Chronic Family Stress and Adolescent Health: The Moderating Role of Emotion Regulation

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ABSTRACT

Objective: The aim of the study was to assess whether the association between chronic family stress and physiological measures is moderated by emotion regulation strategies in an adolescent sample.

Methods: Chronic family stress was assessed via a semistructured interview and emotion regulation strategies (cognitive reappraisal and suppression) via questionnaire among 261 adolescents (14.57 (1.07) years). Several metabolic (waist-hip ratio, systolic and diastolic blood pressure) and inflammatory markers (basal and stimulated proinflammatory cytokine production in response to bacterial challenge) as well as glucocorticoid sensitivity were assessed.

Results: There were no main effects of chronic family stress, cognitive reappraisal, or suppression on physiological measures (all p's > .10). Emotion regulation moderated the association between chronic family stress and physiological measures. As chronic family stress increased, adolescents higher in cognitive reappraisal had smaller waist-hip ratios ($B = -.003$, SE = .001, $p = .015$) and lower systolic blood pressure ($B = -.303$, SE = .143, $p = .035$), although no moderation was found with respect to inflammatory markers and glucocorticoid sensitivity (all p's > .30). In addition, as chronic family stress increased, adolescents higher in suppression showed evidence of higher stimulated proinflammatory cytokine production ($B = .046$, SE = .020, $p = .021$) and lower glucocorticoid sensitivity ($B = .051$, SE = .021, $p = .015$), although basal inflammation and metabolic measures were not moderated by suppression (all p's > .50).

Conclusions: This study suggests that the types of emotion regulation strategies used by adolescents may affect the extent to which chronic family stress affects important metabolic and immune processes.

Key words: chronic family stress, emotion regulation, glucocorticoid sensitivity, inflammation, metabolic health.

INTRODUCTION

Psychological stress involves a person's appraisal of a situation as both threatening and surpassing his or her abilities to successfully cope (1). These stressors can be categorized as being either acute or chronic. Acute stressors are typically time limited and discrete (e.g., public speaking), whereas chronic stressors persist for longer periods (e.g., dysfunctional family relationships). Chronic stress in childhood has been associated with adverse physical and psychological health (2), including greater risk for developing depression (3), autoimmune disorders (4), and cardiovascular disease (5). In addition, early markers of physiological risk can be observed in adolescence (6).

One pathway through which chronic stress may increase disease risk is via dysregulation of key aspects of physiological functioning, such as inflammatory and metabolic processes (7,8). Chronic stress has been linked to increased low-grade inflammation (9), in part following the dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis in the face of ongoing chronic stress (10,11). Specifically, chronic stress may disrupt the negative feedback loop of the HPA axis via the continual release of high concentrations of cortisol. This may result in excess inflammation in response to pathogens because proinflammatory cytokines become less sensitive to cortisol signaling (12,13). Chronic stress has also been linked to cardiovascular disease risk factors, e.g., increased blood pressure and waist-hip ratio (WHR) (8).

Despite the established links between chronic stress and psychological and physiological functioning, individuals under chronic stress do not always succumb to disease (14,15), suggesting potential individual differences moderating the link between chronic stress and well-being (16,17). One psychological process that has received attention as a possible moderator between chronic stress and health is emotion regulation (18–20), a combination of processes and strategies through which an individual manages fluctuations in emotions (21). These strategies include cognitive reappraisal and suppression (18,19,22). Cognitive reappraisal involves positively reframing a stimulus in a way that alters the potential emotional and physiological response (e.g., reframing criticism as a learning opportunity), whereas suppression involves

BMI = body mass index, **CRP** = C-reactive protein, **CV** = coefficient of variance, **DBP** = diastolic blood pressure, **ERQ** = Emotion Regulation Questionnaire, **HPA** = hypothalamic-pituitary-adrenal, **IL** = interleukin, **LPS** = lipopolysaccharide, **LSI** = Life Stress Inventory, **M (SD)** = mean (standard deviation), **SBP** = systolic blood pressure, **SST** = serum separator tubes, **WHR** = waist-to-hip ratio

SDC Supplemental Content

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Received for publication December 5, 2017; revision received June 22, 2018.

DOI: 10.1097/PSY.0000000000000624

the inhibition of overt emotional expression in response to stressful stimuli (e.g., outwardly presenting as composed in the face of criticism, but experiencing physical arousal and inhibiting facial expressions). Generally speaking, use of cognitive reappraisal has been linked to better health (22) and fewer negative emotions (21), whereas suppression has been associated with poorer psychosocial outcomes (22–24), such as less social connectedness (25).

Similar patterns emerge when considering the physiological sequelae of emotion regulation use. Within laboratory settings, adult participants' use of cognitive reappraisal has been linked to reduced sympathetic arousal (26,27), lower blood pressure (28), and an adaptive cardiovascular challenge response (29–32), whereas use of emotion suppression has been linked to greater sympathetic arousal (26), higher blood pressure (28,33), and greater cardiovascular threat responses (29,30). Similarly, observational data suggest greater use of cognitive reappraisal and suppression to be associated with lower and higher C-reactive protein levels, respectively (34). These findings suggest that, at least among adults, associations between stress and physiological measures depend on the specific emotion regulation strategies on which individuals draw.

