Longitudinal Study of Familism, Inflammation, and Metabolic Syndrome in Black Adolescents From Lower-Income Households

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Objective: This study examined longitudinal associations between familism (valuing the family unit more than the individual, feelings of obligation and responsibility toward the family), inflammation, and metabolic syndrome (MetS) in a sample of Black adolescents from lower-income households.

Methods: Four hundred Black adolescents (ages 14 to 19) were recruited into this study. Adolescents and one parent independently completed a questionnaire measure of familism (expectations about the obligations adolescent have to the family) at baseline. As well, in adolescents, assessments of 6 markers of low-grade inflammation and MetS (counts of the number of MetS components above clinical cutoffs; composite z-scores of each MetS component) were taken at baseline. One year later, inflammation was re-assessed. Two years later, MetS was re-assessed. Discrepancies in familism (directional difference between parent and adolescent reports of familism; absolute difference between parent and adolescent familism scores) were calculated.

Results: Adolescents who held higher familism beliefs than their parents had higher levels of low-grade inflammation one year later, b = 0.032, SE = 0.016, $P < .05$, which in turn predicted higher levels of MetS 2 years later (count: b = 0.537, SE = 0.077, $P < .001$; composite: b = 0.294, SE = 0.051, $P < .001$). The indirect effects were significant (for prospective associations, count: coefficient = 0.016, 95%CI [0.002, 0.034]; composite: coefficient = 0.009, 95%CI [0.001, 0.020]). No associations were found with absolute difference in familism scores.

Conclusions: These findings suggest the importance of understanding family values from multiple perspectives (eg, parent, adolescent). Adolescents' perceptions about their families, particularly in relation to how their parents perceive the family, may have implications for the development of cardiovascular risk in young people over time.

Key Words: family, adolescents, familism, inflammation, metabolic syndrome.

Abbreviations: **CRP** = C-reactive protein, **IL** = interleukin, **MetS** = metabolic syndrome, **SES** = socioeconomic status, **suPAR** = soluble urokinase plasminogen activator receptor.

(*Biopsychosoc Sci Med* 2026;88:535–546)

INTRODUCTION

Familism is a construct centered around the idea that the family unit has a higher priority and focus than the individual. It includes dimensions of support, or the extent to which an individual feels close to and that they can depend on family members; obligations, or the extent to which individuals feel a responsibility to assist family members; and referent familism, or the extent to which individuals engage in behaviors that are consistent with family values and expectations.[1–3] Familism has traditionally been studied in Hispanic populations; however, related constructs including communalism (an emphasis on social relationships and interdependence over the individual) and filial piety (honoring one's family and carrying out family wishes) have been studied in Black and Asian populations. Moreover, these 3 constructs have been found to cluster onto a single factor,[4] and familism has been linked to outcomes in non-Hispanic samples as well.[5,6]

Levels of familism have been found to be higher in Black and Hispanic individuals compared with White individuals.[5,7] Familism has also been found to be higher in individuals from lower socioeconomic status (SES) households.[8,9] Theoretically, it has been proposed that familism may have particular importance in groups that more frequently face marginalization, discrimination, and life stressors, where the family could serve as an important source of support, protection, and help.[10,11] This is then proposed to lead to higher levels of obligation to, dependence on, and provision of assistance to the family on average in these groups.[12] Although familism has generally been conceptualized as a protective factor in

Received for publication December 10, 2025; accepted April 30, 2026.
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Supplemental Digital Content is available for this article. Direct URL citations are provided in the HTML and PDF versions of this article on the journal's website, journals.lww.com/bsam.

DOI: 10.1097/PSY.0000000000001499

family relationships, the effects it would be expected to have on individual psychological and physical health outcomes are less clear. On the one hand, the strong family ties that accompany familism might serve to buffer individuals from the detrimental effects of stress. On the other hand, the high levels of obligations associated with familism might accumulate and take a toll on family members' health and well-being.

Previous literature has, in general, linked higher levels of familism to better psychosocial outcomes, with the most reliable effects being associations with fewer depressive symptoms and fewer internalizing behaviors most frequently documented in Hispanic adolescents and adults (though apparent in other racial/ethnic groups as well),[1–3,13] and with effects also being documented longitudinally in Black, Hispanic, and low-income families.[3,14] Although less robust or sometimes less well-studied, associations have also been documented between higher levels of familism (or related constructs such as family obligations and family assistance) and higher self-esteem in Hispanic adolescents,[15] lower levels of substance use in Hispanic samples,[13,16] and better psychological/emotional well-being across various racial/ethnic groups.[17,18]

However, associations of familism with physical health are less well-established. Mixed patterns of associations between familism and health behaviors were described in one review.[13] Some studies have found associations of higher levels of familism (or related constructs like communalism) with better self-reported physical health across various racial/ethnic groups,[2,19] lower blood pressure in Black women,[20] lower rates of chronic conditions such as asthma across Black, White, and Hispanic individuals,[21] and reduced pro-inflammatory profiles among Black and Hispanic adolescents.[5] However, other studies have found associations of higher levels of familism (or family obligations/family assistance) with poorer self-rated health in Black and White individuals,[22] with flatter daily cortisol slopes (linked to greater daily family assistance) in Hispanic adolescents,[23] and with higher levels of inflammatory biomarkers among Hispanic and White adolescents.[24] And some studies documented no association of familism with cortisol reactivity to an acute laboratory stressor[25] or daily cortisol levels[23] in Hispanic adolescents.

