for Step 2 = 0.033, p = .026			
Overall model: R^2 = 0.120; $F(7,134)$ = 2.62, p = .014			
LPS-stimulated IL-10			
Step 1			
Intercept	2.279	.021	<.001
Age	−.020	.020	.32
Sex	−.011	.043	.81
White versus Asian	−.135	.047	.005
White versus ''other''	−.054	.064	.40
Family chaos	.011	.008	.19
SES	−.007	.011	.52
ΔR^2 for Step 1 = 0.071, p = .12			
Step 2			
Intercept	2.279	.021	<.001
Age	−.021	.020	.30
Sex	−.008	.043	.85
White versus Asian	−.128	.047	.007
White versus ''other''	−.049	.064	.44
Family chaos	.010	.008	.21
SES	−.010	.011	.39
Family chaos × SES	−.007	.004	.13
ΔR^2 for Step 2 = 0.016, p = .13			
Overall model: R^2 = 0.087; $F(7,135)$ = 1.83, p = .087			

CHAOS = Confusion, Hubbub, and Order Scale; SES = socioeconomic status; IL-6 = interleukin-6; CRP = C-reactive protein; LPS = lipopolysaccharide.
The LPS-stimulated cytokine composite represents the sum of standardized LPS-stimulated interleukin-1β, IL-6, and interleukin-8 levels.

Stimulated Cytokine Production

The family chaos × SES interaction term was significantly associated with our composite variable of LPS-stimulated proinflammatory cytokine production (B = −0.098, SE = 0.044, p = .026; ΔR^2 = 0.033). Similar to the effect above, as SES declined, the effect of family chaos became more pronounced, meaning that greater family chaos was associated with increased stimulated proinflammatory cytokine production among adolescents from lower SES households.[1] See Figure 2 for a

[1]In ancillary analyses, we also examined the effect of family chaos and SES on the stimulated production of each of the proinflammatory cytokines included in our composite variable to check whether our composite results were being driven by one particular cytokine. In general, patterns for individual cytokines were all in the same direction, although at times weaker, suggesting that no one proinflammatory cytokine was driving the results.

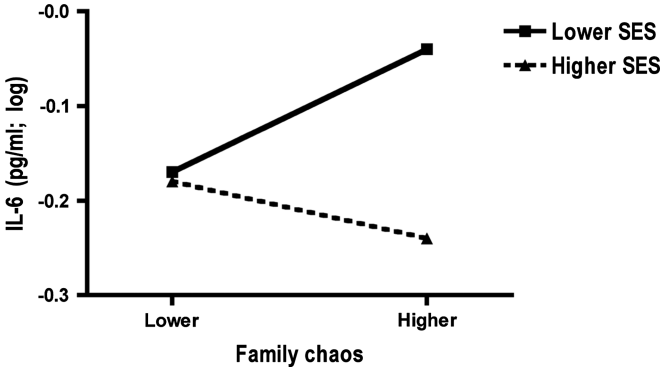

Figure 1. SES moderates the association between family chaos and basal levels of adolescent IL-6. Interaction of family chaos and family SES predicting basal levels of IL-6 among adolescents (IL-6: $B = -0.010$, SE = 0.004, $p = .026$). Both family chaos and family SES are depicted at ±1 SD. SES = socioeconomic status; IL-6 = interleukin-6; SE = standard error; SD = standard deviation.

graphical representation of these results. Finally, there was no effect of the family chaos × SES interaction term on stimulated IL-10 production ($B = -0.007$, SE = 0.004, $p = .13$; $\Delta R^2 = 0.016$).

Effect of BMI

Greater BMI has been linked to greater inflammation (41), so we tested whether including BMI as an additional covariate would alter our findings. After the inclusion of BMI, main effects of SES and family chaos remained largely unchanged, with the exception that family chaos no longer predicted CRP ($B = 0.009$, SE = 0.010, $p = .36$; $\Delta R^2 = 0.003$).

Regarding our interaction effects, after the inclusion of BMI, the family chaos × SES interaction term remained a significant predictor of basal levels of IL-6 production ($B = -0.010$, SE = 0.004, $p = .015$; $\Delta R^2 = 0.021$) and of the stimulated proinflammatory cytokine production composite ($B = -0.095$, SE = 0.042, $p = .027$; $\Delta R^2 = 0.031$).

Effect of Parenting

To determine whether the influence of family chaos is separate from other, relational family characteristics, we also investigated the influence of parenting styles. Generally, patterns between SES and family chaos remained unchanged when including harsh or nurturant parenting as additional covariates, suggesting that there is something unique about family chaos as a moderator of SES effects on inflammatory processes.

