

# Familism and Inflammatory Processes in African American, Latino, and White Youth

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**Objective:** African Americans and Latinos make up the two largest minority groups in the United States, and compared with Whites, these ethnic minority groups face disproportionate risk for certain physical health problems. However, factors that may protect these groups against early risk for poor health are not entirely understood. Familism, which emphasizes family interdependence and commitment, and is more prevalent among Latino and African American families, may be one such factor. The current study examined whether values and behaviors related to familism were differentially associated with inflammatory processes among White, African American, and Latino youth. **Method:** Participants were 257 youth who completed measures of familism values and behaviors and whose parents reported on their ethnicity. Participants also provided blood samples for the assessment of proinflammatory cytokine responses to bacterial challenge and of sensitivity to anti-inflammatory features of cortisol and interleukin (IL)-10. **Results:** Significant familism values and behaviors by ethnicity interactions were observed. For Latino and African American youth but not for White youth, more familism values were associated with greater sensitivity to IL-10. Additionally, for African American youth, more familism behaviors were associated with decreased cytokine responses to bacterial challenge and greater sensitivity to cortisol and IL-10. By contrast, familism behaviors were associated with lower sensitivity to cortisol in White youth and were not associated with any inflammatory outcomes in Latino youth. **Conclusion:** This pattern of findings suggests that for African American youth and to some extent for Latino youth, familism values and behaviors may be protective against the elevated risk for poor health they face.

**Keywords:** family, inflammation, ethnicity, adolescence, health disparities

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African Americans and Latinos comprise 13.3% and 17.8% of the U.S. population, respectively, making them the two largest ethnic minority groups in America (U.S. Census Bureau, 2017). Importantly, compared with the White majority, they face differential risk for physical health problems. For instance, rates of obesity and diabetes are elevated in African Americans and Latinos relative to Whites (Carnethon et al., 2017; Rodriguez et al., 2014). African Americans are also at greater risk for hypertension, cardiovascular diseases, and most cancers (Carnethon et al., 2017; DeSantis et al., 2016), and Latinos face greater risk for certain cancers, including those of the liver, stomach, and cervix (Siegel et

al., 2015) compared with Whites. Despite such disparities, African Americans and Latinos remain largely understudied in health-related research. Consequently, little is understood about culture-specific risk factors that may contribute to these ethnic health disparities and protective factors that may help reduce them. Investigating this question during adolescence may be particularly important because sensitivity to context may increase during the teenage years (Blakemore & Mills, 2014; Somerville, 2013) and because many chronic conditions begin to develop in the early decades of life (Shonkoff, Boyce, & McEwen, 2009). For instance, preclinical signs of cardiovascular disease, such as excess adiposity, raised blood pressure, and early-stage atherosclerotic plaque can be observed in some adolescents (Berenson et al., 1998; May, Kuklina, & Yoon, 2012). The current study examined the link between familism, a cultural value emphasizing family interdependence, and inflammatory processes involved with chronic disease in a sample of African American, Latino, and White adolescents.

## Familism Values and Links to Health

Familism refers to a constellation of values prioritizing family obligations, family as a first source of support, interconnectedness and unity among family members, and a willingness to defer to family preferences over individual preferences (Sabogal, Marín, Otero-Sabogal, Marín, & Perez-Stable, 1987). The construct of familism was initially developed to describe differences in Latino

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and White families in the United States; however, communalism, which has been primarily studied in African Americans, similarly emphasizes family relationships and interdependence, and prioritizes group duties and responsibilities over individual preferences (Boykin, Jagers, Ellison, & Albury, 1997). In fact, familism and communalism have been shown to map onto a single underlying family primacy construct (Schwartz et al., 2010), and both African Americans and Latinos report higher levels of familism values and engaging in familism-related behaviors (e.g., helping and spending time with family) more frequently than Whites (Fuligni, Tseng, & Lam, 1999; Hooper, Wallace, Doehler, & Dantzler, 2012; Khafi, Yates, & Luthar, 2014; Telzer & Fuligni, 2009).

Given the emphasis of interdependence and family relationships in Latino and African American culture, familism and other similar collectivist-oriented constructs are generally thought to contribute to favorable outcomes among Latino and African American youth. Indeed, familism values have been associated with lower levels of internalizing symptoms and substance use, and higher levels of psychological well-being in Latino youth (Fuligni & Pedersen, 2002; Telzer, Gonzales, & Fuligni, 2014; Telzer, Tsai, Gonzales, & Fuligni, 2015). Similarly, communalism and beliefs in family duty have been related to greater empathy, and lower levels of violent behavior, delinquency, and alcohol use among African American youth (Gorman-Smith, Tolan, Zelli, & Huesmann, 1996; Humphries & Jagers, 2009; Nasim, Belgrave, Jagers, Wilson, & Owens, 2007). Whether these findings extend to youths' physical health outcomes is unclear, but two studies of adults begin to suggest that they do. In one study, communalism was associated with lower blood pressure in African American women but not in White women (Abdou et al., 2010). In the second study, familism was related to fewer self-reported physical health symptoms, though this link did not vary by ethnicity (Corona, Campos, & Chen, 2017).

### Familism Behaviors and Links to Health

Whereas familism values refer to internal psychological beliefs about family functioning and relationships, familism behaviors refer to the concrete acts through which familism values may manifest (Sabogal et al., 1987). Common familism-related behaviors include spending time with family members and assisting the family with tasks such as caring for siblings and performing household chores. Although these behaviors are part of the everyday lives of adolescents regardless of ethnicity, adolescents from interdependent cultures including those of Latin American and African American backgrounds engage in these behaviors more frequently than their White peers (Jurkovic, Thirkield, & Morrell, 2001; Telzer & Fuligni, 2009).

