Brain, Behavior, and Immunity 116 (2024) 114–123

Contents lists available at ScienceDirect

Brain Behavior and Immunity

journal homepage: www.elsevier.com/locate/ybrbi

Responsive parental support buffers the link between chronic stress and cardiometabolic risk among adolescents

Phoebe H. Lam [a,*], Edith Chen [b,c], Tao Jiang [c], Hee Moon [c], Veronica Passarelli [c], Jungwon Kim [c], Gregory E. Miller [b,c]

[a] Department of Psychology, Carnegie Mellon University, Pittsburgh, PA, USA
[b] Department of Psychology, Northwestern University, Evanston, IL, USA
[c] Institute for Policy Research, Northwestern University, Evanston, IL, USA

ARTICLE INFO

Keywords:
Parental support
Timing
Adolescence
Inflammation
Metabolic syndrome

ABSTRACT

Youth exposed to chronic stress exhibit increased cardiometabolic risk which parental social support may attenuate. Notably, theories emphasize that support should be delivered responsively for it to exert buffering effects, but this has not been thoroughly tested empirically. This study examined whether timing of support is an important but unrecognized element of responsiveness during adolescence in buffering the link between chronic stress and cardiometabolic risk. Participants were 242 adolescents aged 15 years (63 % female, 38 % Black). Adolescents completed assessments of chronic stress (Life Stress Interview), and trained personnel collected anthropometric measures and blood samples to assess cardiometabolic risk (reflected in low-grade inflammation and metabolic syndrome). Adolescents also completed an eight-day diary assessment to report daily stressor exposure and parental support. Using the diary data, responsiveness of parental support was operationalized as the within-individual difference in parental support received on stressor (vs. non-stressor) days, such that increased parental support on stressor days reflected more timely support. Results suggest that responsive parental support buffered the link between chronic stress and cardiovascular risk. Specifically, chronic stress was associated with greater risk only when parental support was not temporally aligned with stress exposure, but this association was not observed among adolescents who received timely parental support. These findings shed light on why parental support may not always exert buffering effects during adolescence, highlighting the importance of taking a developmental approach to understanding protective effects.

Exposure to psychosocial stress during the first two decades of life, particularly when it is chronic in nature, has been prospectively linked to poorer cardiometabolic health in adulthood (Godoy et al., 2021; Halfon et al., 2012). For example, children and adolescents exposed to socioeconomic disadvantage, peer victimization, or adverse family environment are more likely to develop type 2 diabetes, coronary heart disease, and stroke as adults (Galobardes et al., 2006; Suglia et al., 2008, 2021; Tamayo et al., 2010). Although cardiometabolic health problems do not clinically manifest until adulthood, pathogenic processes are thought to begin much earlier in life (Adler and Stewart, 2010; Miller, Chen, et al., 2011). Indeed, youth exposed to chronic stress exhibit preclinical signs of cardiometabolic disease, including insulin resistance, higher blood pressure, central adiposity, and chronic inflammation (Brady and Matthews, 2006; Goodman et al., 2007; Pervanidou and Chrousos, 2011). Of note, recent evidence suggests that the link between psychosocial stress and disease risk strengthens across the lifecourse (Chiang et al., 2022; Lam et al., 2021), implying it would be valuable to identify protective factors earlier in life that might curtail accumulating disease risks. Investigating this question during adolescence may be particularly effective because sensitivity to social environments increases during this stage (Blakemore and Mills, 2014; Somerville, 2013) and because cardiovascular risk markers begin to have prognostic value around adolescence, relative to childhood (Juhola et al., 2011; Juonala et al., 2010).

What factors during adolescence may confer protection against the negative health impacts of chronic stress? Adolescence is marked by rapid and drastic changes biologically (e.g., puberty introduces changes to multiple body systems), psychosocially (e.g., increased autonomy), and behaviorally (e.g., emergence of risky behaviors). During such time of flux, family relationships may serve as a stable source of comfort or

* Corresponding author at: 4909 Frew St, Suite 354E, Pittsburgh, PA 15213, USA.
E-mail address: phoebelam@cmu.edu (P.H. Lam).