One reason for these mixed patterns may be that familism may be important to understand at the family level, and yet it has traditionally been measured at the individual level (ie, by probing an individual's beliefs about familism). However, different family members—particularly when it comes to parents/caregivers (hereafter referred to as "parents") and their adolescent children—may hold different beliefs about familism (ie, different expectations about the obligations adolescent children have to the family). Beliefs that are discrepant across family members may have implications for health. In particular, when parents and their adolescent children articulate different beliefs about family values, this may create tension for adolescents as they are transitioning into adult responsibilities (and hence taking on more family obligations). If these discrepancies in expectations regarding the family are experienced as stressful by adolescents, this is in turn could have implications for stress-relevant physiological pathways such as low-grade inflammation, and subsequent physical health as well.[26–32]

Discrepancies can come in 2 forms: directional differences between parents and adolescents and absolute differences. Previous research suggests the effects of parent-adolescent discrepancies in other types of cultural and parenting variables on adolescent psychological outcomes. For example, when Hispanic adolescents report being more acculturated to the majority culture than their parents report being, these adolescents are more likely to engage in substance use.[33,34] In a similar vein, when adolescents perceive the parenting in their household to be more negative than parents report it to be, adolescents have higher levels of depressive symptoms, anxiety, and delinquent/externalizing behaviors, and as well, have poorer sleep across various racial/ethnic groups.[35–38] When adolescents perceive family communication to be worse than parents perceive it to be, these adolescents have higher levels of internalizing and externalizing problems, and girls also have lower levels of psychological well-being.[39] Absolute discrepancies have also been linked to adolescent outcomes. For example, the greater the absolute difference between parents and adolescents in reports of family functioning (regardless of the direction of the difference), the more adolescent girls engage in dieting behaviors.[40] Similarly, the greater the absolute discrepancies in reports of closeness between parents and adolescents, the lower adolescents' parasympathetic regulatory ability.[41] Taken together, these findings suggest the possibility that discrepancies in familism beliefs between parents and adolescents may be linked to adolescent outcomes. In the present study, we hypothesize that when there are discrepancies between what parents versus adolescents expect in terms of adolescents' obligations to the family, that this can create potentially stressful family contexts that will then be linked to indicators relevant to physical health in adolescents.

With respect to health outcomes, adolescence is typically a period of good health, and hence adolescent health studies typically have to focus on risk markers for diseases, given that many chronic diseases do not manifest until decades later in adulthood. In the present study, we focus on low-grade inflammation as one mechanistic pathway, given evidence that inflammation serves as a key biological process connecting childhood stress to the development of multiple chronic diseases in adulthood;[42] given the important mechanistic role that inflammation is acknowledged to play in chronic conditions such as obesity, atherosclerosis, and cardiovascular disease;[43–45] and given that biomarkers of inflammation such as C-reactive protein (CRP) and interleukin 6 (IL-6) have been prospectively associated with risk for type 2 diabetes, coronary heart disease, myocardial infarction, and stroke.[46–51] In this study, we constructed a composite of biomarkers

to reflect low-grade inflammation, given that composite biomarkers have been found to predict health outcomes more reliably than analyzing biomarkers individually.[52] The composite included CRP as the most commonly assessed biomarker of inflammation,[53] pro-inflammatory cytokines (eg, IL-6), and soluble urokinase plasminogen activator receptor (suPAR), a newer inflammatory biomarker that also has emerged as a predictor of cardiovascular disease, diabetes, cancer, and all-cause mortality, even independently of CRP,[54,55] and that has been speculated to reflect vascular inflammation.[56] With respect to clinical health outcomes, in the present study we focus on metabolic syndrome (MetS). MetS is a diagnosable condition consisting of a cluster of risk factors for diabetes, heart disease, and stroke that is detectable in childhood and adolescence.[57–59] Metabolic profiles assessed from the age of 12 onward have been found to forecast carotid atherosclerosis in adulthood, even independently of adult risks,[60] and cardiometabolic risk factors assessed in childhood predict clinical cardiovascular disease in adulthood.[61] Hence, MetS serves as a relevant physical health outcome and early warning sign of potential health problems in our relatively young sample.

The present study conducted a longitudinal investigation of familism, inflammation, and MetS in a sample of Black adolescents from lower-income households. We focus on Black adolescents from lower-income households for several reasons: (1) because of the relevance of familism to both Black families and those from low-income households; (2) because both Black individuals and those from low-income households on average experience poorer health than other groups, for example, higher rates of obesity, hypertension, as well as greater morbidity and mortality from cardiovascular diseases compared with White or higher-income individuals.[62–66] Adolescents were assessed at 3 timepoints, with each assessment being spaced 1 year apart. Adolescents and one parent completed a familism measure at baseline assessing each family member's expectations regarding adolescents' obligations to and prioritization of the family. We calculated the difference between parent and adolescents' familism scores. We tested associations with both absolute difference scores and directional difference scores. Directional difference scores tested the hypothesis that if adolescents report greater familism beliefs than their parents—meaning that they perceive a higher level of obligation to the family than their parents expect—that this relatively higher sense of obligation, particularly as adolescents are taking on more adult responsibilities in their lives, will be experienced as a burden that will be associated with greater inflammation, which in turn would then subsequently predict higher levels of MetS over time in adolescents. Absolute difference scores tested the hypothesis that differing opinions in the household about familism and family obligations create a stressful environment that will be associated with greater inflammation, which in turn would then predict higher levels of subsequent MetS in adolescents. To document the directionality of pathways over time, we tested whether households with greater absolute or directional difference familism scores at baseline would have adolescents who displayed higher levels of inflammation 1 year later, and subsequently, whether those with greater inflammation at 1 year would go on to be at greater risk for MetS 2 years later.