Familism behaviors have been linked to both positive and negative outcomes among youth, including greater feelings of positive affect and stress, as well as improved interpersonal outcomes and greater substance use (East, 2010; Kuperminc, Wilkins, Jurkovic, & Perilla, 2013; Telzer & Fuligni, 2009; Telzer et al., 2014). These seemingly contradictory findings may be due to other moderating factors. For instance, one study of older adolescents found that family assistance was associated with higher levels of inflammation; however, adolescents who derived greater meaning from helping their families had lower levels of inflammatory biomarkers than their peers who derived little or no meaning (Fuligni et al.,

2009). A more recent study similarly found that providing tangible assistance to and spending time with the family more frequently were associated with worse asthma outcomes in youth, but only among those from disadvantaged backgrounds (Lam et al., 2018). In addition to socioeconomic context and meaning attached to familism-related behaviors, ethnicity may also be an important moderator. Given that familism is valued to a greater extent in African American and Latino families, engaging in familism behaviors may be beneficial for these ethnic minority youth but not for Whites. Indeed, previous work has linked familism-related behaviors to better psychosocial outcomes in African American youth and worse outcomes in White youth (Khafi et al., 2014). Whether these findings extend to physical health-related outcomes has not been examined.

### Present Study

The above studies suggest some connection between familism values and behaviors and youths' physical health. However, much of the evidence comes from studies focusing on psychosocial and behavioral outcomes. Studies examining physical health-related outcomes have focused on familism-related behaviors. Thus, whether endorsing familism values contributes to youths' physical health and whether it differs from the effects of actually engaging in familism-related behaviors are currently unknown. Furthermore, past studies have not adequately probed the role of familism in the health of African American youth. Yet, as noted earlier, communalism largely overlaps with familism, and African Americans have been shown to endorse higher levels of familism values and engage in more familism behaviors than Whites. As such, familism may also be relevant to their health.

The current study begins to address these questions by investigating the associations between familism values and behaviors and inflammatory processes, and determining whether these associations vary by ethnicity in a sample of eighth-graders from African American, Latino, and White backgrounds. We focused on inflammatory processes given that inflammation is thought to be a key pathway through which the early family environment affects later physical health (Miller, Chen, & Parker, 2011). In the face of invading pathogens or injury, the immune system mounts an inflammatory response, during which monocytes, dendritic cells, and macrophages secrete proinflammatory cytokines (IL-1 $\beta$ , IL-6, TNF- $\alpha$ ). These proinflammatory cytokines have a range of functions that facilitate an effective immune response to injury and infection, including attracting other immune cells to the site of infection or injury and activating their functions related to eliminating threats and repairing wounds. As such, the inflammatory response is critical for survival and healing. However, if unregulated and sustained for prolonged periods of time, inflammation can damage tissue and organs in ways that increase risk for a range of chronic diseases, including cardiovascular disease and certain cancers (Nathan & Ding, 2010). Thus, the body has anti-inflammatory mechanisms that regulate the inflammatory response. Two key mechanisms involve cortisol from the hypothalamic-pituitary-adrenocortical (HPA) axis, and IL-10, a predominantly anti-inflammatory cytokine. When cortisol and IL-10 bind to their corresponding receptors, immune cell release of proinflammatory cytokines declines.

To model this process, we cultured youths' immune cells with a common bacterial product, lipopolysaccharide (LPS), and then measured the amount of inflammatory cytokines those cells produced. We also simultaneously cultured the cells with glucocorticoids or IL-10 to assess their sensitivity to anti-inflammatory signals. We focused on these outcomes for conceptual and methodological reasons. Conceptually, these assays simulate how aggressively monocytes and macrophages respond to bacterial stimuli *in vivo*, and how effectively anti-inflammatory signals regulate that process. Accordingly, this approach provides mechanistic insights that cannot be gleaned from the circulating inflammatory biomarkers (e.g., IL-6 and TNF- $\alpha$ ) used in most previous research. Those molecules are released by multiple tissues, including immune, endothelial, skeletal, and adipose cells, so neither their cellular origin nor their triggering stimulus can be discerned. This *ex vivo* approach is also methodologically advantageous because youth typically have quite low concentrations of circulating inflammatory biomarkers, making their accurate measurement a challenge. Based on previous work focusing on psychosocial and behavioral outcomes, we hypothesized that both familism values and behaviors would be related to a more optimal inflammatory phenotype, characterized by a smaller inflammatory response to LPS and higher sensitivity to anti-inflammatory signals, particularly among ethnic minority youth. Because a number of biomedical and psychological factors, including body mass index (BMI), depression, family stress, household structure, and social support, have established links with inflammation, we also considered whether any observed effects were above and beyond these factors.

## Method

### Participants and Procedures

Participants were 277 eighth grade students ( $M_{age} = 13.92$ ,  $SD = .54$ ) from diverse ethnic backgrounds: 27.4% identified as White, 38.3% as African American, 28.5% as Latino, and 5.8% as Other (Asian, Native Hawaiian/Pacific Islander, and/or American Indian/Alaskan native). Given our interest in potential ethnic differences between Whites, African Americans, and Latinos, and that the number in the Other ethnic minority group was insufficient to make meaningful comparisons, we excluded the 16 adolescents in the Other ethnic group. Four additional youths were excluded

due to missing information on study variables, leaving a final analytic sample of 257. Sample characteristics are displayed in Table 1.

Participants were recruited from the greater Chicago area via advertisements posted in local media and transit stations, announcements at schools and community events, and a direct mail campaign. Interested youth and a parent were screened and were eligible to participate if they were in the eighth grade, had no history of chronic medical or psychiatric illness, were free of infectious diseases during the past two weeks, were not taking any prescription medication during the prior three months, and were not currently pregnant. Eligible participants were scheduled for a morning laboratory visit and were instructed to arrive at the session having fasted overnight.

Upon arrival, youth written assent and parent written consent were obtained. Next, trained personnel collected blood samples from youth via antecubital venipuncture. Youth then provided anthropometric measures and completed a number of questionnaires, interviews, and behavioral tasks while their caregivers provided information on socioeconomic background and family health history via interviews conducted by study staff. All study procedures were approved by the Northwestern University Institutional Review Board.

### Measures

**Familism values.** Values of familism were assessed using 11 items from the Attitudinal Familism Scale (Lugo Steidel & Contreras, 2003) with several items revised to make them appropriate for youth. Items assessed two central dimensions of familism: familial support and subjugation of self for family. Using a 10-point scale (1 = *strongly disagree*, 10 = *strongly agree*), adolescents indicated the extent to which they agreed with items such as "Children should regularly help their parents with young brothers and sisters, for example, help them with homework, help them taking care of them, and so forth", and "A person should always support members of the extended family, for example, aunts, uncles, and in-laws, if they are in need even if it is a big sacrifice." Responses were averaged across items to compute an overall familism values score ( $\alpha = .84$ ).

