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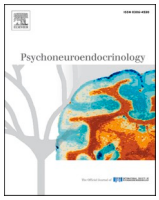

Longitudinal links between early adolescent temperament and inflammation among young black adults

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ABSTRACT

A large body of research demonstrates that inflammation is involved in physical health problems that cause substantial morbidity and early mortality. Given inflammation's role in the etiology of chronic diseases, pediatric scientists have begun to study childhood factors that presage elevation of inflammatory biomarkers later in life. The purpose of this study was to test hypotheses designed to determine whether early adolescent emotionally intense and low attention temperaments forecast (a) inflammation at ages 25 and 29 years and (b) worsening levels of inflammation between these two data points. Toward this end, 307 Black children from the rural southeastern United States participated in an 18-year longitudinal study (mean age at baseline, 11.2 years) to determine whether and how early adolescent's behavioral styles or emotionally intense and low attention temperaments may be associated with absolute and worsening levels of inflammation in young adulthood. When children were 11–13 years of age, different teachers at each age provided assessments of emotionally intense and low attention temperaments. Thus, multiple measures of the same temperament constructs were obtained across 3 years for each participant. At age 25, participants provided data on their self-regulation abilities. Peripheral blood was collected at ages 25 and 29 years from which inflammation was quantified, using soluble urokinase plasminogen activator (suPAR), the proinflammatory cytokines interleukin (IL) IL-6, IL-10, and tumor necrosis factor-alpha (TNF-α). Covariates associated with inflammation in prior studies were also assessed; these included socioeconomic risk, gender, cigarette smoking, body mass index (BMI), adverse childhood experiences (ACEs), depressive symptoms, and medication use. An early adolescent emotionally intense temperament was associated directly with higher suPAR and cytokine levels at age 29, and with worsening cytokine levels between ages 25 and 29. A low attention temperament was associated with suPAR levels at age 29. Collectively, these observations highlight pathways that could underlie health risks associated with early adolescent temperaments. The findings suggest that emotionally intense and low-attention early adolescent temperaments forecast higher and worsening inflammation levels across young adulthood.

1. Introduction

A large body of research demonstrates that excessive inflammatory activity is involved in physical health problems that cause substantial morbidity and mortality (Couzin-Frankel, 2010). These problems include asthma, rheumatoid arthritis and other autoimmune disorders, certain cancers, cardiovascular disease, and stroke (Miller et al., 2011). More than 50% of all deaths in the world today are attributable to inflammation-related disease conditions (Furman et al., 2019). Given excessive inflammation's role in the etiology of chronic diseases across the lifespan, pediatric scientists have begun to investigate childhood experiences that presage elevation of inflammatory biomarkers later in life. To date, many of these efforts have focused on severe and chronic psychosocial stressors such as maltreatment (Danese et al., 2007), bullying (Copeland et al., 2014), sexual abuse (Bertone-Johnson et al., 2012), and economic hardship (Chen et al., 2012). Although these studies are provocative, their focus on severe and fairly chronic stressors limits understanding of the breadth of factors that contribute to inflammation later in life.

In this study, we used an 18-year, multiple-wave, multiple-informant research design to determine whether and how early adolescent's behavioral styles, or *temperaments*, are associated with absolute and

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worsening levels of inflammatory biomarkers in young adulthood. Psychometrically sound, age-appropriate, measures are available that allow the study of temperament in early adolescence, a period of biological and social – emotional growth second only to infancy (Rothbart, 2011). Research identifying temperamental risks during early adolescence can inform novel etiological models and prevention strategies that fit individuals' needs. We tested the hypothesis that a preadolescent temperament characterized by high emotional intensity or low attention would presage (a) higher inflammatory biomarker levels at ages 25 and 29 years and (b) worsening levels of these biomarkers in the period between these two data points. We also hypothesized that self-regulatory abilities during young adulthood would serve as a mediator connecting inflammatory biomarkers with temperaments characterized by emotional intensity or low attention levels.

Temperament refers to enduring individual differences in emotional, behavioral, and biological reactivity that are present in childhood and adolescence (Rothbart, 2011). Rothbart's model, which often guides research on youth temperament, posits three key constructs: negative emotionality (NE), effortful control (EC), and positive emotionality (PE). Whereas NE and EC represent cohesive domains, empirical evidence does not suggest the same for PE (Snyder et al., 2015). Thus, the current study focused on emotional intensity, a facet of NE, and low attention, a facet of EC. Emotional intensity describes general tendencies to experience and express negative affect such as anger, hostility, and distress. Low attention involves a propensity to be easily distracted, hindering the ability to focus on a task, and ignore competing stimuli. Both emotional intensity and low attention evince stability across childhood through adolescence and persist as distinct domains across the lifespan (Putnam et al., 2001). Thus, individual differences in emotional intensity and attention control are present at an early age and are relatively enduring.

Although an abundance of research has delineated associations of childhood emotionally intense and low attention temperaments with adolescent and adult mental health problems - including anxiety, depression, and conduct disorder (Atherton et al., 2020) - research has barely examined the potential links between these early adolescent temperament dimensions and inflammation. In a study of 67 adolescents, researchers observed higher levels of C-reactive protein (CRP) among participants with temperaments characterized by negative emotionality and low attention (Nelson et al., 2018). Although this study suggests a relationship between adolescent temperament and inflammation, questions remain about the durability of any direct relationships over time, and about mediating processes through which early adolescent temperaments are linked to inflammation. This study addressed both of these issues.

Two areas of research support a hypothesized association between early adolescent emotionally intense or low attention temperaments and inflammation later in life. The temperament indicators in this study have been associated with outflows to both the autonomic nervous system (ANS; Spangler and Friedman, 2015) and the hypothalamic-pituitary-adrenal (HPA) axis (Mayer et al., 2014). These are fundamental stress response systems that, when dysregulated, may predispose youth to increased risk of excessive inflammation (McEwen, 2012; Miller et al., 2011). Second, researchers also have documented that temperaments comprising negative emotionality and low attention contribute to the development of adult personalities that are associated with inflammation. Low attention contributes to Conscientiousness, and negative emotionality is analogous to Neuroticism (Shiner and DeYoung, 2013). Adults who are conscientious, in that they are well organized, planful, and responsible, live longer, healthier lives. Conscientiousness predicts lower disease incidence (Goodwin and Friedman, 2006), better cognitive health (Wilson et al., 2015), lower levels of inflammatory biomarkers across the lifespan (Luchetti et al., 2014), and greater longevity (Turiano et al., 2015). Neuroticism, a tendency to experience frequent negative emotions and emotional instability, is also linked to physical health. In general, individuals high in neuroticism are at greater risk for experiencing chronic illnesses (Hampson, 2012) and early mortality (Almada et al., 1991). One hallmark of neuroticism is the tendency to experience longer lasting and more intense negative emotions when perceived stressors occur (Suls and Martin, 2005). Frequently occurring episodes of intense negative emotions are associated consistently with higher levels of inflammatory biomarkers (Renna et al., 2021). Given these relations, we expected to find associations of high emotional intensity or low attention control with absolute and worsening levels of inflammatory biomarkers during young adulthood.