Understanding these connections during adolescence, a sensitive period marked by changes in biological and psychosocial systems (35,36) and self-regulatory processes, such as emotion regulation (36), is important. Although there is a shift towards the prioritization of friendships, family relationships remain important contributors to youth's emotional and physiological well-being (37–40), at times over and above the influence of peers (37,41). Adolescents also report more frequent conflicts with parents compared with peers (38), suggesting that emotion regulation strategies may be more consistently used within the family context. Finally, adolescence is marked by puberty-driven increases in sex hormone production (35), which influence HPA axis sensitivity (17). HPA axis basal activation and stress reactivity appear heightened (17), which may advance alterations in glucocorticoid sensitivity of proinflammatory cytokines (12,13) that in turn contribute to pronounced inflammation in response to chronic stress during adolescence (3,42). Given these concurrent, interconnected biological and psychological changes, adolescents may be particularly vulnerable to the effects of chronic stress (13).

This study investigates emotion regulation as a possible moderator of the association between chronic family stress and relevant adolescent inflammatory and metabolic measures (10,13). In line with previous research, we hypothesize that greater chronic family stress during adolescence will be associated with poorer metabolic and inflammatory profiles; that the association between chronic family stress and these measures will be moderated by emotion regulation, specifically cognitive reappraisal and suppression; that the adverse influence of chronic family stress will be weaker among adolescents who use higher levels of cognitive reappraisal; and that greater use of suppression will be associated with more adverse physiological consequences in response to chronic family stress.

METHODS

Participants

Participants were 261 adolescents between the ages of 13 and 16 years (53% female) who were accompanied to the laboratory by one of their caregivers (76% mothers). The dyads were from the greater Vancouver, British Columbia, area and were recruited between January 2010 and March 2012 via local media advertisements. Participants were prescreened over the phone. They needed to be fluent in English and not have any chronic illness diagnoses. All dyads who met the necessary requirements were then scheduled for after-school appointments. If adolescents reported acute illnesses, they rescheduled for 4 weeks after the end of symptoms. In addition, if initial results of complete blood cell counts demonstrated elevated white blood cell counts, then blood draws were rescheduled. Parents and youth received CAD 75 each for their participation in the laboratory session. Most participating adolescents identified as being of European descent (49.4%), with the remainder identifying as Asian (36%) or other (14.6%) descent. Parents reported a range of socioeconomic backgrounds, which are detailed in Table 1 along with other participant characteristics.

Procedure

Adolescents and caregivers provided written assent and consent, respectively, when they arrived for their appointment. Each provided demographic information through interviews with trained research assistants. The adolescent participated in a semistructured interview with a trained research assistant assessing chronic stress and completed a self-report questionnaire on emotion regulation. Finally, adolescents underwent a peripheral blood draw through antecubital venipuncture, performed by a trained phlebotomist. The dyads were reimbursed for their participation. The study was approved by the Clinical Research Ethics Board of The University of British Columbia.

Measures

Chronic Family Stress

Adolescents participated in the University of California Los Angeles Life Stress Interview (LSI), Adolescent Version (43), with a trained research assistant. The LSI addressed chronic stress for the past 6 months across the following four domains: family relationships, peer relationships, school, and home life. Of focus for this study was the family relationships domain, which addressed the adolescents' relationships with all family members for the previous 6 months. Harsh family relationships have been consistently associated with socioemotional vulnerabilities (36,44) and physiological measures including greater inflammation and disease risk (13,42). In addition, chronic family stress has been shown to more strongly predict physiological markers among adolescents than chronic stress outside the home (e.g., (37,45)). Family ratings reflect adolescents' responses to open-ended questions focusing on levels of trust, openness, and conflict between the adolescent and other family members, as well as how long the relationships have been this way. Research assistants rated family relationships on a scale of 1–5, with 1 representing exceptional quality in relationships. A "5" represented poor family relationships, characterized by frequent arguments and parent unavailability. Within this sample, most scores fell within the 1–3 range. Although, on average, scores were on the lower end of the scale, these ratings suggest qualitatively different family environments. A "1" suggests secure relationships with both parents, few disagreements and successful problem solving. For a "3," families were more likely to experience regular arguments, not be comfortable sharing information, and may not provide emotional support. Scores of 3.5 or higher were reflective of more severe dysfunction, such as temporary foster care placement. The interrater reliability within this study ranged from .88 to .94 across subscales.

Emotion Regulation

Adolescents completed the Emotion Regulation Questionnaire (ERQ; (19)). The ERQ consists of 10 items assessing two emotion regulation strategies: cognitive reappraisal (6 items; e.g., "I control my emotions by changing the way I think about the situation I'm in") and expressive suppression (4 items; e.g., "I keep my emotions to myself"). Items were rated using a seven-point Likert scale (1 = strongly disagree, 7 = strongly agree) such that