**Familism behaviors.** A modified scale developed in prior research (Fulgini et al., 1999) was used to assess how often youth

Table 1  
Sample Characteristics and Descriptive Statistics

Characteristic	African-American ( <i>n</i> = 105)	Latino ( <i>n</i> = 77)	White ( <i>n</i> = 75)	Statistical test
	<i>M</i> ( <i>SD</i> )	<i>n</i> (%)	<i>n</i> (%)	
Age	13.94 (.58)	13.99 (.50)	13.80 (.53)	$F(2, 254) = 2.65, p = .073$
Gender: Female, <i>n</i> (%)	66 (62.90)	48 (62.30)	47 (62.70)	$\chi^2 = .005, p = .997$
Puberty status	3.82 (.81)	3.70 (.80)	3.60 (.72)	$F(2, 254) = 1.77, p = .173$
Family income	4.25 (1.92)	4.62 (1.68)	7.19 (1.65)	$F(2, 254) = 65.89, p < .001$
Familism values	6.92 (1.56)	7.09 (1.42)	6.38 (1.46)	$F(2, 254) = 4.80, p = .009$
Familism behaviors	3.59 (.66)	3.53 (.68)	3.42 (.57)	$F(2, 254) = 1.64, p = .195$
LPS stimulated cytokine production	.18 (.64)	-.24 (.58)	.002 (.65)	$F(2, 254) = 10.30, p < .001$
Cortisol sensitivity	-.15 (.62)	.09 (.47)	.12 (.66)	$F(2, 254) = 5.96, p = .003$
IL-10 sensitivity	-.12 (.58)	.09 (.43)	.08 (.56)	$F(2, 254) = 4.53, p = .012$

engage in familism behaviors. Using a five-point scale (1 = *almost never*, 5 = *almost always*), adolescents reported how frequently they provide tangible assistance to and spend time with their families. Example items include how often do you “run errands that the family needs done” and “spend time at home with your family.” Responses were averaged across items to compute an overall familism behavior score ( $\alpha = .76$ ).

**Ethnicity.** Participants’ parents indicated whether participants were White, African American, Latino, or Other (i.e., Asian, Native Hawaiian/Pacific Islander, and/or American Indian/Alaskan native), although as noted earlier, those in the Other category were excluded from analyses. Multiracial individuals who identified as African American and any of the other ethnicity categories were coded as African American. Any individuals who identified as Latino and any other ethnicity except African American were coded as Latino. Whites were coded as so only if they did not identify as any other ethnicity. From the ethnicity variable, two dummy variables were created with White as the reference group.

**Inflammatory parameters.** Ten mL of antecubital blood was collected into sodium heparin tubes following an overnight fast to assess two features of the inflammatory process: how large cells’ inflammatory response were to a common bacterial product and how sensitive they were to anti-inflammatory signals that normally regulate this process. Response to the bacterial product was assessed by culturing whole blood with LPS and quantifying production of a panel of proinflammatory cytokines, which included IL-1 $\beta$ , IL-6, IL-8, and TNF- $\alpha$ . Sensitivity to anti-inflammatory signals was assessed by coincubating the whole blood with LPS and molecules that provide local (IL-10) or systemic (cortisol) anti-inflammatory feedback. Production of the panel of cytokines noted above was then quantified.

Whole blood was diluted to a 9:1 ratio with R10 media, and then dispensed in 400  $\mu$ l aliquots into wells containing (a) LPS (50 ng/ml) alone, (b) LPS (50 ng/ml) and hydrocortisone at doses of either  $2.76 \times 10^{-6}$  or  $2.76 \times 10^{-7}$  M, and (c) LPS (50 ng/ml) and varying amounts of IL-10 ( $1.075 \times 10^{-9}$ ,  $5.376 \times 10^{-9}$ , or  $2.688 \times 10^{-8}$  M). The plate also included a negative control well, where cells were incubated with R10 media, to measure background cytokine release. After a 6-hr incubation period at 37 °C in 5% carbon dioxide, contents were transferred to microfuge tubes and centrifuged at 17,000g for five minutes. Supernatants were harvested and frozen at -80 °C until the study was completed, at which point they were assayed for IL-1 $\beta$ , IL-6, IL-8, and TNF- $\alpha$ . Assays were performed in duplicate using Human Proinflammatory II Tissue Culture kits on a MSD Sector Imager 2400 (all from Meso Scale Diagnostics, Rockville, MD). Lower limits of detection ranged from .455 to 2.255 pg/ml. Average intraassay coefficients of variation for IL-1 $\beta$ , IL-6, IL-8, and TNF- $\alpha$  were 2.76%, 4.20%, 2.78%, and 3.30%, respectively.

Because we did not have a priori hypotheses about specific cytokines and wanted to minimize the number of outcome variables and attendant risks of Type I error, we computed three composite variables from the assay data, which has also been done in previous research assessing a panel of inflammatory cytokines in both adults and youth (Hostinar, Lachman, Mroczek, Seeman, & Miller, 2015; Schreier, Roy, Frimer, & Chen, 2014). The first composite reflected the magnitude of the inflammatory cytokine response to LPS, operationalized as the average of standardized IL-1 $\beta$ , IL-6, IL-8, and TNF- $\alpha$  values in the LPS-only well (after

background production of these molecules, as measured in the negative control well, had been subtracted out). Thus, higher values on the composite indicate a larger cytokine response. Principal components analysis indicated that a single component for the four cytokines explained 43.8% of the variance.