After the inclusion of nurturant or harsh parenting as additional covariates in the interaction analyses, the interaction term continued to significantly predict IL-6 (harsh parenting: $B = -0.009$, SE = 0.004, $p = .033$; $\Delta R^2 = 0.018$; nurturant parenting: $B = -0.009$, SE = 0.004, $p = .044$; $\Delta R^2 = 0.016$) and trended toward predicting CRP (harsh parenting: $B = 0.011$, SE = 0.006, $p = .066$; $\Delta R^2 = 0.014$; nurturant parenting: $B = 0.012$, SE = 0.006, $p = .047$; $\Delta R^2 = 0.016$). The effect of the interaction term on the stimulated cytokine composite remained marginally significant (harsh parenting: $B = -0.081$, SE = 0.046, $p = .080$; $\Delta R^2 = 0.021$; nurturant parenting: $B = -0.079$, SE = 0.046, $p = .086$; $\Delta R^2 = 0.021$). We did not test the effect of the inclusion of parenting on stimulated IL-10 production because stimulated IL-10 was not significantly predicted in the original model.

We also examined whether parenting interacted with SES. Here, we found no evidence of SES × nurturant parenting or SES × harsh parenting interactions on adolescent inflammatory outcomes (all p values >.24).

DISCUSSION

To our knowledge, this is the first study that has linked greater family chaos to a proinflammatory phenotype among healthy adolescents. We found that adolescents from more chaotic families had higher levels of CRP, indicative of greater systemic inflammation, and also showed evidence of significantly higher levels of LPS-stimulated proinflammatory cytokine production in vitro. However, the latter effect was qualified by an interaction with SES, such that the detrimental effect of greater family chaos on systemic inflammation and LPS-stimulated cytokine production was increasingly pronounced as adolescents' family SES declined, essentially placing these already vulnerable youth at a particularly high risk for adverse inflammatory profiles. Although, in general, effect sizes were small (accounting for 2%–4% of the variance), this is in line with the effects of other psychosocial variables on inflammatory markers in other studies (42).

These findings are in line with previous research that has linked other family factors, for example, the provision of daily family assistance and the family climate, to inflammation among youth (19,32). It also further supports previous research that has found moderating effects of SES on a number of psychosocial factors; for example, hostile child-rearing attitudes of mothers and individuals' sense of control seem to have stronger influences on health outcomes among youth and adults from low SES backgrounds (12,16). Nonetheless, only a few studies to date have specifically examined interaction effects of family stress and SES and their influence on inflammatory outcomes, providing evidence that low SES adults exposed to greater maternal warmth during childhood exhibit reduced

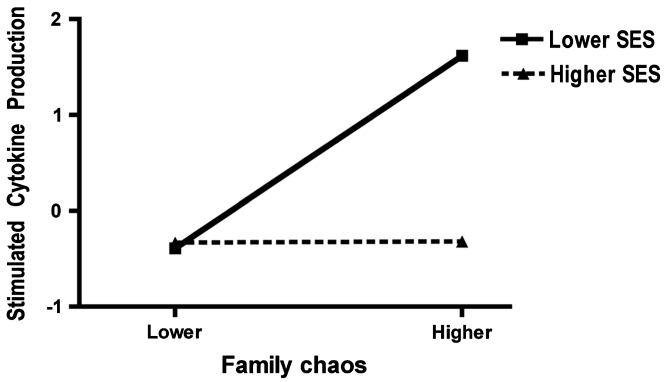

Figure 2. SES moderates the association between family chaos and adolescent-stimulated proinflammatory cytokine production. Interaction of family chaos and family SES predicting levels of LPS-stimulated proinflammatory cytokine production (a composite of IL-1β, IL-6, and IL-8 production) among adolescents ($B = -0.098$, SE = 0.044, $p = .026$). Both family chaos and family SES are depicted at ±1 SD. Stimulated cytokine production represents a composite of LPS-stimulated IL-1β, IL-6, and IL-8, computed by standardizing and subsequently summing cytokine concentrations. SES = socioeconomic status; LPS = lipopolysaccharide; IL-1β = interleukin-1β; IL-6 = interleukin-6; and IL-8 = interleukin-8; SE = standard error; SD = standard deviation.

proinflammatory profiles (43), that low SES children with asthma show greater proinflammatory airway responses after family conflict (44), and that youth exposed to greater cumulative risk had greater levels of allostatic load, but only among youth also exposed to low maternal responsiveness (13). The present findings suggest that a chaotic family environment represents another type of family characteristic—more related to the structural aspects of family life than interpersonal relationships—that is associated with health-relevant inflammatory processes in youth, and that, as SES declines, adolescents are particularly vulnerable to the negative effects of chaotic family environments, warranting further study of interaction effects between SES and family psychosocial variables. In addition, these results were consistent even after controlling for different styles of parenting, including nurturant and harsh parenting, suggesting that family chaos represents more than just a marker of relational family characteristics.