METHODS

Participants

Participants were drawn from an initial sample of 400 Black adolescents ages 14 to 19 recruited from the greater Chicago area through advertisements, presentations at schools, outreach to community organizations, and through a direct mail campaign. Study inclusion criteria included adolescents who identified as Black, were between ages 14 and 19, and whose family reported their income at screening to be below 2 times the federal poverty threshold for their household size. Other eligibility criteria included being English speaking, having no current major chronic illnesses that necessitated taking regular medication, having no mental health disorder serious enough to warrant hospitalization in the past year, and having no pervasive developmental disorder that would make the adolescent unable to complete the study protocol. Participants who were currently pregnant or acutely ill were offered the option of rescheduling. Adolescents provided either written assent or consent (depending on age), and parents provided written consent for all study procedures, which were approved by the Northwestern University Institutional Review Board. Sample size was determined by power analyses that were conducted for the broader aims of the grant that funded this project, which were related to longitudinal trajectories of academic achievement and health in Black youth. Data collection for the baseline (time 1) visit occurred between November 2018 and December 2022.

Eligible adolescents were invited for their time 1 baseline laboratory visit, during which they completed psychosocial questionnaires and health measures including a fasting blood draw (scheduled for the morning, generally between 8 and 10 am, to minimize diurnal variation). Parents also completed questionnaires at baseline. 28 adolescents did not have a parent who was available to participate, leaving a total sample at baseline for study analyses of 372.

One year later, 361 of the 400 adolescents returned for their time 2 visit. Two years after the baseline visit, 358 adolescents returned for their time 3 visit. Data for study analyses below draw from health data provided by adolescents at times 2 and 3.

Measures

Familism Obligations

Values of familism were assessed at baseline in both parents and adolescents using 11 items from the Attitudinal Familism Scale,[67] with the wording of several items reframed to make them appropriate for adolescents, as

previous research has done.[5] This measure has been used across multiple racial/ethnic groups.[5] Parents completed an identical measure to adolescents, largely framed around what children should be doing with respect to the family. Items assessed 2 central dimensions of familism: obligation to support (the belief that a person has an obligation to support other family members) and subjugation of self for family (the belief that a person must be submissive and yield to the family). Using a 10-point scale (1 = strongly disagree, 10 = strongly agree), adolescents and parents indicated the extent to which they agreed with items such as "Children should regularly help their parents with younger brothers and sisters, for example, help them with homework, help take care of them, and so forth", and "Children should follow their parent's/guardian's advice (eg, about friends, jobs, college) even when they don't agree with them." Responses were averaged across items to compute an overall familism score for adolescents and an overall score for parents (Cronbach alpha = 0.83 and 0.82 in adolescents and parents, respectively). Directional difference scores were calculated as adolescent score minus parent score; thus, higher positive numbers indicate adolescents who feel greater obligations to the family than their parents expect. Absolute difference scores were calculated as the absolute difference of adolescent and parent familism scores; thus, higher numbers indicate a greater difference between parents and adolescents in their familism beliefs (regardless of the direction of that difference).

Health Measures

Our interest was in testing longitudinal patterns related to the development of cardiometabolic risk, and more specifically, a model in which risk factors (inflammation) develop before indicators of clinical disease (metabolic syndrome). All health measures were assessed at baseline (time 1). We then utilized samples collected at time 2 to reassess inflammation 1 year later. We utilized health measures collected at time 3 to reassess metabolic syndrome 2 years later.

Low-Grade Inflammation

At time 1 and time 2, 6 circulating biomarkers of low-grade inflammatory activity implicated in cardiovascular disease[68–70] were quantified in antecubital blood drawn into Serum Separator Tubes (Becton-Dickinson). The specimens were centrifuged within an hour of venipuncture, and serum was harvested and frozen at −80°C until study completion. CRP, IL-6, IL-8, IL-10, tumor necrosis factor (TNF)-α, and suPAR were assayed in batch. CRP was measured in duplicate by high-sensitivity immunoturbidimetric assay on a Roche/Hitachi cobas c502 instrument. The cytokines were measured in triplicate by 4-plex immunoassay on a microfluidic platform (Simple Plex; Protein Simple). suPAR was measured in duplicate by immunoassay (Human Quantikine ELISA; R&D Systems). Intra-assay coefficients of variation ranged from 1.96% to 7.18%.

Most of the inflammatory biomarkers were skewed and/or kurtotic, so we normalized their distributions with log-10 transformations. The logged values were then standardized and averaged into a composite (α = 0.65), consistent with previous research.[71–73] The composite has the advantage of reducing the number of statistical tests performed, and therefore the rate of false discoveries. It also better reflects in vivo conditions, where pro-inflammatory molecules have redundant and synergistic effects on targets. Higher values reflected greater inflammation.