The next two composites reflected how sensitive the cytokine response was to regulation by cortisol and IL-10. These outcomes were operationalized as the slope of the regression line in models where cytokine values were regressed upon inhibitor dose. Multi-level modeling was used to accommodate the nested structure of these data; that is, repeated assessments of cytokines (Level 1) within each participant (Level 2):

Level 1:

$$\text{Cytokine}_{ij} = \beta_{0i} + \beta_{1i}(\text{cortisol/IL-10 dose})_{ij} + e_{ij}$$

Level 2:

$$\beta_{0i} = \gamma_{00} + u_{0i}$$

$$\beta_{1i} = \gamma_{10} + u_{1i}$$

Level 1 of the models includes parameters representing the level of a given cytokine produced in response to LPS ( $\beta_{0i}$ ) and the link between the increasing dose of the inhibitor (cortisol or IL-10) and the amount of cytokine production ( $\beta_{1i}$ ). More technically,  $\beta_{1i}$  reflects the difference in cytokine production between wells with LPS only and wells with a low, moderate, or high dose of inhibitor. Level 1 also includes a residual parameter ( $e_{ij}$ ). Level 2 of the models includes parameters representing the sample’s average levels of each cytokine ( $\gamma_{00}$ ) and of cortisol or IL-10 sensitivity ( $\gamma_{10}$ ) across doses. It also contains the variance parameters  $u_{0i}$  and  $u_{1i}$ , which reflect the extent of each individual’s deviation from these sample-wide averages. Sensitivity to anti-inflammatory signaling for each individual was computed by summing his or her estimated  $u_{1i}$  value and the sample fixed effect for sensitivity to anti-inflammatory agents ( $\gamma_{10}$ ). Separate slopes were estimated for each of the four cytokines, standardized, and then averaged to form a composite. Principal components analysis indicated that a single component for the four slopes computed for each cytokine explained average 43.7% and 50.9% of the variance for sensitivity to cortisol and IL-10, respectively. Composite variables were then multiplied by negative one, so that higher values indicate greater sensitivity to inhibition (i.e., larger decreases in cytokine production in response to increasing doses of cortisol or IL-10), and therefore a more tightly regulated inflammatory response.

**Covariates.** Age, gender, household income, and pubertal status were included as covariates, as these factors have been shown to influence inflammatory processes (O’Connor et al., 2009; Straub, 2007; Trigunaite, Dimo, & Jørgensen, 2015). Participants reported their date of birth, from which age was computed, and gender (0 = male, 1 = female). On a nine-point scale (1 = less than \$5,000, 9 = \$200,000 and higher), parents reported total household income from wages, government benefits, and subsidies in the past year. Pubertal status was assessed using the Pubertal Development Scale (Petersen, Crockett, Richards, & Boxer, 1988) which includes sex-specific items assessing body hair, facial hair, deepening of voice, breast growth and menstruation. Scores ranged from 1 (prepubertal) to 5 (post-pubertal). Sensitivity analyses included additional covariates (BMI, depressive symptoms, family stress, parent marital status, social support) for which detailed information can be found in the [online supplemental material](#).

## Statistical Analyses

A series of hierarchical regression analyses were conducted in Stata 14 to test whether the relations between familism values and behaviors and inflammatory processes differed between White and ethnic minority youth. Inflammatory parameters (i.e., cytokine production in response to LPS alone, to LPS and IL-10, and to LPS and cortisol) were predicted from covariates, familism, and ethnicity in the first step, and familism by ethnicity interaction terms added in the second step. Significant interaction effects were followed with tests of simple slopes for each ethnic group. All continuous variables were mean-centered, and separate models were estimated for familism values and behaviors and for each inflammatory outcome.

## Results

Descriptive statistics of study variables are presented in Table 1 and bivariate correlations among study variables are reported in the online supplement (Supplemental Table 1). Overall, youth reported moderate levels of familism values and engagement in familism behaviors. There were significant ethnic differences in familism values, but not familism behaviors. White youth endorsed lower levels of familism values than their African American,  $t(178) = -2.35, p = .020, d = -.36$ , and Latino peers,  $t(150) = -3.05, p = .003, d = -.50$ . Familism values did not differ between African American and Latino youth,  $t(180) = -.76, p = .446, d = -.11$ .

There were also ethnic differences in inflammatory outcomes. African American youth had higher inflammatory cytokine release in response to LPS compared with White youth,  $t(178) = -1.86, p = .032, d = -.28$ , and with Latino youth,  $t(180) = 4.62, p < .001, d = .69$ . Compared with Whites, Latinos had less inflammatory cytokine release in response to LPS,  $t(150) = 2.45, p = .02, d = .40$ . With respect to sensitivity to anti-inflammatory signals, African American youth were less sensitive to the anti-inflammatory properties of both cortisol and IL-10 compared with White youth (cortisol:

$t[178] = 2.86, p = .005, d = .43$ ; IL-10:  $t[178] = 2.28, p = .024, d = .34$ ) and with Latino youth (cortisol:  $t[180] = -2.87, p = .005, d = -.43$ ; IL-10:  $t[180] = -2.67, p = .008, d = -.40$ ). There were no differences between White youth and Latino for sensitivity to both cortisol,  $t(150) = .35, p = .729, d = .06$ , and IL-10,  $t(150) = -.16, p = .872, d = -.03$ .

With respect to covariates, household income differed by ethnicity. Specifically, White youth came from households with higher income compared with African American,  $t(178) = 10.68, p < .001, d = 1.62$ , and Latino youth,  $t(150) = 9.49, p < .001, d = 1.54$ . Household income did not differ between African American and Latino youth,  $t(180) = -1.34, p = .182, d = -.20$ . Furthermore, neither age nor the gender distribution differed by ethnic group.

## Familism Values

**Inflammatory cytokine response to LPS.** As Table 2 shows, familism values were not directly related to inflammatory cytokine production in response to LPS stimulation ( $p = .996$ ), and there was no evidence this association was moderated by ethnic group ( $ps > .299$ ).

**Sensitivity to anti-inflammatory signaling.** Table 2 also shows that familism values were not directly related to sensitivity to anti-inflammatory signals from cortisol ( $p = .967$ ) or IL-10 ( $p = .660$ ). For sensitivity to cortisol, there was no evidence of moderation by ethnic group ( $ps > .140$ ). However, for IL-10, familism values interacted with Latino ethnicity ( $p = .015$ ) and African American ethnicity ( $p = .036$ ). As depicted in Figure 1a, familism values were associated with higher sensitivity to IL-10 in Latino youth ( $b = .11, SE = .05, p = .020$ ), but not White youth ( $b = -.03, SE = .03, p = .225$ ). As depicted in Figure 1b, familism values were also associated with higher IL-10 sensitivity in African American youth ( $b = .08, SE = .04, p = .044$ ) but not in White youth ( $b = -.04, SE = .03, p = .251$ ).