We also examined a scenario whereby self-regulation abilities serve as a mediator connecting early adolescent temperaments with inflammation during young adulthood. Self-regulation is defined as a set of abilities involved in the regulation of cognition, emotion, and behavior (de Ridder et al., 2012). Young adult self-regulation differs from child temperament, in that child temperaments are simple characteristics that reflect a "style" of behavior. They become increasingly complex over time, with cognitive and social maturation and experience. Early adolescent temperaments thus provide the substrate or foundation from which more complex young adult self-regulation abilities develop (Rothbart, 2011). Deficiencies in self-regulation have long been suspected as an underlying vulnerability to health problems in adulthood. Young adults who develop and practice good self-regulation live longer; are less likely to engage in unhealthful behaviors like smoking, overeating, and drug use; are less likely to develop psychiatric disorders; and are more likely to comply with medical regimens (Moffitt et al., 2011). We expected early adolescents with temperaments characterized by intense negativity or low attention control to evince lower levels of self-regulation as young adults, which in turn we predicted would presage higher inflammatory biomarkers at ages 25 and 29, along with worsening of these biomarkers between the data points.

The study hypotheses were tested with secondary data analyses of a longitudinal cohort of rural Black youth followed from age 11 to age 29 who resided in the Southern United States. Populations of Black children and youth have been largely overlooked by temperament studies that rely on relatively small samples from middle to high socioeconomic backgrounds (Scott et al., 2016). For rural Black Americans, the young adult years have significant potential to affect inflammation. Extant studies conducted with predominately well-resourced samples characterize young adulthood as a period of positive physical and mental health (Fulmer, 2016). Young adults begin to consolidate careers and enjoy the health benefits of increased income stability. Vast racial and economic inequalities, however, exist in young adult's experiences (Silva, 2012). The influence of poverty, community disadvantage, and racial discrimination combine to render rural Black Americans' transitions to productive adult roles especially challenging and stressful (Brody, Yu, Chen et al., 2013). Job turnover rates are high, and the effects of discriminatory hiring practices make obtaining stable and satisfying employment and financial security a protracted and often demoralizing process (Brody, Yu, and Beach, 2016). Understanding whether and how preadolescent temperaments contribute to inflammation during young adulthood may provide insights into why some Black young adults are more likely to develop inflammation at older ages while others do not.

The following data were collected in the secondary data analyses. When children were 11–13 years of age, teachers provided temperament assessments. At age 25, young adults provided data on their self-regulation abilities. Peripheral blood was collected at ages 25 and 29, from which inflammatory biomarkers were quantified, using the cytokines IL-6, IL-10, and TNF-α and soluble urokinase plasminogen activator (suPAR). Proinflammatory cytokines and SuPAR are biomarkers of systemic inflammation. Both markers are released into the bloodstream under proinflammatory conditions; the blood concentrations of both are thought to reflect a person's overall level of immune activity, and both are associated with development, presence, and progression of multiple chronic diseases (Miller et al., 2011). Indeed cytokine and suPAR blood levels predict morbidity and mortality from cardiovascular disease, type

2 diabetes, cancer, infections, and early death (Hayek et al., 2020; Shields et al., 2020). While the cytokines have been studied extensively in health psychology, interest in suPAR has grown recently based in part on the hypothesis that it is less affected by acute changes in diet and health than biomarkers like CRP (Kany, Vollrath, and Relja, 2019; Rasmussen et al., 2019). Covariates associated with inflammation in past research were also assessed; these included socioeconomic (SES) risk, gender, cigarette smoking, body mass index (BMI), adverse childhood experiences (ACEs), depressive symptoms, and medication use. These variables could potentially confound associations of early adolescent temperaments or self-regulation with subsequent cytokine and suPAR levels.

2. Methods

2.1. Sample

Data for the study were drawn from the Strong African American Families Healthy Adults Project (SHAPE; Brody et al., 2013). Starting in 2001, SHAPE enrolled 667 Black children in fifth grade (mean age = 11.2 years, *SD* = 0.3) along with their primary caregivers. Families resided in rural counties of Georgia in which poverty rates are among the highest in the nation. Economically, these households were characterized as working poor. At enrollment, primary caregivers had a median household income of $1612 per month, and 42.3% lived below federal poverty thresholds. In 2009–2010, when participants were 19 years old, 500 were randomly selected, due to funding constraints, to participate in a collection of biological data. In 2015 and 2019, when participants were 25 and 29 years of age, we conducted fasting antecubital blood draws of the 391 (age 25) and 327 (age 29) participants, from which the cytokines IL-6, IL-10, TNF-α, and suPAR were assayed. The sample for the present study was composed of 307 participants (106 men and 201 women) from whom fasting antecubital blood was drawn at both ages 25 and 29 years. Compared with the original study cohort, the analytic sample had a higher percentage of female participants (65.5% vs. 52.8%); the samples were similar on the other study variables (see Table 1). Informed consent forms were completed at all data collection points. The University of Georgia's Institutional Review Board reviewed and approved all study procedures.

2.2. Measures

2.2.1. Early adolescent temperament

Three waves of data were collected from teachers when the target youth were 11, 12, and 13 years of age. Emotional intensity and low attentional control were assessed using the 7-item emotional intensity subscale and the 6-item low attentional control subscale from the short form of the Early Adolescent Temperament Inventory (Capaldi and Rothbart, 1992; Ellis and Rothbart, 2001; April; Rothbart, 2011). Each item was rated on a Likert scale ranging from 1 (*very false*) to 5 (*very true*). Emotional intensity, an indicator of negative emotionality (NE), entails a general tendency to express negative emotions such as distress and anger; a sample item is "This child usually gets very irritated when someone criticizes her/him." Alphas across waves were.90. Low attentional control, an indicator of effortful control (EC), assessed the ability to focus on a task and pursue goals in the presence of competing desires; a sample item is "This child often is in the middle of doing one thing, and then goes off to do something else, without finishing it." Alphas across waves ranged from.84 to.88. The three waves of temperament assessments were then averaged to form the emotional intensity temperament and low attention temperament indicators (Emotional intensity, rs =.47 for wave 1 with wave 2,.24 for wave 2 with wave 3, and.19 for wave 1 with wave 3; Low attention, rs =.42 for wave 1 with wave 2,.41 for wave 1 with wave 3, and.31 for wave 1 with wave 3; all *ps* <.001).