TABLE 1. Sample Descriptives

	Total Sample (n = 261)		Adolescents Subsampled for Cytokine Production and Glucocorticoid Sensitivity (n = 151)	
	n (%)	M (SD)	n (%)	M (SD)
Male	122 (46.7)		68 (45.0)	
Female	139 (53.3)		83 (55.0)	
Age, y		14.5 (1.1)		14.6 (1.1)
Ethnicity				
European	129 (49.4)		80 (53)	
Asian	94 (36.0)		49 (32.5)	
Other	38 (14.6)		22 (14.6)	
BMI, kg/m^2		21.37 (3.70)		21.25 (3.64)
Total family income				
<$5000	4 (1.5)		2 (1.3)	
$5000–$19,999	12 (4.6)		7 (4.6)	
$20,000–$34,999	21 (8.0)		14 (9.3)	
$35,000–$49,999	34 (13.0)		16 (10.6)	
$50,000–$74,999	59 (22.6)		35 (23.2)	
$75,000–$99,999	36 (13.8)		17 (11.3)	
$100,000–$149,999	52 (19.9)		29 (19.2)	
$150,000–$199,999	28 (10.7)		19 (12.6)	
>$200,000	13 (5.0)		11 (7.3)	
Chronic family stress		2.10 (.70)		2.09 (.71)
ERQ cognitive reappraisal		28.65 (5.94)		28.71 (6.00)
ERQ expressive suppression		14.76 (4.91)		14.69 (5.18)
Metabolic markers				
Waist-to-hip ratio		1.02 (.81)		
Systolic blood pressure		102.50 (9.49)		
Diastolic blood pressure		62.22 (8.96)		
Inflammatory markers				
IL-6, pg/ml; log		−.16 (.34)		
CRP, mg/l; log		−.41 (.44)		
LPS only				
IL-1β, pg/ml; log				3.73 (.37)
IL-6, pg/ml; log				4.50 (.16)
IL-8, pg/ml; log				4.20 (.28)
LPS and hydrocortisone				
IL-1β, pg/ml; log				2.74 (.45)
IL-6, pg/ml; log				3.82 (.26)
IL-8, pg/ml; log				3.59 (.32)

BMI = body mass index; IL-6 = interleukin-6; CRP = C-reactive protein; LPS = lipopolysaccharide; M (SD) = mean (standard deviation).

χ^2 tests and 2 sample t tests were used to compare the adolescents subsampled for cytokine production and glucocorticoid sensitivity to the entire sample. The subsample of adolescents for blood cultures did not significantly differ from the total sample on any of the main variables identified previously.

higher scores reflected more habitual use of the emotion regulation strategies. Responses to items representing each emotion regulation skill were summed to produce total cognitive reappraisal and expressive suppression scores. There was adequate internal consistency for the cognitive reappraisal (Cronbach's α = .76) and expressive suppression (Cronbach's α = .72) scales within our study, comparable with previous estimates (19).

Metabolic Markers

After a 5-minute wait period during which they were asked to sit quietly, three readings of adolescents' systolic (SBP) and diastolic blood pressure (DBP) were taken at 2-minute intervals for a 6-minute period using a VSM-100 BpTRU (BpTRU Medical Devices; Coquitlam, British Columbia) automatic blood pressure monitor and standard occluding cuff. An average score was calculated for SBP and for DBP based on the three readings.

Using a cloth tape measure, research assistants measured the circumference of the midpoint between the hip bone and lowest rib to determine waist size. Hip measurements were taken at the widest point around the buttocks, including the gluteofemoral fold. Measurements were taken twice to ensure consistency of values. WHR for each adolescent was determined by

TABLE 2. Multiple Regression Analyses of the Interaction Effects of Chronic Family Stress and Emotion Regulation on Adolescent Metabolic and Inflammatory Biomarkers

	Cognitive Reappraisal				Suppression			
	Unstd. *B*	Std. *B*	SE	*p*	Unstd. *B*	Std. *B*	SE	*p*
Metabolic markers								
Waist-to-hip ratio								
Intercept	.819		.009	<.001	.821		.017	<.001
Chronic family stress	−.003	−.033	.007	.628	−.001	−.015	.007	.833
Emotion regulation	<.001	.020	.001	.766	7.348E-5	.005	.001	.941
Stress by emotion regulation	−.003	−.158	.001	.015**	−.001	−.037	.001	.563
Overall model	R^2 = .053; F(8,244) = 1.655, p = .110				R^2 = .030; F(8, 244) = .907, p = .511			
Systolic blood pressure								
Intercept	104.713		1.108	<.001	104.813		1.127	<.001
Chronic family stress	−.702	−.052	.896	.434	−.603	−.045	.916	.511
Emotion regulation	−.101	−.063	.104	.331	.151	.078	.129	.243
Stress by emotion regulation	−.303	−.135	.143	.035**	.024	.008	.181	.895
Overall model	R^2 = .073; F(8, 244) = 2.318, p = .021				R^2 = .057; F(8, 244) = 1.792, p = .079			
Diastolic blood pressure								
Intercept	60.201		1.042	<.001	60.344		2.014	<.001
Chronic family stress	−1.519	−.121	.846	.074	−1.382	−.110	.862	.110
Emotion regulation	−.040	−.027	.098	.682	.067	.037	.121	.580
Stress by emotion regulation	−.258	−.123	.135	.057*	.017	.006	.170	.922
Overall model	R^2 = .060; F(8, 243) = 1.878, p = .064				R^2 = .046; F(8, 243) = 1.422, p = .188			
Inflammatory markers								
IL-6								
Intercept	−.153		.038	<.001	−.150		.038	<.001
Chronic family stress	−.011	−.022	.030	.727	.007	.014	.031	.825
Emotion regulation	−.006	−.101	.004	.100	−.005	−.076	.004	.223
Stress by emotion regulation	.006	.068	.005	.256	−.001	−.005	.006	.933
Overall model	R^2 = .187; F(9, 242) = 5.969, p = <.001				R^2 = .179; F(9, 242) = 5.650, p = <.001			
CRP								
Intercept	−.418		.049	<.001	−.402		.050	<.001
Chronic family stress	−.026	−.042	.040	.511	−.015	−.023	.041	.718
Emotion regulation	.004	.050	.005	.418	−.006	−.061	.006	.333
Stress by emotion regulation	−.009	−.079	.007	.194	−.006	−.044	.008	.472
Overall model	R^2 = .169; F(9, 243) = 5.302, p = <.001				R^2 = .167; F(9, 243) = 5.199, p = <.001			
Stimulated proinflammatory cytokine production								
Intercept	.262		.254	.304	.215		.252	.394
Chronic family stress	−.055	−.046	.106	.606	−.106	−.090	.108	.330
Emotion regulation	.004	.029	.012	.742	−.0002	.-002	.015	.985
Stress by emotion regulation	−.018	−.086	.018	.317	.046	.205	.020	.021**
Overall model	R^2 = .099; F(9, 139) = 1.341, p = .222				R^2 = .116; F(9, 139) = 1.889, p = .059			
Glucocorticoid sensitivity								
Intercept	.621		.268	.022	.533		.262	.044
Chronic family stress	.044	.035	.111	.693	−.039	−.032	.113	.727
Emotion regulation	−.006	−.041	.013	.637	.017	.101	.015	.258
Stress by emotion regulation	−.010	−.046	.019	.590	.051	.213	.021	.015**
Overall model	R^2 = .070; F(9, 139) = 1.085, p = .378				R^2 = .122; F(9, 139) = 2.195, p = .026			