Table 2  
Results of Models Predicting Inflammatory Cytokine Production From Familism Values, Ethnicity, and Their Interactions

Variable	LPS-stimulated		Cortisol sensitivity		IL-10 sensitivity	
	Step 1 <i>b</i> (SE)	Step 2 <i>b</i> (SE)	Step 1 <i>b</i> (SE)	Step 2 <i>b</i> (SE)	Step 1 <i>b</i> (SE)	Step 2 <i>b</i> (SE)
Intercept	.001 (.11)	.02 (.11)	-.04 (.10)	-.06 (.10)	-.04 (.09)	-.07 (.09)
Age	.03 (.08)	.03 (.08)	-.01 (.07)	-.02 (.07)	-.01 (.06)	-.04 (.06)
Gender	-.09 (.10)	-.09 (.10)	.17 (.10) <sup>‡</sup>	.15 (.10)	.10 (.09)	.07 (.09)
Puberty status	-.05 (.07)	-.06 (.07)	.05 (.06)	.05 (.06)	.10 (.06) <sup>‡</sup>	.11 (.05) <sup>*</sup>
Family income	.03 (.02)	.02 (.02)	.03 (.02)	.03 (.02)	.04 (.02) <sup>*</sup>	.04 (.02) <sup>*</sup>
African American	.27 (.12) <sup>*</sup>	.26 (.12) <sup>*</sup>	-.20 (.11) <sup>‡</sup>	-.17 (.11)	-.11 (.10)	-.07 (.10)
Latino	-.17 (.12)	-.20 (.12)	.04 (.11)	.07 (.11)	.10 (.10)	.13 (.10)
Familism values	-.0001 (.03)	.02 (.05)	.001 (.02)	-.06 (.05)	.01 (.02)	-.08 (.04) <sup>‡</sup>
African American × Familism values		-.07 (.06)		.09 (.06)		.11 (.05) <sup>*</sup>
Latino × Familism values		.03 (.07)		.08 (.07)		.15 (.06) <sup>*</sup>

Note. Gender was coded as 0 = male and 1 = female. Ethnicity was dummy coded with White as the reference group.

<sup>‡</sup>  $p < .056-.078$ . <sup>\*</sup>  $p < .05$ .

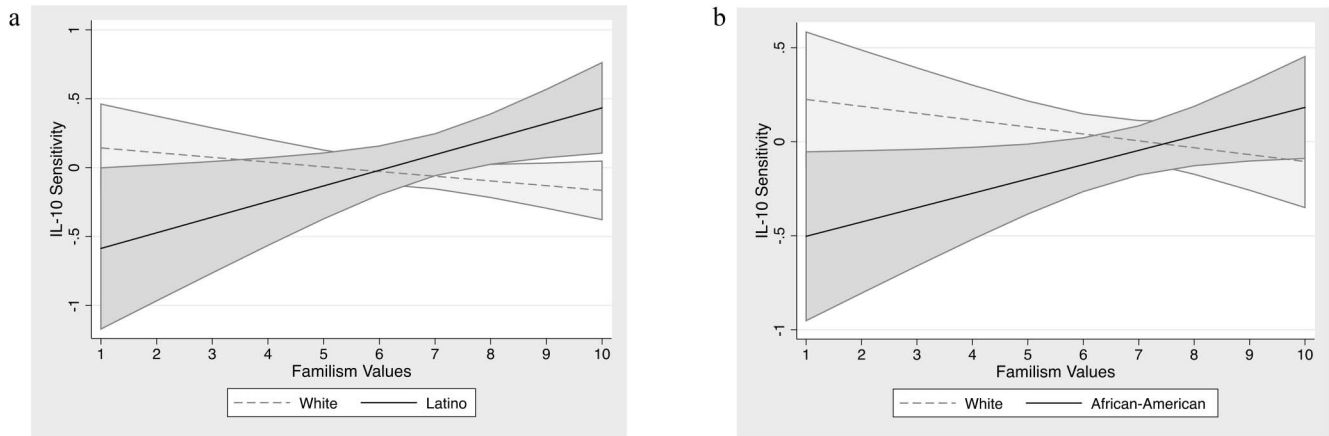


Figure 1. Ethnic differences in the link between familism values and IL-10 sensitivity for (a) Latino and (b) African American compared with White youth. Shaded areas reflect 95% confidence intervals.

**Familism Behaviors**

**Inflammatory cytokine response to LPS.** As shown in Table 3, familism behaviors were not directly related to inflammatory cytokine production in response to LPS ( $p = .458$ ). However, there was a significant interaction between familism behaviors and African American ethnicity ( $p = .012$ ). As depicted in Figure 2, endorsing more familism behaviors was associated with lower cytokine LPS-evoked production among African American youth ( $b = -.26, SE = .11, p = .013$ ), but not among White youth ( $b = .13, SE = .10, p = .157$ ).

**Sensitivity to anti-inflammatory signaling.** Table 3 also shows that familism behaviors were not directly related to sensitivity to anti-inflammatory signals from cortisol ( $p = .977$ ) or IL-10 ( $p = .199$ ). However, there was a significant interaction between familism behaviors and African American ethnicity for cortisol sensitivity ( $p = .021$ ). As depicted in Figure 3a, endorsing more familism behaviors was marginally associated with higher

sensitivity to cortisol’s anti-inflammatory properties among African American youth ( $b = .19, SE = .10, p = .061$ ), and marginally associated with lower cortisol sensitivity among White youth ( $b = -.16, SE = .09, p = .079$ ). There was also a marginally significant interaction between familism behaviors and African American ethnicity for IL-10 sensitivity ( $p = .082$ ; Table 3). As depicted in Figure 3b, more familism behaviors was associated with higher sensitivity to IL-10 among African American youth ( $b = .19, SE = .09, p = .035$ ), but not White youth ( $b = -.04, SE = .08, p = .583$ ).