Table 1
Comparisons of Participants Included vs. Not Included in Analyses.

	With Data	Without Data		
	% or Mean (*SD*)	% or Mean (*SD*)	χ^2 *or t*	*p*
Age 11 Variables (n = 667)	(n = 307)	(n = 360)		
Gender (male)	34.5%	58.1%	36.80	.000
Parent age (in years)	37.78 (7.58)	37.69 (7.67)	0.15	.883
Parent education < high school	20.2%	19.9%	0.10	.921
Parent marital status (single)	57.5%	55.2%	0.35	.551
Family poverty status	40.3%	44.2%	0.92	.337
Parent unemployment status	20.8%	22.9%	0.41	.523
Family TANF status	6.8%	7.8%	0.21	.644
Family inadequate income	35.5%	30.3%	2.00	.157
Age 11–13 Variables (n = 667)	(n = 307)	(n = 360)		
Family SES-related risk	2.31 (1.32)	2.18 (1.37)	1.31	.192
Emotional intensity temperament	2.77 (0.66)	2.79 (0.66)	-0.26	.792
Low attention temperament	2.74 (0.65)	2.80 (0.69)	-1.22	.223
Age 25 variables (n = 408)	(n = 307)	(n = 101)		
ACEs	1.20 (1.36)	1.27 (1.60)	-0.40	.689
BMI	30.79 (8.96)	28.95 (7.51)	1.86	.063
Smoking	0.12 (0.23)	0.18 (0.24)	-1.88	.061
Depressive symptoms	12.48 (8.01)	11.80 (7.35)	0.76	.450
Medication use status	18.9%	10.9%	3.46	.063
Self-regulation	56.23 (7.81)	56.98 (7.79)	-0.84	.401
Shift-and-Persist coping	60.95 (6.40)	61.41 (5.70)	-0.64	.525
Emotion reactivity	16.13 (5.18)	15.24 (4.90)	1.52	.130
Age 25 biological variables (n = 391)	(n = 307)	(n = 84)		
Log suPAR (pg/mL)	3.33 (0.14)	3.31 (0.11)	1.15	.249
Log IL-6 (pg/mL)	0.24 (0.34)	0.17 (0.38)	1.62	.107
Log IL-10 (pg/mL)	0.15 (0.35)	0.16 (0.29)	-0.41	.684
Log TNF-α (pg/mL)	0.57 (0.12)	0.56 (0.12)	1.03	.303
Age 29 biological variables (n = 327)	(n = 307)	(n = 20)		
Log suPAR (pg/mL)	3.36 (0.13)	3.32 (0.11)	1.35	.177
Log IL-6 (pg/mL)	0.37 (0.33)	0.24 (0.25)	1.70	.090
Log IL-10 (pg/mL)	0.41 (0.19)	0.38 (0.10)	0.75	.451
Log TNF-α (pg/mL)	0.89 (0.12)	0.88 (0.11)	0.25	.806

ACEs: adverse childhood experiences; BMI: body mass index; IL: interleukin; suPAR: soluble urokinase plasminogen activator; TANF: Temporary Assistance for Needy Families; TNF: tumor necrosis factor.

2.2.2. Young adult self-regulation

At age 25, participants completed three scales assessing three aspects of self-regulation: cognitive control, self-regulated coping, and emotion regulation. For the assessment of cognitive self-control, they completed the 23-item Self-Regulation Questionnaire, which assesses future orientation, setting goals, and making plans to meet them (Brown et al., 1999). Each item was rated on a Likert-type scale ranging from 1 (*strongly disagree*) to 4 (*strongly agree*). Example items include, "Once I have a goal, I can usually plan how to reach it" and "I set goals for myself and keep track of my progress." Cronbach's alpha was.92. To assess self-regulated coping, participants completed the 17-item Shift-and-Persist Scale (Chen et al., 2015). This coping style entails a combination of shifting (accepting life for what it is and adapting to it) and persisting (enduring adversity by holding on to meaning and optimism), which, together mitigate the health impact of stressors that many low-SES youth face (Chen and Miller, 2012). Each item was rated on a Likert-type scale ranging from 1 (*not at all*) to 4 (*a lot*). Example items include, "I believe that there is a larger reason or purpose for my life" and "When something stressful happens in my life, I think about what I can learn from the situation." Cronbach's alpha was.84. To assess poor emotion regulation characterized by impulsive negative emotionality,

participants completed the 6-item Emotion Reactivity Subscale in the MacArthur Reactive Responding Scale (Taylor and Seeman, 1999). Each item was rated on a Likert-type scale ranging from 1 (*strongly disagree*) to 5 (*strongly agree*). Example items include, "I often respond quickly and emotionally when something happens" and "Sometimes I overreact to situations." Cronbach's alpha was.73. The three measures were highly correlated (*rs* = −.28 to.58; $p < .001$) and confirmatory factor analyses (CFA) model supported a single-factor model. Each indicator was standardized, and the first two indicators were summed; the score for emotion reactivity was subtracted from the summed score. Thus, high values on the self-regulation composite indicated high levels of planful cognitive self-regulation, self-regulated coping, and low levels of poor emotional self-regulation.

2.2.3. Inflammation

Two biomarkers of inflammation, proinflammatory cytokines and soluble urokinase-type plasminogen activator receptor (suPAR), were assessed at ages 25 and 29. At both of these assessments, a phlebotomist visited each participant's home in the morning to perform an antecubital blood draw. To minimize circadian variation, venipuncture was performed between 8:00 am and 10:00 am. Participants fasted for 8 h beforehand to minimize dietary influences. Participants were instructed to contact the research team and reschedule the home visit if they were ill. Blood was drawn into Serum Separator tubes (Becton, Dickinson and Company, Franklin Lakes, NJ, USA). Specimens were centrifuged on site at 1500g for 20 min. The serum was harvested, divided into aliquots, and immediately frozen on dry ice. Upon arrival at the lab, it was placed in storage at − 80 °C until the end of the project. Both suPAR and the cytokines interleukin (IL)− 6, IL-10, and tumor necrosis factor –α (TNF-α) were assayed in batch with technicians blind to other participant data. The cytokines were measured in triplicate via four-plex immunoassay on a microfluidic platform (Simple Plex; Protein Simple). IL-8 was also assayed, but it was not significantly associated with the other cytokines (rs = −.07 with IL-6 and.07 with IL-10). And factor analyses supported a two-factor model with IL-6 (.73), IL-10 (.67), and TNF-α (.78) loading on one factor and IL-8 (.94) loading on the other factor. Thus, IL-8 was not included in the cytokine composite. A newer inflammatory biomarker, suPAR also has emerged as a predictor of cardiovascular disease, diabetes, and all-cause mortality (Botha et al., 2015), and has been speculated to reflect vascular inflammation (Lyngbaek et al., 2013). suPAR was measured in duplicate by immunoassay (Human Quantikine ELISA; R&D Systems). Assays were run after each wave of data collection, so each subject's specimens were assayed on different plates.