IL-6 = Interleukin-6; CRP = C-reactive protein; SE = standard error.

All analyses controlled for age, sex, ethnicity, and income. BMI was also controlled for when considering inflammatory measures.

* p < .10.

** p < .05.

dividing waist circumference by hip circumference. Metabolic measures were examined individually.

Inflammatory Markers

We considered three indicators of inflammatory mechanisms: basal inflammation, stimulated cytokine production, and glucocorticoid sensitivity. Basal inflammation reflects the number of immune cells present in the peripheral blood in the absence of acute infection; stimulated cytokine production refers to immune cell response to in vitro stimulation with a bacterial pathogen; and glucocorticoid sensitivity is an indication of the responsiveness of immune cells to anti-inflammatory signals from glucocorticoids.

Basal Inflammation

Adolescents' peripheral blood was drawn into serum separator tubes (SST; Becton-Dickinson, Franklin Lakes, NJ). SST were spun for 10 minutes at 1200 relative centrifugal force between 60 and 120 minutes after the blood draw, and serum was stored at −30°C until analysis. Basal levels of interleukin 6 (IL-6) and C-reactive protein (CRP) were measured to assess systemic, low-grade inflammation. Low-grade inflammation is present throughout the body rather than being localized to the site of a particular injury. Although of lower intensity than acute inflammation, it is sustained over time and has been associated with increased risk for cardiovascular disease (9,46,47). A high-sensitivity enzyme-linked immunosorbent assay (ELISA) kit was used to measure serum IL-6 levels (R&D Systems, Minneapolis, MN; intra-assay coefficient of variance [CV] <10%; detection threshold = 0.04 pg/ml). A high-sensitivity, chemiluminescent technique was used for the CRP assays (interassay CVs = 2.2%; detection threshold = −0.20 mg/l).

Stimulated Cytokine Production

Lipopolysaccharide (LPS)-stimulated cytokine activity was assessed in vitro in a randomly selected subsample of 151 adolescents (mean (SD) age = 14.6 (1.1), 55% females) to determine adolescents' immune responses to microbial challenge. This subsample of 151 adolescents was randomly selected to limit cost. These participants did not differ significantly from those for whom stimulated cytokine data were not available with respect to sex ($\chi^2(1) = .421$, $p = .616$), age ($t(259) = .535$, $p = .59$), body mass index ($t(259) = .596$, $p = .55$), socioeconomic status ($\chi^2(8) = 9.18$, $p = .33$), ethnicity ($\chi^2(2) = 2.180$, $p = .34$), chronic family stress ($t(257) = -.293$, $p = .770$), reappraisal ($t(246) = .188$, $p = .851$), and suppression ($t(241) = -.259$, $p = .80$).

Peripheral blood mononuclear cells were cultured with LPS, an endotoxin found in the cell wall of gram-negative bacteria and known to stimulate the release of proinflammatory cytokines by immune cells. Adolescents' whole blood was drawn into sodium-heparin Vacutainers (Becton-Dickinson) and then diluted in a 9:1 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. IL-1β, IL-6, and IL-8 were measured in duplicate using MSD Meso Scale Discovery Human Proinflammatory 5-Plex Base Kits (MSD, Rockville) with a minimum detection threshold of 0.15 pg/ml. Inter- and intra-assay CVs were less than 10%. IL-1β, IL-6, and IL-8 are produced by a number of leukocytes, including macrophages, and are part of the acute proinflammatory response. Their standardized scores were averaged to create a proinflammatory cytokine composite score.