Metabolic Syndrome (MetS)

We drew on measures collected at time 1 and time 3 to calculate MetS. Resting blood pressure was monitored with an automated auscultatory device (Carescape V100; GE) while the participant sat quietly. After a 4-minute acclimation, 4 readings were taken every 2 minutes, and the average of the last 3 readings was calculated. Waist circumference was measured at the midpoint of the upper iliac crest and lower costal margin, at the midaxillary line. Fasting blood was drawn into serum separator tubes. Serum was assayed at NorthShore University Health System's Core Laboratory for glucose and a cholesterol panel (visit times always scheduled in the morning to minimize diurnal variation).

MetS was diagnosed according to International Diabetes Federation guidelines.[58] These criteria specify that the diagnosis of MetS requires central adiposity, which for the participants in this sample is defined as waist circumference ≥ 94 cm for males and ≥ 80 cm for females. At least 2 of 4 additional components must also be present. This includes: (a) signs of early hypertension (systolic pressure ≥ 130 or diastolic pressure ≥ 85 mm Hg), (b) elevated triglycerides (≥ 150 mg/dL), (c) elevated fasting glucose (≥ 100 mg/dL), or (d) lowered high-density lipoprotein levels (< 40 mg/dL in males and < 50 mg/dL in females).

Because of the young age of this sample, only 2% met criteria for MetS diagnosis at time 3. Hence we focused analyses on 2 other MetS outcomes, consistent with previous research on younger samples.[74–76] One was a count of the number of MetS components for which participants met clinical cutoff criteria. Second, acknowledging concerns about the validity of dichotomizing youth into risk categories when metabolic functioning is distributed on a continuum,[77,78] a MetS composite was calculated using the mean of z-scores of each MetS component.

Covariates

Youth age, sex at birth (0 = female, 1 = male), pubertal status, and family socioeconomic status (SES) at baseline were included as covariates. Pubertal status was measured using a questionnaire, the Pubertal Development Scale,[79] which has been validated against the Tanner physical examination evaluation for determining pubertal stage, and has been used in similar populations.[80–82] Pubertal stage is scored from 1 to 5 (1 = pre-puberty, 2 = early puberty, 3 = mid-puberty, 4 = late puberty,

5 = post-puberty). Family SES was calculated by standardizing and averaging family income and family savings. This set of covariates is comparable to other adolescent health studies.[74,75,83] In supplemental analyses, parent variables of parent age, parent sex, parent marital status (1 = married, 0 = single/divorced/widowed), and whether the parent and youth were currently living together (1 = yes, 0 = no) were included as additional covariates.

Statistical Analyses

Missing data: 2 adolescents and 1 parent did not complete the familism questionnaire. 4 adolescents did not have sufficient blood samples for inflammation assays at time 1, and 3 adolescents at time 2. 4 adolescents were missing sufficient blood samples at time 1 for MetS calculations, and 2 adolescents at time 3. Analyses were conducted on participants who had full data for all variables included in each analysis.

Study hypotheses were tested using linear regression equations with sequentially entered blocks of variables. In our first set of analyses, to test the longitudinal link between familism and inflammation, the inflammation composite at time 2 (1-year follow-up) was regressed onto (a) covariates at time 1 (baseline) age, sex, pubertal status, and family SES; and (b) time 1 familism score. Separate regression analyses were conducted for directional difference familism scores and for absolute difference familism scores. Additional analyses included baseline adolescent familism score and baseline inflammation composite as covariates. In our second set of analyses, to test the longitudinal link between inflammation and MetS, MetS at time 3 (2-year follow-up) was regressed onto (a) covariates described above; and (b) time 2 inflammation composite. Additional analyses included baseline MetS as a covariate.

We then conducted mediation analyses testing the longitudinal pathway: time 1 familism → time 2 inflammation → time 3 MetS. The Hayes PROCESS macro was used, a regression-based approach that calculates the indirect effect as a product of the regression coefficient between the independent variable and the mediator and the regression coefficient between the mediator and the dependent variable.[84] Nonparametric bootstrapping was used to obtain the bias-corrected and accelerated confidence intervals of parameter estimates for significance testing. The parameter estimate was calculated 5000 times using random sampling with replacement to build a sampling distribution.[85]

RESULTS

Mean, SDs, and percentages for adolescent and parent study variables are presented in Table 1. Participants who participated in all 3 study visits did not differ from those who missed 1 or 2 study visits in terms of any adolescent or parent variables listed in Table 1 that were collected at the baseline visit (all P's > .10). The low-grade inflammation composite correlated at $r = 0.510$, $P < .001$ between time 1 and time 2. The MetS variables correlated 0.677 to 0.763, P's < .001 between time 1 and time 3.