The lower limits of detection for the cytokines were 0.14 pg/mL for IL-10, 0.26 pg/mL for IL-6, and 0.28 pg/mL for TNF-α, and for suPAR was 33 pg/mL. Across runs, the average intra-assay coefficients of variation were 3.1% (IL-6), 3.6% (IL-10), 2.9% (TNF-α), and 1.5% (suPAR). The inter-assay coefficients of variation were 6.4% (IL-6), 7.0% (IL-10), 5.8% (TNF-α), and 1.1% (suPAR). When a sample value was above the highest standard, we diluted and re-assayed. All of the inflammatory biomarkers of IL-6, IL-10, TNF-α, and suPAR were skewed and/or kurtotic, so we normalized their distributions with log-10 transformations. The logged values of IL-6, IL-10, and TNF-α were then standardized and summed to form a composite score of cytokines. Changes of cytokine and suPAR levels across the age 25 and 29 assessments were computed as residual scores using a regression procedure in which biomarkers levels at age 29 were regressed on levels at age 25 (Cohen, West, and Aiken, 2014).

2.2.4. Covariates

Youth gender was dummy coded; male participants were coded 1 and female participants were coded 0. Family SES-related risk at ages 11–13 was measured as the sum of six indicators (0 if absent, 1 if present; see Brody et al., 2013): current family poverty, primary caregiver's noncompletion of high school or an equivalent, primary caregiver current unemployment, single-parent family structure, current receipt of Temporary Assistance for Needy Families, and income rated by the caregiver as inadequate to meet all needs. The SHAPE cohort was initially recruited for a randomized controlled trial of a family-oriented intervention to prevent youth behavior problems and substance abuse. Participation in the intervention was not associated with any of the study outcomes. To minimize any residual confounding, however, we included a dichotomous covariate reflecting intervention condition (treatment vs. control) in all models. At age 25, BMI, smoking, ACEs, depressive symptoms, and medication use were assessed because each has been associated with cytokine and suPAR levels. Trained research staff measured weight using a standard home scale and height using a tape measure in a standardized way. BMI was calculated as weight in kilograms divided by the square of height in meters. Participants reported their past-month cigarette use on a rating scale ranging from 0 = *none at all* to 7 = *more than 2 packs a day*. Past-month cigarette use was log-transformed because its distribution was skewed. Participants reported ACEs on the Adverse Childhood Experiences questionnaire (Felitti et al., 1998). An ACEs score was calculated by summing dichotomized *yes/no* responses across 10 ACEs categories indicating the presence or absence of particular adversities that participants may have experienced before the age of 18 years: living with someone who was mentally ill or depressed; was a problem drinker, an alcoholic, or a user of street drugs; or went to prison; having parents who were separated or divorced; witnessing domestic violence; experiencing physical neglect or emotional neglect; and experiencing physical abuse, verbal abuse, or sexual abuse. At age 25, participants also reported their depressive symptoms using the Center for Epidemiologic Studies Depression scale (CES–D; Radloff, 1977), Alpha was.89. They also reported whether they used any prescribed medications (no = 0, yes =1).

2.2.5. Plan of analyses

Multiple linear regression models were executed to test the study hypotheses. The first set of models was designed to determine whether early adolescent temperaments at ages 11–13 were associated with suPAR and cytokine levels at ages 25 and 29, as well as with changes in them between these two data points. The second set of models were designed to determine whether young adult self-regulation at age 25 would mediate the association between early adolescent temperaments and young adult inflammation outcomes. Mediation was tested using regression-based mediation effect analyses procedures (Hayes, 2018). To do this, regression coefficients were calculated for the association between early adolescent temperaments and self-regulation (path a), and for the association between self-regulation and each inflammation outcome (path b). The indirect effect in which self-regulation serves as a mediator connecting early adolescent temperaments to each young adult inflammation outcome was quantified as the product of the two regression coefficients (a × b). In addition, nonparametric bootstrapping was used to obtain the bias-corrected and accelerated confidence intervals (BCA) of parameter estimates for significance testing (Preacher et al., 2007). The parameter estimate was calculated 5000 times using random sampling with replacement to build a sampling distribution. In all models, gender, intervention status, and family SES risk at ages 11–13, and young adult BMI, smoking, and ACEs at age 25, were included as covariates. We used G*Power to obtain power estimates for the hypothesized models described above. For the study sample of 307, power estimates exceeded.80 for detecting effect sizes as small as.026. Thus, the study had sufficient power to test the planned hypotheses. All analyses were conducted using IBM SPSS 27 and the statistical macro package PROCESS (Hayes, 2018).

3. Results

3.1. Early adolescent temperaments at ages 11–13 and inflammation outcomes at ages 25 and 29 years

Bivariate correlations among the study variables are presented in Table S1. Tables 2 and 3 present regression models that were used to determine whether an early adolescent emotionally intense temperament (Table 2) or a low attention temperament (Table 3) predicted suPAR and cytokine levels and changes in them from ages 25–29, above and beyond the effects of potential demographic and biobehavioral confounds. As can be seen, an early adolescent emotionally intense temperament was a significant predictor of suPAR levels at age 29 (b =.027, 95% CI [.001,.053], β = .133, p = .041) and a significant predictor of cytokine levels at age 29 (b =.649, 95% CI [.187, 1.111], β = .184, p = .006). An early adolescent low attention temperament was also a significant predictor of suPAR levels at age 29 (b =.025, 95% CI [.004,.046], β = .120, p = .018).