Glucocorticoid Sensitivity

In addition, glucocorticoid sensitivity was assessed in the same subsample of 151 adolescents. Peripheral blood mononuclear cells were incubated with LPS in the presence of hydrocortisone (final concentration = 2.76×10^{-5}). Cortisol assists with the downregulation of proinflammatory cytokines. Greater levels of LPS-stimulated in vitro cytokine production in the presence of hydrocortisone suggests decreased glucocorticoid sensitivity. Their standardized scores were averaged to create a composite score.

Covariates

Ethnicity, sex, age, and socioeconomic status were reported by caregivers and adolescents and included as covariates in all analyses. Dummy variables were created to compare adolescents of Asian or "other" descent to adolescents of European descent. Parents reported on total gross family income in Canadian dollars by selecting one of nine income levels ranging from "less than $5000" to "$200,000 and higher." Body mass index (BMI), which was calculated as participants' weight in kilograms divided by height in meters squared, was also included as a covariate when predicting inflammatory measures because it is associated with increased inflammatory activity (47).

Statistical Analyses

There were four outliers among the WHR scores, which were winsorized to within three SDs of the mean. Levels of basal and stimulated inflammatory markers were not normally distributed and were log transformed to reduce skewness and kurtosis.

Multiple linear regression analyses were performed to assess both main effects for chronic family stress (LSI), cognitive reappraisal (Emotion Regulation Questionnaire, reappraisal subscale), and suppression (Emotion Regulation Questionnaire, suppression subscale), as well as two-way interaction effects for chronic family stress by each emotion regulation subscale.

When considering the two-way interaction effects, all covariates and predictors were centered at zero. The interaction terms were created by multiplying the centered LSI and Emotion Regulation Questionnaire, reappraisal subscale and centered LSI and Emotion Regulation Questionnaire, suppression subscale scores, respectively, as recommended by Aiken and West (48). In step 1 of the analyses, centered covariates and predictors were entered, followed by the interaction term in step 2. Analyses were performed using SPSS Version 24.0 (IBM, New York, NY).

RESULTS

Scores for cognitive reappraisal ranged from 9 to 42 (sample mean (SD) = 28.65 (5.94)) and suppression scores ranged from 4 to 28 (sample mean (SD) = 14.76 (4.91)), with higher scores on each scale indicating greater use of that strategy. Metabolic measures were weakly to moderately correlated (all r's between .016 and .411), whereas IL-1β, IL-6, and IL-8 were highly correlated after stimulation with LPS only (r's between .407 and .604, all p's < .010) and stimulation with LPS and hydrocortisone (r's between .629 and .770, all p's < .010). For additional information regarding correlations between the main study variables in the full sample and subsample, please refer to Tables S1 and S2, respectively (Supplementary Digital Content 1, http://links.lww.com/PSYMED/A497). Main analyses were conducted using the composite scores (Table 2), but models for individual cytokines have been included for completeness (Table 3). When considering individual cytokines separately, findings were generally in the same direction as the findings when considering composite scores, albeit weaker.

Main Effects of Chronic Family Stress and Emotion Regulation

We first considered the independent main effects of chronic family stress and emotion regulation on adolescent metabolic and inflammatory measures. There were no main effects of chronic family stress, cognitive reappraisal, and suppression on any of the outcomes (all p's > .090).

TABLE 3. Multiple Regression Analyses of the Interaction Effects of Chronic Family Stress and Emotion Regulation on Individual Cytokines Within the Adolescent Subsample

	Cognitive Reappraisal				Suppression			
	Unstd. *B*	Std. *B*	SE	*p*	Unstd. *B*	Std. *B*	SE	*p*
Proinflammatory cytokine production (LPS only)								
IL-1β, pg/ml; log								
Intercept	3.789		.057	<.001	3.770		.056	<.001
Chronic family stress	−.043	−.083	.046	.351	−.064	−.122	.047	.174
Emotion regulation	.002	.031	.005	.719	−.005	−.069	.006	.431
Stress by emotion regulation	−.003	−.028	.008	.739	.026	.236	.009	.003**
Overall model	R^2 = .099; F(9, 139) = 1.594, p = .123				R^2 = .157; F(9, 139) = 2.681, p = .007			
IL-6, pg/ml; log								
Intercept	4.531		.025	<.001	4.528		.025	<.001
Chronic family stress	−.700	−.030	.020	.739	−.015	−.065	.021	.483
Emotion regulation	.002	.058	.002	.497	.001	.020	.003	.821
Stress by emotion regulation	−.003	−.085	.003	.318	.005	.117	.004	.185
Overall model	R^2 = .099; F(9, 244) = 1.591, p = .124				R^2 = .103; F(9, 139) = 1.662, p = .105			
IL-8, pg/ml; log								
Intercept	4.201		.043	<.001	4.190		.043	<.001
Chronic family stress	.002	.005	.035	.953	−.009	−.022	.036	.804
Emotion regulation	−.002	−.048	.004	.573	−.0003	−.005	.005	.953
Stress by emotion regulation	−.007	−.104	.006	.223	.015	.203	.007	.020**
Overall model	R^2 = .115; F(9, 138) = 1.854, p = .065				R^2 = .139; F(9, 138) = 2.319, p = .019			
Glucocorticoid sensitivity (LPS and hydrocortisone)								
IL-1β, pg/ml; log								
Intercept	2.834		.069	<.001	2.809		.068	<.001
Chronic family stress	−.005	−.008	.056	.928	−.037	−.060	.057	.516
Emotion regulation	−.002	−.032	.007	.718	.004	.041	.008	.652
Stress by emotion regulation	−.0004	−.004	.009	.963	.024	.204	.010	.022**
Overall model	R^2 = .057; F(9, 139) = .870, p = .554				R^2 = .098; F(9, 139) = 1.575, p = .129			
IL-6, pg/ml; log								
Intercept	3.900		.041	<.001	3.888		.040	<.001
Chronic family stress	.014	.038	.033	.672	−.006	−.015	.034	.868
Emotion regulation	−.001	−.033	.004	.699	.005	.089	.005	.318
Stress by emotion regulation	−.004	−.063	.005	.458	.012	.164	.006	.062*
Overall model	R^2 = .094; F(9, 139) = 1.505, p = .153				R^2 = .167; F(9, 139) = 2.070, p = .037			
IL-8, pg/ml; log								
Intercept	3.611		.049	<.001	3.591		.048	<.001
Chronic family stress	.027	.062	.040	.490	.001	.002	.040	.980
Emotion regulation	−.002	−.039	.005	.658	.003	.055	.005	.533
Stress by emotion regulation	−.004	−.049	.007	.568	.020	.239	.007	.007**
Overall model	R^2 = .077; F(9, 139) = 1.206, p = .296				R^2 = .133; F(9, 139) = 2.207, p = .025			