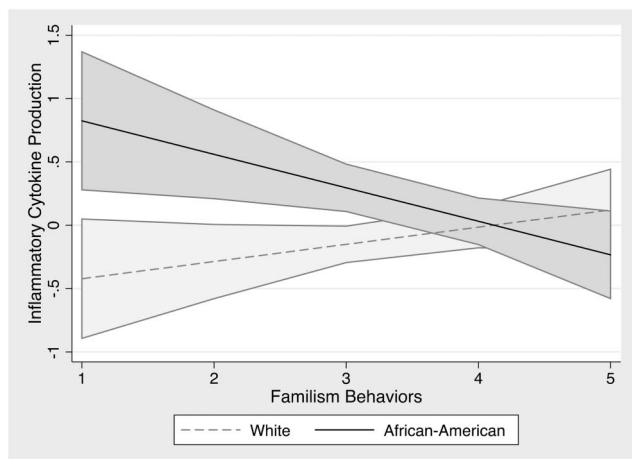
**Sensitivity Analyses**

We examined the robustness of our results above and beyond other factors known to vary by ethnicity or to be associated with inflammation. These included BMI, depressive symptoms, family stress, parent marital status (as a proxy for household structure), and social support. As reported in detail in the online supplemental

Table 3  
Results of Models Predicting Inflammatory Cytokine Production From Familism Behaviors, Ethnicity, and Their Interactions

Variable	LPS-stimulated		Cortisol sensitivity		IL-10 sensitivity	
	Step 1 <i>b</i> ( <i>SE</i> )	Step 2 <i>b</i> ( <i>SE</i> )	Step 1 <i>b</i> ( <i>SE</i> )	Step 2 <i>b</i> ( <i>SE</i> )	Step 1 <i>b</i> ( <i>SE</i> )	Step 2 <i>b</i> ( <i>SE</i> )
Intercept	-.01 (.11)	.06 (.11)	-.04 (.10)	-.09 (.10)	-.04 (.09)	-.07 (.09)
Age	.03 (.08)	.03 (.08)	-.01 (.07)	-.01 (.07)	-.01 (.06)	-.03 (.07)
Gender	-.08 (.10)	-.11 (.10)	.17 (.10) <sup>‡</sup>	.20 (.10) <sup>*</sup>	.09 (.09)	.10 (.09)
Puberty status	-.05 (.07)	-.06 (.07)	.05 (.06)	.04 (.06)	.11 (.06) <sup>*</sup>	.11 (.06) <sup>*</sup>
Family income	.03 (.02)	.02 (.02)	.03 (.02)	.04 (.02)	.04 (.02) <sup>*</sup>	.04 (.02) <sup>*</sup>
African American	.28 (.12) <sup>*</sup>	.24 (.12) <sup>*</sup>	-.20 (.11) <sup>‡</sup>	-.16 (.11)	-.12 (.10)	-.09 (.10)
Latino	-.17 (.12)	-.21 (.12) <sup>‡</sup>	.04 (.11)	.08 (.11)	.10 (.10)	.13 (.10)
Familism behaviors	-.05 (.06)	.20 (.13)	-.002 (.06)	-.21 (.12)	.07 (.05)	-.11 (.11)
African American × Familism behaviors		-.40 (.16) <sup>*</sup>		.35 (.15) <sup>*</sup>		.23 (.13) <sup>‡</sup>
Latino × Familism behaviors		-.20 (.17)		.17 (.16)		.24 (.14)

Note. Gender was coded as 0 = male and 1 = female. Ethnicity was dummy coded with White as the reference group.  
<sup>‡</sup>  $p < .068-.082$ . \*  $p < .05$ .



**Figure 2.** Ethnic differences in the link between familism behaviors and LPS stimulated inflammatory cytokine production in African American versus White youth. Cytokine production reflects a composite score of IL-1 $\beta$ , IL-6, IL-8, and TNF- $\alpha$ . Shaded areas reflect 95% confidence intervals.

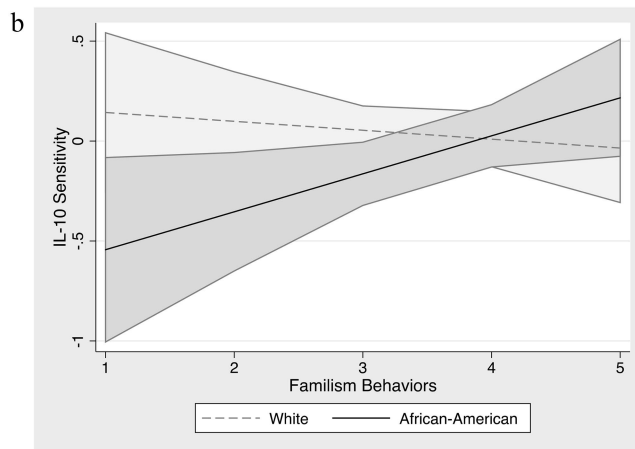
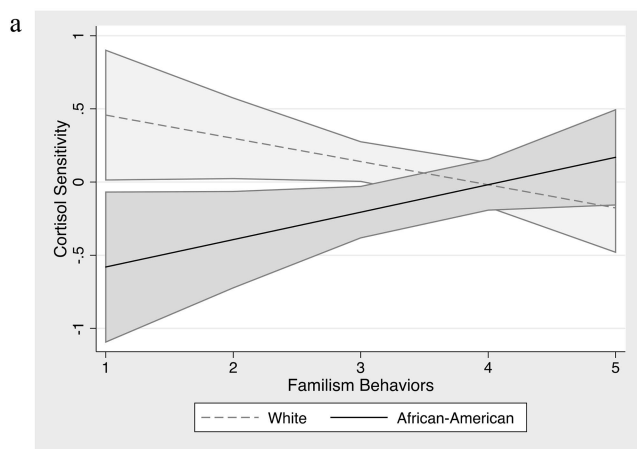
**material**, when these variables were added as additional covariates, results changed little overall and improved for the interaction effect between familism behaviors and African American ethnicity on IL-10 sensitivity.