The second set of models examined whether early adolescent temperaments were associated with worsening suPAR and cytokine levels between ages 25 and 29 years. As Tables 2–3 illustrate, early adolescent temperaments were not associated with worsening levels of suPAR from age 25–29 years (emotional intensity temperament: b =.013, 95% CI [−.014,.039], β = .074, p = .346; low attention temperament: b =.015, 95% CI [−.003,.033], β = .087, p = .101). The mean levels of log-transformed suPAR (pg/mL) at age 25 was 3.33 (SD = 0.14) and at age 29 was 3.36 (SD = 0.13), indicating suPAR levels were stable and evinced little change across the four years that separated the data points. The analysis for cytokines showed a different pattern. An early adolescent emotionally intense temperament predicted worsening cytokine levels over time (b =.519, 95% CI [.073,.966], β = .156, p = .023). A low attention temperament was not associated with worsening of cytokine levels over time (b =.255, 95% CI [−.122,.633], β = .075, p = .184). The mean cytokine levels that comprised the cytokine composite at age 25 years were: log-transformed IL-6 (pg/mL), 0.24 (SD = 0.34); log-transformed IL-10 (pg/mL), 0.15 (SD = 0.35); and log-transformed TNF-α (pg/mL), 0.57 (SD = 0.12). The mean cytokine levels at age 29 years were: log-transformed IL-6 (pg/mL), 0.37 (SD = 0.33); log-transformed IL-10 (pg/mL), 0.41 (SD = 0.19); and log-transformed TNF-α (pg/mL), 0.89 (SD = 0.12). These means show that cytokine levels that comprised the cytokine composite increased, making it possible to detect associations between early adolescent temperament and changes in cytokines. Again, these effects were over and above the contribution of demographic and biobehavioral confounds. Finally, we examined whether the study findings survived correction for multiple comparisons and found only the association between an emotionally intense temperament and cytokine levels at age 29 survived. This limitation suggests the study results should be considered preliminary until they are substantiated in studies with a larger sample size and more thorough assessments of preadolescent temperament.

3.2. Does young adult self-regulation mediate associations between early adolescent temperaments and young adult inflammation outcomes?

Regression models tested the hypothesis that young adult self-regulation mediated significant associations reported previously between early adolescent temperaments and inflammation outcomes. The results of regression models suggested that an early adolescent emotionally intense temperament was not associated with young adult self-regulation (b = −.312, 95% CI [−.725,.101], β = −.087, p = .138). The results of the regression models presented in Table 4 shows that young adult self-regulation did not mediate associations between an emotionally intense temperament and inflammation outcomes.

An early adolescent low-attention temperament was negatively associated with young adult self-regulation (b = −.557, 95% CI [−.968, −.145], β = −.152, p = .008). However, the results of the regression models presented in Table 5 show that young adult self-regulation did not mediate associations between a low attention temperament and suPAR levels at ages 25 and 29. Although a low attentional control temperament was associated with lower young adult self-regulation, self-regulation was not associated significantly with suPAR levels at age 25 (b =.003, 95% CI [−.004,.011], β = .058, p = .391) or at age 29 (b = −.002, 95% CI [−.008,.003], β = −.043, p = .408). A different pattern of results emerged when the regression models were applied to the associations reported previously between a low attention temperament and cytokine levels at age 29 and changes in cytokines from ages 25–29 years. The results of the regression models presented in Table 5 show that young adult self-regulation was negatively associated with cytokine

Table 2
Early Adolescent Emotional Intensity Temperament with Young Adult suPAR and Cytokine Levels.

	suPAR (age 25)			suPAR (age 29)			Changes in suPAR (from ages 25–29)		
Predictors	*b*	[95% CI]	β	*b*	[95% CI]	β	*b*	[95% CI]	β
1. Gender, male	-.033 *	[− .065, − .000]	-.113	-.043 *	[− .077, − .009]	-.153	-.026	[− .059,.007]	-.108
2. Intervention, SAAF	-.003	[− .033,.028]	-.009	.001	[− .029,.031]	.004	.003	[− .023,.028]	.011
3. Family SES risk (ages 11–13)	.003	[− .010,.015]	.025	.001	[− .010,.013]	.012	-.000	[− .011,.011]	-.003
4. BMI (age 25)	.005 * **	[.003,.007]	.318	.005 * **	[.004,.006]	.328	.002 * **	[.001,.004]	.185
5. Smoking (age 25)	.047	[− .014,.107]	.081	.072 *	[.004,.139]	.126	.047	[− .017,.111]	.098
6. ACEs (age 25)	-.006	[− .017,.005]	-.060	-.001	[− .013,.010]	-.011	.002	[− .007,.011]	.025
7. Emotional intensity temperament (ages 11–13)	.027	[− .002,.056]	.131	**.027** *	[.001,.053]	.133	.013	[− .014,.039]	.074
	Cytokines (age 25)			Cytokines (age 29)			Changes in Cytokines (from ages 25–29)		
Predictors	*b*	[95% CI]	β	*b*	[95% CI]	β	*b*	[95% CI]	β
1. Gender, male	-.220	[− .767,.327]	-.048	.033	[− .521,.587]	.007	.113	[− .413,.638]	.025
2. Intervention, SAAF	-.175	[− .685,.334]	-.040	.279	[− .271,.830]	.059	.343	[− .180,.867]	.077
3. Family SES risk (ages 11–13)	.075	[− .114,.264]	.046	.164	[− .009,.339]	.093	.137	[− .031,.306]	.083
4. BMI (age 25)	.050 * **	[.024,.077]	.208	.059 * **	[.034,.084]	.227	.041 * **	[.017,.064]	.167
5. Smoking (age 25)	.514	[− .690, 1.719]	.056	.403	[− 1.017, 1.823]	.041	.216	[− 1.171, 1.603]	.023
6. ACEs (age 25)	-.119	[− .283,.044]	-.075	-.016	[− .242,.209]	-.009	.027	[− .186,.240]	.017
7. Emotional intensity temperament (ages 11–13)	.358	[− .032,.748]	.109	**.649** * *	[.187, 1.111]	.184	**.519** *	[.073,.966]	.156

N = 307; b = unstandardized regression coefficient; CI = confidence interval; β = standardized regression coefficient. SAAF: Strong African American Families program; BMI: Body mass index; ACEs: Adverse childhood experiences. Family SES risk at ages 11–13, gender, intervention status, and BMI, smoking, and ACEs at age 25 were covariates.
$^{+}p < .10$. * $p < .05$. * * $p < .01$. * ** $p < .001$.