IL-6 = Interleukin-6; CRP = C-reactive protein; SE = standard error.

All analyses controlled for age, sex, ethnicity, income, and BMI when considering inflammatory measures.

* $p < .10$.

** $p < .05$.

Interaction Effects of Chronic Stress and Emotion Regulation

Next, we considered whether emotion regulation moderated the association between chronic family stress and adolescent metabolic and inflammatory measures (Tables 2, 3). Multicollinearity statistics were examined, and there was no evidence of multicollinearity when testing interaction effects (for all models, variance inflation factor < 1.13 and tolerance > .88).

Metabolic Markers

Cognitive reappraisal moderated the association between chronic family stress and SBP (B = −.303, SE = .143, p = .035), WHR (B = −.003, SE = .001, p = .015), and marginally DBP (B = −.258, SE = .135, p = .057), such that as chronic family stress increased, adolescents who reported using more cognitive reappraisal showed evidence of lower SBP, smaller WHR, and marginally lower DBP (Figure 1). Suppression did not moderate the association between chronic family stress and metabolic measures of SBP, DBP, and WHR (all p's > .50).

Inflammatory Markers

Basal Inflammation

Neither cognitive reappraisal nor suppression moderated the association between chronic family stress and CRP (both p's > .10) or chronic stress and IL-6 (both p's > .20).

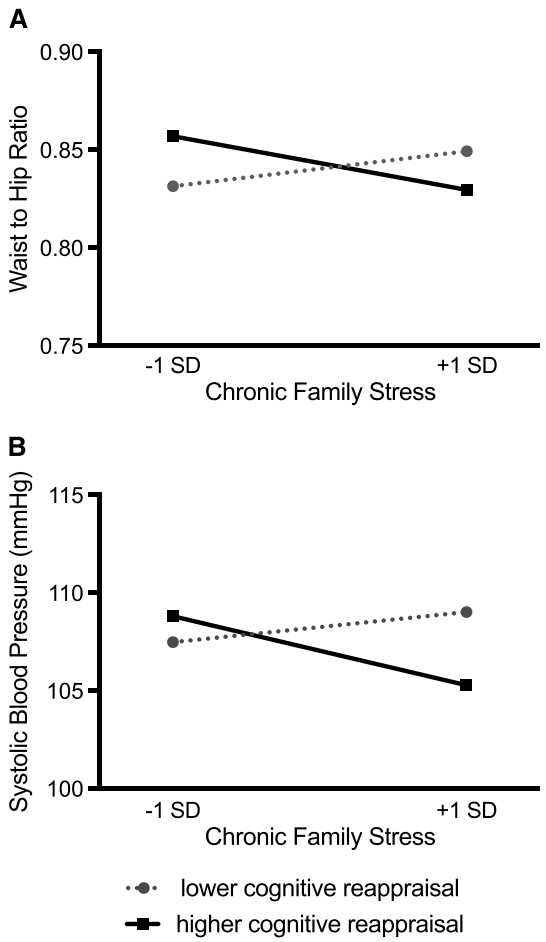

FIGURE 1. Cognitive reappraisal moderates the association between chronic family stress and waist-to-hip ratio (A, B = −0.003, SE = 0.001, p = .015) and chronic family stress and systolic blood pressure (B, B = −0.303, SE = 0.143, p = .035). Chronic family stress and cognitive reappraisal are depicted at ±1 SD.