TABLE 1. Descriptive Information About Sample (N = 400 Adolescents at Time 1, 361 at Time 2, 358 at Time 3, N = 372 Parents at Time 1)

	%	Mean	SD
Adolescent age, time 1		16.39	1.56
Adolescent sex (% female)	64		
Adolescent pubertal status, time 1		4.18	0.72
Parent age, time 1		44.72	9.51
Parent sex (% female)	90		
Parent marital status (% married), time 1	28		
Adolescent living with parent (% yes)	92		
Family socioeconomic status, time 1		0.00	0.71
Familism directional difference, time 1		0.43	2.01
Familism absolute difference, time 1		1.64	1.25
Inflammation composite, time 1		0.00	0.60
Metabolic syndrome count, time 1		0.94	0.90
Metabolic syndrome z-score, time 1		0.00	0.59
Inflammation composite, time 2		0.00	0.58
Metabolic syndrome, count, time 3		0.97	0.87
Metabolic syndrome z-score, time 3		0.00	0.57

T1 = time 1 (baseline). T2 = time 2 (1-y follow-up). T3 = time 3 (2-y follow-up). Family socioeconomic status = standardized and averaged family income and family savings. Familism directional difference = adolescent minus parent familism score. Familism absolute difference = absolute value of difference between adolescent and parent familism score. Inflammation composite = standardized and average scores for biomarkers IL-6, IL-8, IL-10, TNF-α, CRP, and suPAR. MetS count = count of number of metabolic syndrome components above clinical cutoffs. MetS z-score = average of z-scores of each metabolic syndrome component.

Longitudinal Link: Directional Difference Familism Score at Time 1 and Inflammation at Time 2

After controlling for age, sex, pubertal status, and family SES, familism discrepancy scores calculated as directional differences at time 1 predicted the inflammation composite at time 2, b = 0.032, SE = 0.016, $P = .047$. This association indicated that adolescents who reported higher beliefs about familism than their parents did had higher levels of low-grade inflammation 1 year later. Even after controlling for baseline levels of adolescents' familism, associations were still found with familism discrepancy scores, indicating that those adolescents who reported higher beliefs about familism than their parents at baseline had higher levels of low-grade inflammation 1 year later, b = 0.044, SE = 0.020, $P = .031$. When baseline levels of inflammation were additionally included as a covariate, familism discrepancy still significantly predicted time 2 inflammation, b = 0.037, SE = 0.018, $P = .042$. This association indicates that adolescents who had higher familism beliefs than their parents at baseline showed greater increases in inflammation over time. See Table 2.

Longitudinal Link: Absolute Difference Familism Score at Time 1 and Inflammation at Time 2

After controlling for age, sex, pubertal status, and family SES, the absolute difference familism score at time 1 did not predict the inflammation composite at time 2, b = 0.013, SE = 0.026, $P = .605$.

Longitudinal Link: Inflammation at Time 2 and MetS at Time 3

After controlling for age, sex, pubertal status, and family SES, adolescents' inflammation composite at time

TABLE 2. Discrepancies in Familism Obligations (Adolescent Minus Parent Familism Scores) at Baseline Predicting Adolescent Inflammation at 1-Year Follow-Up

	Standard covariates			Standard covariates + adolescent familism			Standard covariates + adolescent familism + baseline inflammation		
	b	[95% CI]	*P*	b	95% CI	*P*	b	95% CI	*P*
Step 1									
Age	0.015	[−0.028, 0.059]	.494	0.015	[−0.029, 0.058]	.511	0.013	[−0.026, 0.051]	.517
Sex	0.149	[−0.009, 0.306]	.064	0.153	[−0.006, 0.311]	.059	0.062	[−0.077, 0.201]	.383
Pubertal status	−0.114	[−0.226, −0.002]	.045	−0.110	[−0.224, 0.003]	.057	−0.074	[−0.174, 0.025]	.142
SES	−0.032	[−0.116, 0.052]	.455	−0.032	[−0.117, 0.052]	.454	−0.008	[−0.082, 0.066]	.836
Adolescent familism				0.011	[−0.031, 0.053]	.615	0.011	[−0.026, 0.047]	.573
T1 inflammation							0.473	[0.382, 0.564]	< .001
Step 2									
Familism discrepancy	0.032	[0.000, 0.063]	.047	0.044	[0.004, 0.084]	.031	0.037	[0.001, 0.072]	.042

SES = family socioeconomic status.
b = unstandardized regression coefficient. CI = confidence interval. T1 = time 1 (baseline). Familism discrepancy = adolescent minus parent familism score. Inflammation = standardized and average scores for inflammatory biomarkers IL-6, IL-8, IL-10, TNF-α, CRP, and suPAR.

2 predicted their MetS count at time 3, b = 0.537, SE = 0.077, $P < .001$, such that higher levels of low-grade inflammation at the 1-year follow-up longitudinally predicted higher MetS count score in adolescents at the 2-year follow-up. After controlling for baseline MetS count, associations remained significant, b = 0.193, SE = 0.066, $P = .004$, indicating that low-grade inflammation also predicted change in MetS count score over time. See Table 3.

Similarly, the inflammation composite at time 2 also predicted MetS composite scores at time 3, b = 0.294, SE = 0.051, $P < .001$, such that higher levels of low-grade inflammation at the 1-year follow-up longitudinally predicted a higher MetS composite score in adolescents at the 2-year follow-up. After controlling for baseline MetS composite, this association weakened, b = 0.065, SE = 0.037, $P = .083$. See Table 3.