Table 3
Early Adolescent Low Attention Temperament with Young Adult suPAR and Cytokine Levels.

	suPAR (age 25)			suPAR (age 29)			Changes in suPAR (from ages 25–29)		
Predictors	*b*	[95% CI]	β	*b*	[95% CI]	β	*b*	[95% CI]	β
1. Gender, male	-.036 *	[− .071, − .002]	-.127	-.049 * *	[− .083, − .015]	-.174	-.030	[− .063,.003]	-.126
2. Intervention, SAAF	-.001	[− .032,.030]	-.003	.003	[− .027,.033]	.011	.003	[− .023,.029]	.014
3. Family SES risk (ages 11–13)	.003	[− .010,.015]	.027	.001	[− .010,.012]	.010	-.001	[− .011,.010]	-.006
4. BMI (age 25)	.005 * **	[.003,.007]	.314	.005 * **	[.003,.006]	.322	.002 * **	[.001,.004]	.180
5. Smoking (age 25)	.043	[− .017,.103]	.074	.067	[− .000,.134]	.117	.044	[− .020,.107]	.091
6. ACEs (age 25)	-.006	[− .017,.006]	-.056	-.001	[− .013,.011]	-.011	.002	[− .007,.011]	.023
7. Low attention temperament (ages 11–13)	.018	[− .003,.040]	.087	**.025** *	[.004,.046]	.120	.015	[− .003,.033]	.087
	Cytokines (age 25)			Cytokines (age 29)			Changes in Cytokines (from ages 25–29)		
Predictors	*b*	[95% CI]	β	*b*	[95% CI]	β	*b*	[95% CI]	β
1. Gender, male	-.270	[− .828,.288]	-.059	-.025	[− .605,.556]	-.005	.073	[− .468,.615]	.016
2. Intervention, SAAF	-.151	[− .665,.363]	-.034	.324	[− .228,.876]	.068	.379	[− .143,.901]	.085
3. Family SES risk (ages 11–13)	.078	[− .115,.271]	.047	.175	[− .001,.350]	.099	.147	[− .024,.317]	.088
4. BMI (age 25)	.050 * **	[.022,.077]	.204	.058 * **	[.0343,.082]	.223	.040 * **	[.017,.063]	.163
5. Smoking (age 25)	.465	[− .771, 1.701]	.050	.333	[− 1.118, 1.784]	.034	.164	[− 1.240, 1.567]	.018
6. ACEs (age 25)	-.113	[− .284,.057]	-.071	.001	[− .234,.236]	.001	.042	[− .177,.261]	.026
7. Low attention temperament (ages 11–13)	.242	[− .162,.646]	.072	.343	[− .049,.735]	.095	.255	[− .122,.633]	.075

N = 307; b = unstandardized regression coefficient; CI = confidence interval; β = standardized regression coefficient. SAAF: Strong African American Families program; BMI: Body mass index; ACEs: Adverse childhood experiences. Family SES risk at ages 11–13, gender, intervention status, and BMI, smoking, and ACEs at age 25 were covariates.
$^{+}p < .10$. * $p < .05$. * * $p < .01$. * ** $p < .001$.

Table 4
Early Adolescent Emotional Intensity Temperament and Young Adult Self-Regulation with suPAR and Cytokine Levels.

	suPAR (age 25)			suPAR (age 29)			Changes in suPAR (from ages 25–29)		
Predictors	*b*	[95% CI]	β	*b*	[95% CI]	β	*b*	[95% CI]	β
1. Gender, male	-.034 *	[− .067, − .000]	-.117	-.042 *	[− .077, − .008]	-.150	-.025	[− .058,.009]	-.103
2. Intervention, SAAF	-.004	[− .035,.028]	-.013	.002	[− .029,.033]	.007	.004	[− .023,.030]	.016
3. Family SES risk (ages 11–13)	.003	[− .010,.015]	.028	.001	[− .011,.012]	.009	-.001	[− .011,.010]	-.007
4. BMI (age 25)	.005 * **	[.003,.007]	.318	.005 * **	[.003,.006]	.328	.002 * **	[.001,.004]	.186
5. Smoking (age 25)	.052	[− .011,.116]	.090	.068	[− .001,.136]	.118	.040	[− .027,.106]	.083
6. ACEs (age 25)	-.005	[− .016,.006]	-.048	-.002	[− .014,.010]	-.022	.000	[− .008,.009]	.005
7. Emotional intensity temperament (ages 11–13)	.028	[− .002,.057]	.135	.026 *	[.000,.052]	.129	.011	[− .016,.038]	.066
8. Self-regulation (age 25)	.003	[− .004,.011]	.056	-.003	[− .008,.003]	-.047	-.004	[− .010,.001]	-.093
	Cytokines (age 25)			Cytokines (age 29)			Changes in Cytokines (from ages 25–29)		
Predictors	*b*	[95% CI]	β	*b*	[95% CI]	β	*b*	[95% CI]	β
1. Gender, male	-.212	[− .761,.337]	-.046	.066	[− .495,.628]	.014	.143	[− .390,.676]	.031
2. Intervention, SAAF	-.168	[− .682,.347]	-.038	.313	[− .229,.855]	.066	.374	[− .142,.889]	.084
3. Family SES risk (ages 11–13)	.073	[− .118,.263]	.044	.154	[− .020,.327]	.087	.127	[− .040,.295]	.077
4. BMI (age 25)	.051 * **	[.024,.077]	.208	.059 * **	[.035,.084]	.229	.041 * **	[.018,.064]	.168
5. Smoking (age 25)	.471	[− .746, 1.687]	.051	.216	[− 1.225, 1.656]	.022	.045	[− 1.361, 1.451]	.005
6. ACEs (age 25)	-.130	[− .299,.040]	-.081	-.060	[− .281,.16`]	-.035	-.013	[− .222,.195]	-.008
7. Emotional intensity temperament (ages 11–13)	.350	[− .040,.740]	.106	.613 *	[.148, 1.078]	.174	.486 *	[.039,.933]	.147
8. Self-regulation (age 25)	-.027	[− .127,.073]	-.030	-.115 *	[− .219, − .011]	-.118	-.105 *	[− .199, − .012]	-.114

N = 307; b = unstandardized regression coefficient; CI = confidence interval; β = standardized regression coefficient. SAAF: Strong African American Families program; BMI: Body mass index; ACEs: Adverse childhood experiences. Family SES risk at ages 11–13, gender, intervention status, and BMI, smoking, and ACEs at age 25 were covariates. *p < .05. * *p < .01. * **p < .001.

levels at age 29 ($b = -.120$, 95% CI [−.226, −.013], β = −.1223, $p = .028$), as well as worsening cytokine levels across ages 25–29 years ($b = -.110$, 95% CI [−.206, −.014], β = −.119, $p = .025$). The indirect effect relating a low attention control temperament to cytokine levels at age 29 and worsening cytokine levels across ages 25–29 years, via self-regulation at age 25, was significant: indirect effect = .067, 95% CI [.004,.158]; standardized indirect effect = .019 for cytokine levels at age 29; indirect effect = .061, 95% CI [.004,.142]; standardized indirect effect = .018 for worsening cytokine levels across ages 25–29 years; see Fig. S1.