### Discussion

The present study examined whether familism values and behaviors were each associated with inflammatory processes among African, Latino, and White adolescents. Compared with other groups, African American youth displayed a more proinflammatory profile, characterized by larger inflammatory cytokine responses to LPS and lower sensitivity to the anti-inflammatory properties of cortisol and IL-10. This inflammatory phenotype is thought to contribute to greater risk for chronic diseases such as heart disease and some cancers (Nathan & Ding, 2010), both for

which African Americans are at disproportionately greater risk (Carnethon et al., 2017; DeSantis et al., 2016). Although Latinos face greater risk for certain inflammatory-related conditions as well, in the present study they exhibited a less proinflammatory profile than Whites. Consistent with hypotheses, endorsing higher levels of familism values was associated with higher sensitivity to IL-10 regulation among African American and Latino youth, but seemed to confer no benefits for White youth. With respect to familism behaviors, African American youth, but not White or Latino youth, had lower LPS-evoked cytokine production if they reported engaging in familism behaviors more frequently. For African American youth, engaging in familism behaviors was also associated with higher sensitivity to regulation by cortisol and IL-10, indicative of a more tightly regulated cytokine response. By contrast, familism behaviors were associated with lower cortisol sensitivity and unassociated with IL-10 sensitivity in White youth. Notably, these patterns of results remained similar or became stronger in sensitivity analyses where we explored alternative explanations involving BMI, depressed mood, family stress, parents' marital status (as a proxy for household structure), and social support. Thus, our results support the hypothesis that familism values and behaviors confer unique health-related benefits for African American and Latino youth, above and beyond established psychosocial and biobehavioral factors.

Our results converge with prior work showing benefits of familism values in Latino youth and of communalism values in African American on several psychosocial and behavioral outcomes (e.g., Gorman-Smith et al., 1996; Telzer et al., 2015). They also converge with prior research indicating that familism behaviors can be both costly and beneficial, depending on context. Specifically, past studies have shown that whereas familism behaviors covary with worse asthma outcomes in low-SES youth (Lam et al., 2018), they relate to lower inflammatory biomarkers and better psychological health when the behaviors are viewed favorably (Fuligni et al., 2009; Hooper et al., 2012). Our findings provide further evidence that the impact of familism behaviors varies depending on individual and contextual factors and extend



**Figure 3.** Ethnic differences in the link between familism behaviors and sensitivity to (a) cortisol and (b) IL-10 in African American versus White youth. Shaded areas reflect 95% confidence intervals.

prior work by linking familism to inflammatory processes at the cellular level.

Endorsing familism values and behaviors may be beneficial for African American and Latino youth because doing so aligns with their family cultural context—generally speaking, African American and Latino youth are likely to be part of families that prioritize interdependence among family members and provision of instrumental support such as caring for siblings and older family members (Boykin et al., 1997; Coleman, Ganong, & Rothrauff, 2006; Nebbitt & Lombe, 2010; Schwartz et al., 2010). Supporting this notion, previous cross-cultural studies have shown that adherence to the norms and expectations of one's culture is associated with positive outcomes. For instance, in one study, endorsing independence in the United States where independence is emphasized was associated with a healthier diet whereas endorsing interdependence in Japan where collectivism is emphasized was associated with a healthier diet (Levine et al., 2016).

How might having family values consistent with those of one's family cultural context be beneficial for inflammatory processes? One possibility is that it minimizes parent-child conflict and fosters family connectedness. Research shows that when Latino youths' and their parents' cultural values and behaviors align, they have fewer conflicts and greater cohesion (Nair, Roche, & White, 2018). Those same relationship characteristics have been associated with lower inflammatory biomarker levels in other studies of youth (Ehrlich, Miller, & Chen, 2015; Miller, Brody, Yu, & Chen, 2014). These connections are probably mediated through the autonomic nervous system and HPA axis. Family climate affects the activity of both of these systems (Chiang, Taylor, & Bower, 2015; El-Sheikh & Erath, 2011; Repetti, Robles, & Reynolds, 2011), and their hormonal end products, glucocorticoids and catecholamines, modulate various aspects of the inflammatory response (Irwin & Cole, 2011).

Interestingly, familism values and behaviors were differentially associated with inflammatory outcomes in ethnic minority youth. Among African American youth, familism values were associated only with IL-10 sensitivity whereas familism behaviors were associated with LPS-evoked cytokine production, as well as with sensitivity to anti-inflammatory signaling from both cortisol and IL-10. Among Latino youth, familism values were associated with IL-10 sensitivity whereas familism behaviors were not associated with any of our inflammatory outcomes. These differential associations are consistent with the notion that values and behaviors do not always align (e.g., Bardi & Schwartz, 2003). It may be that some youth have high familism values but engage in the associated behaviors infrequently because of other competing yet equally valuable demands, such as academics, extracurricular activities, and spending time with peers. Conversely, some youth may not necessarily hold familism values but frequently engage in familism behaviors because of external constraints and family circumstances that may create a need for youth to assist and spend more time with their family—for example, a parent who is chronically ill or has to work long hours at multiple jobs. Indeed, in the current sample, familism values and behaviors were only moderately correlated.

That familism values were associated only with IL-10 sensitivity whereas familism behaviors were associated with all three inflammatory outcomes among African American youth raises the question of why familism behaviors, compared with familism

values, were more strongly linked to inflammatory processes for these youth. It may be that for African American youth, acting in a manner that is in general, consistent with their cultural context may increase feelings of purpose and meaning to a greater extent than endorsing familism values alone, and thus be more rewarding. Higher levels of eudaimonic well-being, in turn, have been associated in adults with decreased expression of proinflammatory genes and circulating levels of inflammation (Fredrickson et al., 2013; Ryff, Singer, & Dienberg Love, 2004) as well as lower risk for poor health outcomes such as stroke and mortality (Hill & Turiano, 2014; Kim, Sun, Park, & Peterson, 2013). Familism behaviors may also positively influence inflammatory processes in African American youth to a greater extent than familism values because behaviors may provide a concrete means for enhancing feelings of family belonging. Relative to other groups, African Americans experience higher levels of mistreatment and disadvantage, both of which pose risks for physical health problems (Adler et al., 1994; Borrell et al., 2007; Pascoe & Smart Richman, 2009; Semega, Frontenot, & Kollar, 2017). Assisting and spending time with the family in such challenging contexts may strengthen bonds with family members, thereby increasing feelings of family connectedness and integration. Engaging in familism behaviors may also provide opportunities for youth to seek and receive emotional and informational support about the specific stressors they may face. Family connectedness and support can lower levels of inflammation as noted above, and can mitigate the negative effects of youths' stress on inflammation and on the ANS and HPA axis (Barton, Yu, Brody, & Ehrlich, 2018; Luecken, Hagan, Wolchik, Sandler, & Tein, 2016; Miller et al., 2014).