Mediation Analyses

Table 4 presents results of mediation analyses. Because the analyses above report the association between the IV (familism discrepancy) and the mediator (inflammation), as well as between the mediator (inflammation) and the DV (MetS), reporting of results of mediation analyses below focus on indirect effects tests. Three different types of indirect effects tests were conducted. Prospective analyses tested the temporal pathway familism discrepancy at V1 → inflammation at V2 → MetS at V3 controlling for age, sex, pubertal status, and family SES. Change in inflammation analyses tested the same pathway but included inflammation at V1 as an additional covariate. Thus, what this mediation analysis tests is whether familism discrepancy at baseline predicts change in inflammation over a 1-year period, and whether this change is then in turn associated with MetS at the 2-year follow-up. Change in MetS tested the same pathway above but now controlling for both inflammation at V1 and MetS at V1. Thus what this mediation analysis tests is whether familism discrepancy at baseline predicts change in inflammation over a 1-year period, and whether this change is then in turn associated with change in MetS across the 2-year follow-up period. Significant effects below are indicated by confidence intervals that do not include 0.

In prospective analyses, the indirect pathway from baseline familism discrepancy scores to 1-year inflammation scores to the 2-year MetS score was significant for both MetS counts, b = 0.016, SE = 0.008, 95% CI [0.002, 0.033] and MetS composite (z-score), b = 0.009, SE = 0.005, 95% CI [0.0005, 0.019]. The direct effect from familism discrepancy to MetS was not significant with the mediator and covariates included.

In change in inflammation analyses, the indirect pathway from baseline familism discrepancy scores to 1-year change in inflammation scores to the 2-year MetS score was significant for both MetS counts, b = 0.009, SE = 0.005, 95% CI [0.001, 0.021] and MetS composite z-score, b = 0.004, SE = 0.003, 95% CI [0.0001, 0.011]. The direct effect from familism discrepancy to MetS was not significant with the mediator and covariates included.

TABLE 3. Adolescent Inflammation at 1-Year Follow-Up Predicting Adolescent Metabolic Syndrome at 2-Year Follow-Up

		b	[95% CI]	*P*	b	[95% CI]	*P*
Outcome:	T3 MetS count						
Step 1	Age	0.016	[−0.046, 0.079]	.610	−0.011	[−0.058, 0.036]	.649
	Sex	0.470	[0.234, 0.705]	< .001	0.242	[0.064, 0.420]	.008
	Pubertal status	−0.077	[−0.244, 0.090]	.365	−0.054	[−0.179, 0.072]	.399
	SES	−0.006	[−0.130, 0.118]	.922	−0.036	[−0.130, 0.057]	.442
	T1 MetS count				0.629	[0.552, 0.706]	< .001
Step 2	T2 inflammation	0.537	[0.385, 0.688]	< .001	0.193	[0.064, 0.323]	.004
Outcome:	T3 MetS z-score						
Step 1	Age	0.052	[0.011, 0.092]	.012	0.020	[−0.007, 0.047]	.146
	Sex	−0.204	[−0.357, −0.051]	.009	−0.069	[−.171, 0.032]	.181
	Pubertal status	−0.095	[−0.204, 0.014]	.086	−0.043	[−.115, 0.028]	.236
	SES	−0.019	[−0.099, 0.062]	.650	−0.020	[−.074, 0.033]	.449
	T1 MetS z-score				0.706	[.639, 0.773]	< .001
Step 2	T2 inflammation	0.294	[0.194, 0.395]	< .001	0.065	[−.008, 0.138]	.083

SES = family socioeconomic status.

b = unstandardized regression coefficient. CI = confidence interval. T1 = time 1 (baseline). T2 = time 2 (1-year follow-up). T3 = time 3 (2-year follow-up). MetS count = count of number of metabolic syndrome components above clinical cutoffs. MetS z-score = average of z-scores of each metabolic syndrome component. Inflammation = standardized and averaged scores for inflammatory biomarkers IL-6, IL-8, IL-10, TNF-α, CRP, and suPAR.

In change in MetS analyses, the indirect pathway from baseline familism discrepancy scores to 1-year change in inflammation scores to 2-year change in MetS was not significant for either MetS counts or MetS composite score. The direct effect from familism discrepancy to MetS was also not significant. See Table 4.

Supplemental Analyses

To determine whether patterns of results were robust to additional covariates, we conducted supplemental analyses repeating the above regression analyses between familism discrepancy and inflammation, and between inflammation and MetS controlling for parent demographic variables. After inclusion of parent age, sex, marital status, and whether the adolescent was currently living at home with their parent as covariates (together with the original covariates of adolescent age, sex, pubertal status, family SES, adolescent familism score, and baseline inflammation), the directional difference familism score at time 1 continued to predict the inflammation composite at time 2, b = 0.037, SE = 0.018, $P = .045$. See Table S1, Supplemental Digital Content, http://links.lww.com/PSYMED/B191.

Analyses testing associations between inflammation at time 2 and MetS at time 3 also showed similar patterns as described above after adding parent variables as covariates. When including both parent and youth demographic covariates, adolescents' inflammation composite at time 2 continued to predict their MetS count at time 3, b = 0.504, SE = 0.078, $P < .001$, and their MetS composite z-scores at time 3, b = 0.276, SE = 0.052, $P < .001$. After adding the additional control of baseline MetS, adolescents' inflammation composite at time 2 continued to predict increases in MetS counts over time, b = 0.166, SE = 0.067, $P = .014$, but not increases in MetS composite scores, b = 0.057, SE = 0.038, $P = .132$. See Table S2, Supplemental Digital Content, http://links.lww.com/PSYMED/B191.