3.3. Additional analyses

Additional analyses were conducted with age 25 participants' depressive symptoms and medication use status as additional covariates. The results of associations between early adolescent temperaments and young adult cytokine and suPAR levels at ages 25 and 29, as well as worsening cytokine levels across ages 25–29 years reported in the preceding paragraph did not change (see Table S2). However, the association between an early adolescent low attention temperament and young adult self-regulation was non-significant after controlling for age 25 depressive symptoms (see Table S3). The multicollinearity between self-regulation and depressive symptoms ($r = .56$, $p < .001$) most likely

Table 5
Early Adolescent Low Attention Temperament and Young Adult Self-Regulation with suPAR and Cytokine Levels.

	suPAR (age 25)			suPAR (age 29)			Changes in suPAR (from ages 25–29)		
Predictors	*b*	[95% CI]	β	*b*	[95% CI]	β	*b*	[95% CI]	β
1. Gender, male	-.038 *	[− .074, − .002]	-.132	-.048 * *	[− .083, − .014]	-.170	-.028	[− .062,.006]	-.118
2. Intervention, SAAF	-.002	[− .033,.030]	-.006	.004	[− .027,.034]	.013	.004	[− .022,.031]	.019
3. Family SES risk (ages 11–13)	.003	[− .009,.016]	.030	.001	[− .011,.012]	.008	-.001	[− .011,.010]	-.010
4. BMI (age 25)	.005 * **	[.003,.007]	.313	.005 * **	[.003,.006]	.323	.002 * **	[.001,.004]	.182
5. Smoking (age 25)	.048	[− .015,.111]	.083	.063	[− .006,.132]	.111	.037	[− .028,.103]	.078
6. ACEs (age 25)	-.004	[− .015,.007]	-.044	-.002	[− .014,.010]	-.020	.000	[− .008,.009]	.004
7. Low attention temperament (ages 11–13)	.020	[− .003,.043]	.096	.024 *	[.003,.045]	.114	.013	[− .006,.032]	.074
8. Self-regulation (age 25)	.003	[− .004,.011]	.058	-.002	[− .008,.003]	-.043	-.004	[− .010,.001]	-.088
	Cytokines (age 25)			Cytokines (age 29)			Changes in Cytokines (from ages 25–29)		
Predictors	*b*	[95% CI]	β	*b*	[95% CI]	β	*b*	[95% CI]	β
1. Gender, male	-.258	[− .821,.305]	-.057	.030	[− .560,.619]	.006	.123	[− .428,.674]	.027
2. Intervention, SAAF	-.144	[− .662,.374]	-.032	.357	[− .188,.901]	.075	.409	[− .106,.924]	.091
3. Family SES risk (ages 11–13)	.076	[− .118,.270]	.046	.166	[− .009,.340]	.094	.138	[− .031,.307]	.083
4. BMI (age 25)	.050 * **	[.022,.077]	.205	.059 * **	[.034,.083]	.225	.041 * **	[.018,.064]	.166
5. Smoking (age 25)	.424	[− .818, 1.667]	.046	.152	[− 1.319, 1.623]	.015	-.002	[− 1.426, 1.421]	-.000
6. ACEs (age 25)	-.123	[− .298,.052]	-.077	-.043	[− .271,.186]	-.025	.002	[− .211,.215]	.001
7. Low attention temperament (ages 11–13)	.227	[− .180,.633]	.068	.276	[− .122,.674]	.077	.194	[− .188,.576]	.057
8. Self-regulation (age 25)	-.027	[− .128,.074]	-.029	-.120 *	[− .226, − .013]	-.122	-.110 *	[− .206, − .014]	-.119

N = 307; b = unstandardized regression coefficient; CI = confidence interval; β = standardized regression coefficient. SAAF: Strong African American Families program; BMI: Body mass index; ACEs: Adverse childhood experiences. Family SES risk at ages 11–13, gender, intervention status, and BMI, smoking, and ACEs at age 25 were covariates. *p < .05. * *p < .01. * **p < .001.

contributed to this finding. Thus, the indirect effect relating an early adolescent low attention temperament to young adult cytokine outcomes via self-regulation became non-significant. Finally, for interested readers, we also present the results of unadjusted models (without any covariates) in all the online supplemental tables (see Tables S2-S5).

4. Discussion

Excessive inflammation plays an important role in chronic illness, which costs Americans billions of dollars in health care expenses (Fedewa et al., 2014). All told, excessive inflammation is involved in at least eight of the top ten leading causes of death in the United States (Hoyert and Xu, 2012). For these reasons, understanding the early origins of variation in inflammation outcomes later in life is important for basic science and public health practices. Research to date has focused on the ways in which exposures to ACEs - emotional, physical, and sexual abuse; socioeconomic disadvantage; and bullying from peers - are associated with higher levels of systemic inflammation later in life. The hypotheses tested in this paper took a different tack by examining whether, and how, early adolescent's temperaments are associated with inflammatory biomarkers years later, during young adulthood.

The study findings indicated that higher levels of emotional intensity were prospectively associated with higher suPAR and cytokine levels at age 29 and increases in cytokine levels from age 25–29 years. Low attentional control was also prospectively associated with suPAR at age 29. It is not clear why emotional intensity or low control temperaments were not associated with suPAR and cytokine levels at age 25. These findings may reflect how stressors associated with each temperament dimension activate stress-related autonomic and endocrine pathways across adolescence and young adulthood with downstream consequences for inflammation. This scenario suggests a cumulative process with the consequences for inflammation becoming more evident toward the end of the third decade of life. With that said, we refine this hypothesis in the next paragraph.

The aforementioned longitudinal relations are consistent with a developmental scenario in which emotionally intense or low attention temperaments provide a substrate for adult levels of neuroticism and conscientiousness, respectively. Adults who are high on neuroticism and, or, low on conscientiousness have been found to evince higher levels of inflammatory markers across the lifespan (Luchetti et al., 2014; Renna et al., 2021). One pathway that may explain these associations is how people high on neuroticism or low on conscientiousness react to stressful situations. Inflammation has been linked to consequences of heightened stress responses which could result from a tendency to perceive situations as threatening (Miller et al., 2011). Adults high in neuroticism appraise stressors as more severe and see stressors as a greater threat to themselves (Penley and Tomaka, 2002). Adults low in conscientiousness appraise stressors as more severe and perceive themselves as being less able to handle them competently (Gartland et al., 2012; 2014). Over time, repeated exposures to frequent stressors among persons high on neuroticism or low on conscientiousness may cause persistent activation of stress-response systems, in particular the sympathetic nervous system and the hypothalamic-pituitary adrenocortical axis. The hormonal products of these systems, glucocorticoids and catecholamines, contribute to higher levels of inflammation in tissues and organs (McEwen and Stellar, 1993). Future research should explicitly test these hypotheses. We were unable to do so here because we did not assess adult levels of neuroticism or conscientiousness and stress hormone output. A follow-up study with multiple waves of exposure to frequent stressors, assessments of neuroticism and conscientiousness, and hormonal data would be ideally suited to identifying relationships between preadolescent temperaments and young adult inflammatory activity.