One possible explanation for our findings may be that a family environment marked by disorganization ceases to be a refuge from other stressors that youth may experience at school or in their neighborhoods. For example, lower SES youth are more likely to encounter stressors in their neighborhood environments (45). A quiet and structured family environment may provide these youth with a place to recover from these outside stressors; thus, lower SES youth with low family chaos show relatively lower levels of inflammation and lower responsiveness to mitogen challenge. However, in the absence of such a place and when the home environment adds additional burdens to youth's lives rather than providing a healthy balance, the immune systems of lower SES youth living with high family chaos consequently may tip toward a more chronically elevated, proinflammatory state.

Another explanation may be that cumulative exposure to multiple stressors in multiple domains, for example, the neighborhood and the family environment, takes an especially large toll on inflammatory processes in youth. Research on multiple risk exposures posits that individuals from low and high SES backgrounds experience very different environments, low SES environments being marked by the presence of weaker support systems, greater noise and air pollution, and more crowded, lower-quality, and less warm home environments (46,47). It has furthermore been shown that this type of cumulative risk is associated with numerous adverse health outcomes, including a greater risk of obesity, depression, diabetes, and ischemic heart disease, among others (48). In the context of the present study, this may suggest that, among lower SES youth, chaotic homes may provide an additional stressor on top of the many other stressors (e.g., violence and pollution) that lower SES youth frequently experience in their daily lives. In contrast, higher SES youth who live in chaotic homes may experience this as their primary stressor, without additional significant background stressors in their daily lives. In the absence of other stressors, higher SES youth may be able to better adjust to and deal with their chaotic family environments, avoiding the negative physiological consequences of the same.

The cross-sectional nature of this study presents a limitation, and future studies should investigate the combined influence of family chaos and SES on adolescent inflammatory profiles longitudinally, as it is currently unknown whether the effects of family chaos on systemic and LPS-stimulated proinflammatory cytokine production persist or change over time. Going forward, larger sample sizes are also desirable to ascertain the robustness of these findings. In addition, it is worth considering that although families participating in the present study reported a wide range of home disorganization, overall family chaos scores were relatively low (although comparable to norms for the measure) (34), suggesting that effects of family chaos seen here may represent conservative estimates of the negative effects family chaos can have on adolescents. Furthermore, in this study, parents reported on family chaos. However, previous research has shown that different family members experience chaotic home environments slightly differently (6). Because parents and their children may not share all environmental aspects of the home environment, especially as children get older, reach adolescence, and become more independent, adolescent reports of family chaos may represent an important perspective to ascertain the stressors they experience in their homes. We also note that the CHAOS questionnaire is a self-report measure and that behavioral home observations would be a good complement to assessing environmental confusion in homes. In addition, the present study only assessed a limited number of inflammatory markers. Longitudinal studies evaluating the potential long-term implications of these patterns on clinical health outcomes among adolescents are also needed. Finally, one somewhat unusual finding in this study was that age was negatively associated with IL-6 within our sample of 13- to 16-year-olds. However, because CRP did not show similar patterns and was unrelated to age, we do not elaborate on these patterns further.

In sum, as the SES environments that adolescents grow up in decline, these adolescents are more likely to be exposed to more chaotic and less organized home environments (3). Simultaneously, they are also at an increased risk for adverse physical health outcomes (30). The present study demonstrates that youth from lower SES families are particularly vulnerable to proinflammatory phenotypes if simultaneously exposed to both lower SES and a more chaotic family environment. These findings suggest that interventions to aid in the creation of more structured home environments may help lower these adolescents' proinflammatory profiles, which, over the long term, may reduce the risk of the types of chronic diseases of aging that are associated with inflammation in this at-risk population. They also draw attention to the importance of tailoring interventions toward the needs of lower SES families, making issues such as chaotic homes a particularly important point to address among lower SES families. One possible solution to addressing chaotic family environments among lower SES families may be to provide such families with greater access to services delivered in kind, such as childcare and transport and accommodation services, all of which may give lower SES parents additional support with managing their everyday lives and adapting and maintaining more routinized and structured home environments for their children.

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