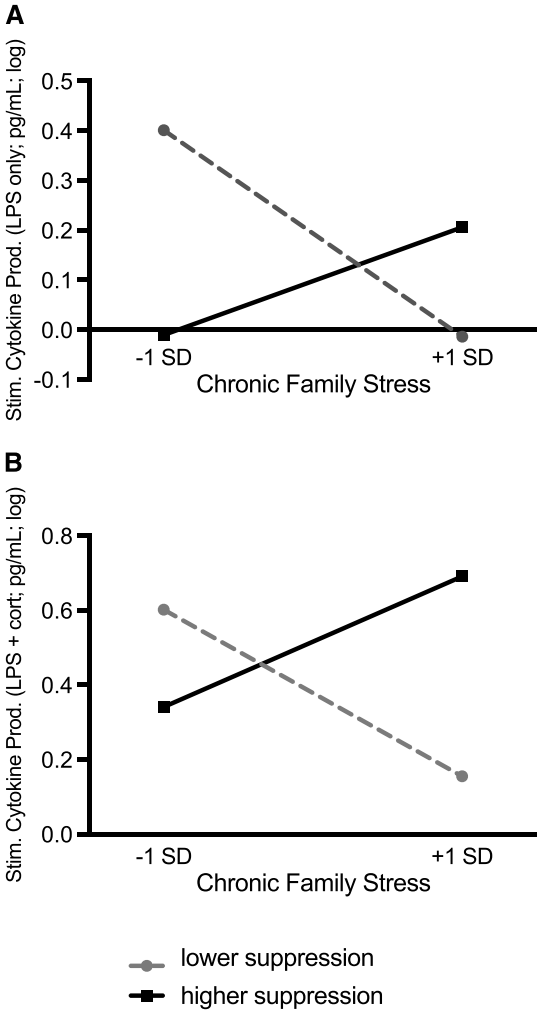

FIGURE 2. Suppression moderates the association between chronic family stress and stimulated proinflammatory cytokine production (A, B = .046, SE = .020, p = .021) and chronic family stress and glucocorticoid sensitivity (B, B = .051, SE = .021, p = .015), as indicated by greater proinflammatory stimulated cytokine production in the presence of hydrocortisone. As chronic family stress increased, adolescents higher in suppression showed greater stimulated proinflammatory cytokine production and reduced glucocorticoid sensitivity. Chronic family stress and suppression are depicted at ±1 SD.

Stimulated Cytokine Production

Suppression moderated the association between chronic family stress and stimulated proinflammatory cytokine production (B = .046, SE = .020, p = .021) (Figure 2), such that as chronic family stress increased, adolescents who reported using more suppression showed evidence of greater stimulated proinflammatory cytokine production. Cognitive reappraisal did not moderate the association between chronic family stress and stimulated proinflammatory cytokine production (p > .30).

Glucocorticoid Sensitivity

Suppression moderated the association between chronic family stress and glucocorticoid sensitivity (B = .051, SE = .021, p = .015) (Figure 2), such that greater chronic family stress

was associated with lower glucocorticoid sensitivity (greater stimulated proinflammatory cytokine production in the presence of hydrocortisone) among adolescents who reported using more suppression. Cognitive reappraisal did not moderate the association between chronic family stress and glucocorticoid sensitivity ($p > .50$).

DISCUSSION

Previous studies have separately addressed the influence of emotion regulation strategies or chronic stress on measures of physiological health, but few have considered them together (20). To our knowledge, this study is the first to assess the moderating effects of emotion regulation on metabolic and immune functioning in response to chronic family stress exposure among adolescents. Although we did not find main effects of either chronic family stress or emotion regulation on physiological risk markers, emotion regulation strategies did moderate the extent to which chronic family stress was associated with metabolic and inflammatory measures. These results suggest that as adolescents are exposed to increasing levels of chronic family stress, the type of emotion regulation strategy they use becomes increasingly important. Generally speaking, we found that in the context of greater chronic family stress exposure, greater use of cognitive reappraisal was associated with improved metabolic measures, whereas greater use of suppression was associated with increased stimulated proinflammatory cytokine production and decreased glucocorticoid sensitivity.

As hypothesized, as chronic family stress increased, adolescents who reported using higher levels of cognitive reappraisal had smaller WHR, lower SBP, and marginally lower DBP compared with adolescents lower in cognitive reappraisal. Interestingly, although higher cognitive reappraisal seemed to be protective against adverse metabolic measures in the face of greater chronic family stress, it was not associated with inflammatory markers and glucocorticoid sensitivity. These findings align with existing research on the effect of cognitive reappraisal on metabolic measures; however, previous studies did not specifically address emotion regulation in the context of chronic stress and focused on adults (31,32,34).

Conversely, we found that adolescents who reported greater tendencies to use suppression in the face of ongoing family stressors showed evidence of greater stimulated (though not basal) proinflammatory cytokine production and reduced glucocorticoid sensitivity as compared with adolescents lower in suppression. The use of suppression did not moderate the association between chronic family stress and metabolic measures. These findings are partially supported by previous research linking suppression to elevated sympathetic and cardiovascular activation and greater levels of CRP, however, again, not within a chronic stress framework or among adolescents (27,34). Among adolescents exposed to lower levels of chronic family stress, proinflammatory cytokine production was greater for those who used suppression less frequently. This could perhaps reflect a person-environment fit in which suppression can be more or less physiologically adaptive depending on an individual's level of chronic stress exposure. Suppression may be appropriate in a lower chronic family stress environment because it would likely be applied less frequently and in response to less severe situations, such as a curt reply from an otherwise caring, empathic parent. Alternatively, relying on suppression as an emotion regulation strategy in a higher family stress environment may be problematic because it may be applied in response to more pervasive problems that are likely to persist for longer periods (e.g., suppressing frequently spiteful or rejecting comments). Within this sample, this was only observed for stimulated cytokine production, and these findings should be replicated before conclusions can be drawn. The divergent moderating effects of these emotion regulation strategies on metabolic and inflammatory measures in this adolescent sample are in part consistent with previous literature that suggests that use of cognitive reappraisal may be physiologically protective (28–32), whereas the use of suppression may come at a physiological cost (33). Specifically, greater use of suppression may be associated with heightened physiological arousal (28,33), which in turn may contribute to heightened inflammation (34).