Why familism behaviors did not relate to inflammatory outcomes among the Latino youth in our sample remains unclear. Similar to African American families, Latino families place great weight on familism and face relatively high rates of mistreatment and disadvantage (Fuligni et al., 1999; Greene, Way, & Pahl, 2006; Semega et al., 2017; Telzer & Fuligni, 2009). Yet, in the present investigation, the Latino youth exhibited the least proinflammatory profile, and there was no evidence that familism behaviors operated as a protective factor for them the way it did for their African American peers. The finding that Latinos had the least proinflammatory profile is consistent with the Latino paradox, which refers to epidemiological findings that Latinos have comparable or better health outcomes than Whites despite facing higher rates of disadvantage (Ruiz, Hamann, Mehl, & O'Connor, 2016). However, the fact that familism behaviors did not differentially associate with inflammatory outcomes between Latinos and White youth diverges from the hypothesis that familism may be a contributing factor to the Latino paradox (Ruiz et al., 2016). It may be that factors, experiences, and behaviors that disrupt, threaten, or do not conform to familism values, rather than those that uphold familism values, have consequences for Latino youths' inflammatory processes. Supporting this notion, it has been theorized that conflict with parents has particular salience for Latina youth, resulting in more guilt, suppressed anger, and internalizing symptoms, which in turn helps explain their higher rates of suicidal behavior relative to African American and White youth (Kuhlberg, Peña, & Zayas, 2010; Zayas & Pilat, 2008). Another possibility is that familism behaviors may reflect other factors that function differently for the health of African American and Latino youth. Religiosity may be one such factor. Religiosity has been linked to more cohesive



family relationships, lower levels of conflict, and higher levels of family assistance (Brody, Stoneman, & Flor, 1996; King, Ledwell, & Pearce-Morris, 2013), as well as to better health outcomes (Powell, Shahabi, & Thoresen, 2003). Notably, the extent to which religion is important in one's life is higher among African American compared with other ethnic groups including Whites and Latinos (Pew Research Center, 2018). This may contribute to the observed findings, as religiosity's links to health-related outcomes have been shown to vary by ethnicity (Caldwell & Takahashi, 2014). Future research should consider whether religiosity and other ethnicity-specific factors may help explain why familism was especially protective for African American youth and less so for Latino youth.

There was also some hint that engaging in more familism behaviors may be harmful for White youth. When controlling for additional covariates in sensitivity analyses, familism behaviors were associated with lower cortisol sensitivity among White youth. This pattern is consistent with another study that found that providing emotional support and assisting the family were associated with more externalizing symptoms and worse child-parent relationships in White youth but with better quality parent-child relationships in African American youth (Khafi et al., 2014). Engaging in familism behaviors may fit less with the values of Whites' cultural context, which generally tends to emphasize independence more than interdependence, even within the family context. Indeed, in our sample, Whites endorsed lower levels of familism values. As such, White youth may view engaging in familism behaviors as more obligatory and burdensome rather than meaningful and rewarding, as ethnic minority youth might.

Several limitations of the current investigation should be acknowledged. First, results are based on cross-sectional data, precluding definitive conclusions about causality and directionality. Other unmeasured factors that covary with familism and inflammatory processes may also be at play. For instance, religiosity rather than familism per se may account for findings observed among the African American youth, as described above. Second, familism behaviors were based on a single assessment that required participants to recall and generalize their behaviors across a broad window of time. Future work should consider incorporating daily diary measures assessing engagement in each behavior and time spent to minimize recall bias and inaccuracies. Third, it is possible that ethnic differences in social and emotional maturity and feelings of autonomy may have influenced reports of familism values and behaviors. Although all models controlled for pubertal status, which may be a proxy for maturity, additional research is needed to address this question directly. Fourth, inflammatory outcomes were based on composite variables that aggregated across four different proinflammatory cytokines. It is possible that familism values and behaviors are differentially associated with activity of specific cytokines; however, as noted above, we had no such a priori hypotheses and instead were interested in overall inflammatory processes and reducing Type I error. Fifth, we were unable to test underlying pathways through which familism behaviors may positively influence inflammatory processes. As described earlier, they may impact inflammatory processes by fostering meaning and purpose, enhancing family ties, providing a means for coping in challenging contexts, and reducing sympathetic activity. It will be important for future work to test these hypotheses and determine whether any of these proposed pathways

are independent of feelings of social connection tied to other contexts such as in youths' school or neighborhood community (Witherspoon, Schotland, Way, & Hughes, 2009). Sixth, we were unable to determine antecedents and parental influences on youths own familism values and behaviors. For instance, whereas some youth may engage in familism behaviors because they value doing so and find it rewarding, others may engage in them because their family circumstances present a need for it. Seventh, Latino ethnicity was assessed broadly. However, the Latino population consists of diverse subethnicities (e.g., Mexican, Cuban, Puerto Rican, South or Central American). Given subethnic differences in acculturative and immigration-related experiences (Rivera et al., 2008) and in health risk (Fenelon, Chinn, & Anderson, 2017; Garcia, Garcia, Chiu, Raji, & Markides, 2018), it is possible that familism functions differently across Latino subgroups. Consequently, whether the present findings generalize to all Latino subgroups remains unclear. Lastly, the clinical significance of our findings is unclear given that we recruited physically healthy youth without any major illness. Future longitudinal studies can help determine whether familism actually predicts better clinical health outcomes later in life for African Americans.

Despite these limitations, the present study deepens our understanding of how cultural values and behaviors may be protective for the health of certain ethnic minorities. Compared with Whites, African Americans face higher rates of cardiovascular disease and cancer, the top two leading causes of death in the United States. Heightened inflammation as a result of repeated large responses to biological threat and/or decreased sensitivity to anti-inflammatory mechanisms has emerged as a contributor to many of these outcomes. Our results suggest that engaging in familism behaviors may be one cultural resource that protects African American youth against heightened inflammation. For these youth, behaving consistently with their cultural family values may help offset the higher risk for poor health that they face. More work is needed to determine what cultural factors may similarly protect Latino youth against poor health outcomes.

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