In addition, we tested whether associations between inflammation and MetS remained after controlling for familism discrepancy scores. Adolescents' inflammation composite at time 2 continued to predict MetS count at time 3, b = 0.512, SE = 0.079, $P < .001$, MetS composite z-scores at time 3, b = 0.287, SE = 0.049, $P < .001$, change in MetS counts over time, b = 0.177, SE = 0.069, $P = .011$, and marginally predicted change in MetS composite over time, b = 0.068, SE = 0.039, $P = .078$. See Table S3, Supplemental Digital Content, http://links.lww.com/PSYMED/B191.

Finally, adding adolescent familism score as a covariate did not alter any of the mediation results. In prospective analyses, the indirect pathway from baseline familism discrepancy scores to 1-year inflammation scores to the 2-year MetS score remained significant for both MetS counts, b = 0.021, SE = 0.011, 95% CI [0.002, 0.045] and MetS composite (z-score), b = 0.012, SE = 0.006, 95% CI [0.002, 0.025]. As well, the indirect pathway from baseline familism discrepancy scores to 1-year change in inflammation scores to the 2-year MetS score remained significant for both MetS counts, b = 0.012, SE = 0.007, 95% CI [0.001, 0.028] and MetS composite z-score, b = 0.006, SE = 0.004, 95% CI [0.0002, 0.014]. See Table S4, Supplemental Digital Content, http://links.lww.com/PSYMED/B191.

DISCUSSION

This study of Black adolescents from lower-income households revealed longitudinal links across a 2-year period among familism, adolescent inflammation, and adolescent MetS. Specifically, this study demonstrated the importance of considering perceptions of family values from both the adolescent and parent perspectives. When predicting adolescent inflammation, it was not adolescents' own sense of familism that was related, but rather the discrepancy between adolescents' and parents' familism values. Adolescents who reported greater values of familism (greater obligations to help the family) than their parents expected of them had higher levels of low-grade

TABLE 4. Mediation Analyses

Pathway	b	SE	[95% CI]
Prospective			
Indirect effect: Familism discrepancy T1 → inflammation T2 → MetS count T3	0.016	0.008	[0.002, 0.033]
Direct effect: Familism discrepancy T1 → MetS count T3	0.026	0.022	[−0.019, 0.070]
Covariates: age, sex, pubertal status, SES			
Indirect effect: Familism discrepancy T1 → inflammation T2 → MetS z-score T3	0.009	0.005	[0.001, 0.019]
Direct effect: Familism discrepancy T1 → MetS z-score T3	0.013	0.014	[−0.015, 0.040]
Covariates: age, sex, pubertal status, SES			
Change in inflammation			
Indirect effect: Familism discrepancy T1 → inflammation T2 → MetS count T3	0.009	0.005	[0.001, 0.021]
Direct effect: Familism discrepancy T1 → MetS count T3	0.025	0.022	[−0.018, 0.069]
Covariates: age, sex, pubertal status, SES, inflammation t1			
Indirect effect: Familism discrepancy T1 → inflammation T2 → MetS z-score T3	0.004	0.003	[0.0001, 0.011]
Direct effect: Familism discrepancy T1 → MetS z-score T3	0.013	0.014	[−0.014, 0.040]
Covariates: age, sex, pubertal status, SES, inflammation t1			
Change in MetS			
Indirect effect: Familism discrepancy T1 → inflammation T2 → MetS count T3	0.002	0.003	[−0.002, 0.009]
Direct effect: Familism discrepancy T1 → MetS count T3	0.025	0.018	[−0.010, 0.060]
Covariates: age, sex, pubertal status, SES, inflammation t1, MetS count t1			
Indirect effect: Familism discrepancy T1 → inflammation T2 → MetS z-score T3	0.001	0.001	[−0.002, 0.004]
Direct effect: Familism discrepancy T1 → MetS z-score T3	0.018	0.010	[−0.002, 0.038]
Covariates: age, sex, pubertal status, SES, inflammation t1, MetS z-score t1			

b = unstandardized regression coefficient. se = standard error. CI = confidence interval. Indirect and direct effects shown in top part of each section for MetS count and in bottom part for MetS z-score. Prospective, change in inflammation, and change in MetS each reflect different sets of covariates.

inflammation 1 year later. In turn, these higher levels of low-grade inflammation predicted higher MetS scores 2 years later in adolescents. In contrast, absolute differences between parents' and adolescents' familism values were not related to low-grade inflammation.

These patterns suggest that the direction of differences between family members in their beliefs about the family matter for adolescent health. Among Black families from lower-income households, adolescents who hold stronger beliefs about the importance of helping the family than even their parents expect of them may be shouldering a higher level of family burdens as they transition into young adulthood. For example, many of these adolescents may hold expectations that they need to be the one taking care of the family (eg, graduating from college, getting a high-paying job, financially supporting both nuclear and extended family members, especially when many of these family members come from low socioeconomic backgrounds[86,87]). These adolescents may also feel pressure to consistently behave as role models to younger siblings and may feel an obligation to have wise answers and advice when younger siblings turn to them (eg, for questions about college, particularly if many of these adolescents come from households where parents didn't have opportunities to attend college).[88,89] Although admirable, this may also increase the burdens experienced by these adolescents, resulting in "parentification" (whereby adolescents assume parental roles that may be developmentally inappropriate). Levels of parentification have been found to be higher in Black than White students, and are associated with higher levels of psychopathology symptoms.[90,91] Moreover, when parents and adolescents are not on the same page about expectations, it may result in these adolescents feeling like they have to handle responsibilities on their own, without help or support from parents.[92] These burdens that adolescents may be shouldering largely in isolation may create high levels of stress that, in turn, may contribute to elevated biological risk factors for CVD during adolescence, such as low-grade inflammation.