Nonetheless, this does not explain why the use of cognitive reappraisal was associated with better metabolic measures, but not inflammatory markers and glucocorticoid sensitivity. These results should be considered preliminary and encourage future studies to assess whether this pattern of diverging physiological measures can be replicated. The observed moderation effects may perhaps be reflective of longstanding developmental processes, because emotion regulation strategies are shaped throughout childhood in response to experiences, challenges, or demands. Although emotion regulation can change throughout the lifespan, early childhood is a critical period for developing these strategies because skills are reinforced or discouraged based on feedback from interactions with the environment and others (19). Unfortunately, emotion regulation strategies from early childhood and the "goodness of fit" between these strategies and the childhood environment could not be assessed within this study but would be advantageous to consider for future studies. Similarly, youth may experience more chronic family stress as a consequence of having poor emotion regulation, or residing in stressful family environments may produce certain emotion regulation strategies. Thus, our results do not allow us to infer whether or not adolescents' emotion regulation strategies are independent of the family environments in which they live or to what extent.

There were also no main effects of chronic family stress or emotion regulation on adolescent physiological measures, which aligns with some existing studies that have reported moderation of the chronic stress–health association in the absence of significant main effects (e.g., (10,49)). This underscores that chronic stress exposure alone does not necessarily in and of itself lead to detrimental health, but rather that individuals' environments as well as their perceptions of and reactions to chronic stress are both important considerations. Participating families largely scored in the low-moderate chronic stress range, thereby limiting the generalizability of these findings. Emotion regulation may take different forms and serve a qualitatively different purpose for adolescents from home environments marked by substantially greater levels of chronic stress. For example, certain emotion regulation strategies (such as hypervigilance) may be adaptive in the short term for youth who experience family stress but lead to adverse long-term consequences if the stressors are pervasive and chronic (50). The lack of significant main effects for the association between chronic family stress and physiological measures could also be attributed to the distribution of chronic family stress within the sample, because most adolescents fell within the range of low to

moderate chronic family stress. Future studies should consider including more adolescents exposed to higher chronic family stress environments.

This study has several strengths. First, we focused on adolescents, a group that may be particularly vulnerable to the physiological consequences of chronic stress with potential implications for the longer-term health (17). Second, we considered a range of relevant health measures, including metabolic and inflammatory markers, as well as glucocorticoid sensitivity. Third, we considered two different types of emotion regulation strategies (cognitive reappraisal and suppression) that have previously been shown to be relevant to measures of physiological health (19,25,28,34).

Despite these strengths, there are some limitations to our study. The cross-sectional design does not allow us to infer causality. Furthermore, it is unclear whether the differences observed here would, over time, translate into clinically meaningful changes in measures of health among adolescents. Moving forward, a within-person design would allow for observations of whether and how the interaction between chronic family stress and certain emotion regulation strategies during adolescence influence physiological measures over time. It may also be advantageous to assess the acute stress responses in adolescents exposed to chronic family stress to consider more immediate metabolic and inflammatory responses to stressors as modulated by various emotion regulation strategies and the contexts in which such strategies are used. Finally, the present study considered adolescents' self-reports of general tendencies toward using different emotion regulation strategies; it is unclear to what extent these self-reports accurately reflect such strategies in response to specific situations and in different contexts. Future studies could incorporate ecological momentary assessment to assess whether use of emotion regulation strategies changes depending on the context and how this influences overall physiological functioning. Although the additional analyses to investigate the impact of chronic family stress and emotion regulation on individual proinflammatory cytokines increased the potential for type 1 error findings, results from these analyses, although weaker, were largely in line with results obtained when considering the inflammatory cytokine composites, supporting our overall conclusions.

These findings suggest that the type of emotion regulation strategies adolescents use may inhibit or exacerbate adverse physiological measures associated with chronic stress. Furthermore, the choice of strategy seems especially important for adolescents experiencing higher levels of chronic family stress, perhaps because low life stress may not require frequent use of such strategies. Adolescents repeatedly drawing on emotion regulation strategies in response to chronic family stress may be more susceptible to both the protective and adverse physiological repercussions. Although the results demonstrate differences in physiological measures that fall within healthy ranges, these small changes could accumulate overtime and contribute to poorer health in adulthood. In tandem with future longitudinal work, this study may encourage interventions that emphasize a greater focus on cognitive reappraisal strategies, rather than suppression, among groups of high-risk adolescents exposed to high levels of chronic stress.

Source of Funding and Conflicts of Interest: E.C. received funding from the Canadian Institutes of Health Research Grant FRN 97872. The authors report no conflicts of interest.

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