We examined the possibility that young adult self-regulation abilities served as a mediator connecting early adolescent temperament to later inflammation. Although superior self-regulation predicts better academic achievement, relationship quality, financial and career success, and lifespan health, as well as later mortality (Bandura, 2005; Moffitt et al., 2011), no empirical attention has been given to self-regulation as a conduit from early adolescent temperaments to inflammation. The study results suggested that low attentional control was associated with lower levels of adult self-regulation which, in turn, was associated with higher cytokine levels at age 29 as well as increases in cytokines from age 25 to age 29 years. These findings are consistent with other findings which describe how early adolescent levels of low attentional control provide a substrate for the development of complex self-regulation abilities during adulthood (Rothbart, 2011). These results extend previous research by showing that individual differences in attentional control temperament

carry forward to self-regulatory processes in adulthood and result in consequences across health and other domains (Moffitt et al., 2011). In future work, researchers should replicate these findings and extend them by addressing protective processes that promote young adult self-regulation among those who evince low attentional control during early adolescence.

The present study is the first to document that early adolescent temperaments forecast increases over time in cytokines; similar results did not emerge for suPAR. One reason for the null finding for suPAR was its relative stability across the study data points, making the detection of an association between early adolescent temperament and subsequent increases in suPAR unlikely. We are reluctant to read too much into this finding other than to say that suPAR has been studied in clinical populations with serious diseases (Rasmussen et al., 2016), and few study participants had those conditions. Rasmussen and colleagues (Rasmussen et al., 2020, 2019) speculated that cytokines reflect both chronic and acute disease states, whereas suPAR may be a more stable marker of chronic inflammation. The present results were consistent with this conjecture given the stability of suPAR across ages 25–29 years and increases in cytokines across the same period. That suPAR evinced such stability was somewhat surprising given the array of stressors that Black young adults have to contend with (Chen, Brody, and Miller, 2022). Poverty, community disadvantage, and discriminatory hiring practices combine to render transitions to productive adult roles especially challenging and chronically stressful (Brody, Yu, and Beach, 2016). Clearly, further research is needed to determine whether findings regarding the expression of inflammatory markers from populations of European descent generalize to Black and other minority populations.

It is notable, with one exception, that the study findings emerged over and beyond the effects of potential confounds (SES risk, gender, smoking, BMI, ACEs, depressive symptoms, and medication use). In follow-up analyses (available from the second author), we considered the possibility that the study covariates, considered separately - SES risk, smoking, BMI, depressive symptoms, ACEs, and medication use - would serve as mediators or moderators connecting early adolescent temperaments and later inflammation. No evidence supported any of these conjectures. That these covariates did not mediate or condition longitudinal associations between young adolescent temperaments and inflammatory activity was surprising. Clearly, further research is needed to elucidate the pathways, beyond the self-regulatory pathway identified in this study, which explain longitudinal associations between preadolescent temperaments and important inflammatory outcomes.

Finally, the study sample, rural Black children from the southern United States, are among the most disadvantaged populations in the United States in terms of life expectancy (Singh and Siahpush, 2014), a consequence of morbidity from chronic diseases of aging (CDAs) including type 2 diabetes, coronary heart disease, and stroke (Hartley, 2004). Emerging evidence suggests these CDAs are conditions that develop over the lifespan, with excessive inflammation playing a key role in their pathogenesis (Brody et al., 2016). Black children seldom have been included in child temperament studies. This study was designed to examine the ways in which early adolescent temperaments influence inflammation years later, during young adulthood. Additional waves of data collection will allow us to link early adolescent temperaments to inflammation and adult indicators of CDAs.

4.1. Strengths and limitations

A major strength of the study was the 18-year, multiple-wave, multiple-informant research design used to determine whether and how Black children's temperaments are associated with absolute and worsening levels of multiple inflammation measures in young adulthood. To avoid issues raised about method variance, different teachers provided data on early adolescent temperaments across three years, and young adults provided data on self-regulation abilities. Recent research demonstrates that, compared with parent and child reports of non-cognitive skills, teacher reports better predict children's later behavior and socioemotional functioning (Feng et al., 2022). Also, a stringent set of covariates associated with inflammation in past research were also assessed to avoid confounding associations of early adolescent temperaments or self-regulation with subsequent suPAR and cytokine levels. In addition to these strengths, limitations to this study also need to be considered. First, measurement of temperament associations with inflammation later in life would benefit from the inclusion of additional indicators of negative emotionality and effortful control. A small number of temperament indicators were included in this study to minimize burden, a limitation typically associated with large-scale longitudinal studies. It would also be valuable to supplement the temperament assessments in this study with other assessment methods such as behavioral observations (Kopala-Sibley et al., 2018). Second, the participants were Black young adults living in rural areas of the southeastern United States, so generalizability is limited. Replications across samples of more varied race, ethnicity, and socioeconomic status should be included in future studies. Third, given the observational nature of the data, we were unable to make definitive claims about causal relations among the study variables. Early adolescent temperaments show stability, and theoretical models of child temperaments and health suggest that temperaments predict self-regulation and adult health outcomes rather than the reverse (Rothbart, 2011). In future work, researchers should extend these findings by examining direct and indirect associations of early adolescent temperaments with other health-related outcomes, including neuroendocrine outflows and cardiometabolic precursors to cardiovascular disease and diabetes. These limitations notwithstanding, the results provide clues about the ways in which early adolescent temperaments carry forward to influence the development of health problems during adulthood.

5. Conclusion

In sum, this study is unique in following a sample of Black youth using a longitudinal design spanning 18 years to test hypotheses about potential associations between early adolescent temperaments and inflammation later in life. The results highlight pathways that underlie health risks associated with early adolescent temperaments. The study findings illustrate that emotionally intense, low-attention temperaments presage higher and worsening inflammation levels across young adulthood.

Declarations of interest

No conflict of interests was ever mentioned in the submission process or in the manuscript.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.psyneuen.2023.106077.

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