https://doi.org/10.1016/j.bbi.2023.11.027
Received 15 July 2023; Received in revised form 31 October 2023; Accepted 23 November 2023
Available online 3 December 2023

support that can buffer adolescents from the negative impacts of stress (Chen et al., 2017). Indeed, although adolescents spend more time with peers and put stronger emphasis on peers' opinions and expectations (Brown and Larson, 2009), conceptual and empirical work points to the importance of family relationships in shaping adolescent health (Bai et al., 2017; Repetti et al., 2012), finding that higher parental/caregiver (hereafter, "parental") warmth is associated with less substance use in adolescence and better inflammatory profiles (Collins and Laursen, 2004; Klevens and Hall, 2014; Manczak et al., 2018). With respect to stress-buffering effects, emerging evidence suggests that higher parental support may attenuate the stress-health link. For example, greater contextual stress (e.g., poverty and discrimination) was associated with worse physical health profiles (e.g., higher allostatic load or fasting epigenetic aging) only among adolescents who had lower parental support, but not among those with higher parental support (Brody et al., 2014, 2016). In addition, studies have found that adolescents with higher parental responsivity had more adaptive cortisol responses to a standardized laboratory stressor (Cameron et al., 2017; Hackman et al., 2013; Kuhlman et al., 2017), suggesting that protective effects emerge not because adolescents with more parental support are simply exposed to fewer stressors, but that parental support may help reduce physiological responses to stressors. Finally, a study observed that by improving family relationships, an intervention weakened the association between parental depression and biological aging among adolescents (Brody et al., 2015).

Importantly, conceptual models emphasize that social support must be delivered responsively for it to be perceived as supportive and thus exert buffering effects (Cohen and Wills, 1985; Feeney and Collins, 2015). Responsive support is generally defined as support that matches and respects the needs of the recipient (Collins and Ford, 2010; Feeney, 2004), and has largely been operationalized in terms of content or quality of support, such as whether the type of support, the amount of support, or the manner in which the support is delivered matches the recipients' needs (Feeney and Collins, 2015; Helgeson, 1993, 2003; Maisel and Gable, 2009). Here, we propose that timing of support may be an overlooked but important element, such that timely support would be perceived as responsive. Because achieving autonomy is a central goal for many adolescents, we hypothesize that parental support will be perceived as responsive only when it is delivered in a timely manner with respect to the adolescents' needs (e.g., when encountering a problem with peers). Otherwise, it may interfere with autonomy goals. Consistent with this notion, some nascent findings suggest receiving hugs on days with interpersonal conflicts attenuated the negative affect that typically ensues, whereas receiving hugs on days without interpersonal conflicts had no relationship with subsequent affect (Murphy et al., 2018), and there is some indications that such patterns may extend to biological outcomes, such as in predicting risks of developing a clinical cold after exposure to a viral challenge (Cohen et al., 2015). However, to our knowledge, studies have yet to examine whether responsive support, as conceptualized as how timely support was provided based on the recipient's needs, would buffer the negative impacts of stress on biological health outcomes, such as cardiometabolic risk.

The current investigation sought to fill this gap in knowledge using a week-long daily diary design. We considered whether responsive parental support would buffer the association between chronic psychological stress and cardiometabolic risk during adolescence, a developmental period that has long-lasting implications for health across the lifecourse (Johnson et al., 2011; Viner et al., 2015). To measure cardiometabolic risk, we assessed low-grade inflammation and metabolic syndrome, which are both early disease markers that can be reliably assessed during adolescence (Efstathiou et al., 2012; Herder et al., 2007; Juhola et al., 2011) and are predictive of future cardiovascular disease, above and beyond demographic background (e.g., socioeconomic status) and health behaviors (e.g., smoking; DeBoer et al., 2017; Ridker et al., 2000; Stoner et al., 2013). Based on previous literature that responsive support would confer protection against stress (Cohen and Wills, 1985; Feeney and Collins, 2015), we hypothesized that parental support that was presented on days adolescents experienced a stressor would attenuate the link between chronic psychological stress and cardiometabolic risks, whereas parental support that was presented on non-stressor days would not change the strength of this link. Reflecting our hypothesis that timing is especially crucial, we expected these buffering patterns to be evident above and beyond the average level of support that parents provided their adolescents.

1. Method

1.1. Participants and procedure

Participants were 277 adolescents recruited from the greater Chicago area through advertisements posted in local media and transit stations, announcements at various schools and community centers, and through a direct mail campaign. Youth were eligible if they were in eighth grade and in good health, defined as having no history of chronic medical or psychiatric illness, being free of infectious diseases during the past two weeks, not taking any prescription medication during the prior month, and not being currently pregnant.