Our study findings are consistent with previous research that has demonstrated that directional differences in family perceptions are related to adolescent psychological well-being. Although not tested with familism per se, previous research has investigated discrepancies in family relationship quality—which is linked to familism[93]—and their associations with behavioral and psychological outcomes. For example, when adolescents perceive parenting to be more negative than parents report it to be, these adolescents have higher levels of depressive symptoms, anxiety, externalizing behaviors, and poorer sleep.[35–38] As well, when adolescents perceive family communication to be worse than parents perceive it to be, these adolescents have higher levels of internalizing and externalizing problems.[39] The present study extends this previous research to the domain of physical health. Furthermore, in line with our speculation that those who were higher on familism discrepancy may have been experiencing high expectations and pressures around juggling multiple roles, previous research documents that high levels of competing demands, role overload, and vital exhaustion/burnout are all associated with a greater risk of health problems and chronic diseases such as heart disease.[12,94–96] In addition, if familism discrepancy results in higher stress related to perceived demands and family roles, this could have implications for inflammation, as seen in multiple decades of research that has established relationships between stress and inflammation.[31,42,97]

In addition, this study illustrates why developmental trajectories are important to assess in health studies of adolescents. Because adolescence is a period of good health, generally speaking, chronic diseases of aging, such as cardiovascular disease, will not be diagnosable yet. Researchers have long advocated the utility of including biological markers of risk in health psychology studies, such as CRP or cortisol or blood pressure reactivity, that have demonstrated links to clinical health outcomes.[48,98–103] However, few human studies exist, particularly longitudinal ones, that link a psychological/social variable to an intermediary biological risk marker to a clinical health outcome within the same study (see[32,104] for some exceptions). In this study, we document a temporal ordering, whereby our psychological variable at baseline (familism discrepancy scores) predicted our biological risk marker (low-grade inflammation) 1 year later in adolescents; in turn, low-grade inflammation at the 1-year timepoint predicted our clinical health outcome (metabolic syndrome scores) at the 2-year timepoint. Further, the indirect effect from baseline familism discrepancy to 1-year low-grade inflammation to two-year MetS was statistically significant across 2 different types of MetS scores for both the prospective model (without T1 variables as covariates) as well as for change in inflammation analyses (whereby familism discrepancy predicted change in inflammation across a 1-y period). Indirect effects analyses were not significant when testing both change in inflammation and change in MetS; it is possible that these types of associations could become evident if we were able to include a longer follow-up period for assessing change in clinical health outcomes. Overall, although, by including multiple timepoints of assessments and both biological risk markers and clinical health outcomes, we were able to document a progression from psychological variables to biological risk, and then to clinical health outcomes as adolescents matured.

Strengths of the present study include the longitudinal component with 3 timepoints of assessments, the inclusion of both the adolescent and a parent when investigating the role of family factors, and the focus on a marginalized population that has been understudied in previous research. Limitations include the observational nature of the study. Future studies that attempt to experimentally manipulate perceptions of familism will be important for drawing causal conclusions about these associations. As well, we were only able to focus on one specific dyad—an adolescent and their parent—when in reality, family situations can be complex with multiple caregivers and other family members influencing relationships and perceptions and values related to the family. In addition, the generalizability of this study is uncertain, given the focus on Black adolescents from lower-income households. Future studies will want to address whether similar patterns emerge in other racial/ethnic and socioeconomic groups. In addition, it will be important for future studies to include even longer follow-up periods to document links between these study variables and chronic disease diagnoses such as cardiovascular disease and diabetes later in life.

Implications of the findings from this study suggest the importance of fostering good communication across family members. When parents and their adolescent children view familism differently, this may be because they are not explicitly discussing what obligations they each believe children should have to the family. If family day-to-day life proceeds with these obligations being more understood than explicitly articulated, adolescents may understandably develop beliefs that depart from those of their parents, leading to discrepancies that are associated with adolescent health outcomes. Although this study would need to be replicated first, if it were, it might suggest that families that are highly discrepant in their familism beliefs might benefit from family interventions promoting open dialogues between parents and adolescents about expectations and obligations in the family context.

In sum, this longitudinal study demonstrated that discrepancies in family values are related to adolescent health among Black individuals growing up in lower-income households. Adolescents who held higher familism beliefs than their parents had higher levels of low-grade inflammation one year later, which in turn predicted higher levels of MetS 2 years later. These findings suggest the importance of understanding family values from multiple perspectives (eg, parent, adolescent). Adolescents' perceptions about their families, particularly in relation to how their parents perceive the family, may be important not only to their well-being but also for prevention efforts around cardiovascular risk.

Transparency and Openness: This study was not preregistered, nor was the data analysis plan. The study protocol, materials, data, and analytic code are not available.

Source of Funding and Conflicts of Interest: This research was supported by NIH grant HD093718. The authors report no conflicts of interest.

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