Youth and one caregiver were invited to two laboratory visits that occurred two years apart, during their eighth and tenth grade years of school. As measures relevant for the current investigation were only administered at the second visit, analyses were performed using data from tenth-grade assessments only. During this visit, trained personnel collected anthropometric measures and fasting blood samples between 8 and 10am via antecubital venipuncture from youth. Youth then completed psychosocial questionnaires, interviews, and behavioral tasks while caregivers completed interviews assessing family socioeconomic background. For eight nights following the laboratory visit, youth were asked to complete daily diaries that assessed stress exposure and parental support. The diaries were completed using their phones at the end of each day right before bedtime.

Participants were included in the current analyses if they had completed measures of all predictors and covariates described below as well as at least one of the outcome assessments (low-grade inflammation or metabolic syndrome), resulting in an analytical N of 242 (24 participants did not complete the second visit, 5 did not have measures for all covariates, 2 did not complete the daily diaries, and 4 did not have blood data). Youth and caregivers provided written assent and consent to participate in all study procedures, which were approved by the Northwestern University Institutional Review Board.

1.2. Measures

1.2.1. Chronic psychological stress

The UCLA Life Stress Interview was used to assess chronic psychological stress experienced over the past 6 months (Hammen and Rudolph, 1999). This semi-structured interview focuses on stress in multiple life domains, including family relationships, friendships, home life, and school life. For each domain, a trained interviewer asked a series of open-ended questions and used the information gathered to rate the level of chronic and ongoing stress on a 1 to 5 scale with behaviorally specific anchors, with higher numbers reflecting more severe and persistent difficulties. The study's Principal Investigator and interviewers met regularly throughout data collection to ensure inter-rater reliability. To capture each participant's overall chronic stress burden, we averaged ratings across life domains to create a composite, such that higher scores indicate more severe chronic stress (alpha = 0.72).

1.2.2. Responsive parental support

Using the daily diary data, responsiveness of parental support was operationalized as the difference in parental support levels on stressor days compared to non-stressor days, such that increased parental support on stressor days would be indicative of more responsive parental

support. To assess daily stress exposure, each night, adolescents reported whether they experienced six different stressors with peers or at school (e.g., *threatened, insulted, or made fun of you, left you out of a group or group event*) and seven different stressors at home (e.g., *parents argued with each other, had a lot of demands made by family*). A binary variable was computed to reflect whether a stressor had occurred ($M_{\text{number of stressor days}} = 3.21$, $SD = 2.33$). Across the 8 days of diaries, 14 % of youth had 0 stressor days, 16 % had 1 stressor day, 14 % had 2 stressor days, 11 % had 3 stressor days, 12 % had 4 stressor days, 13 % had 5 stressor days, 12 % had 6 stressor days, 5 % had 7 stressor days, and 4 % had 8 stressor days.

To assess daily parental support, adolescents also responded to two items indicating whether they received parental support (i.e., *a parent advised, comforted, or listened to you about a problem*, *felt loved and cared for by parents*). Items were summed to compute a summary parental support score for each day that ranged from 0 to 2 (intraclass correlation = 0.46). For descriptive purposes, we dichotomized the sum variable into whether parental support occurred each day: across the 8 days of diaries, 8 % of the youth did not receive parental support on any days, 5 % had 1 support day, 5 % had 2 support days, 5 % had 3 support days, 6 % had 4 support days, 7 % had 5 support days, 14 % had 6 support days, 20 % had 7 support days, 30 % had 8 support days. However, note that primary analyses used the sum variable, which has greater day-to-day variance than the binary variable. Therefore, Fig. 1A further provides a visual depiction of the day-to-day variance per youth and Supplementary Table S1 additional day-level frequency for the sum variable.

To capture responsive parental support, we conducted a multi-level model where days were nested within individuals, which accounted for dependencies amongst the daily observations. As explained in Supplementary Box 1, for each participant, the model estimated a coefficient reflecting the difference in parental support on stressor days vs. non-stressor days. Of note, as depicted in Fig. 1B, substantial variability was observed in this coefficient (random effects b = 0.30, CI_{95} [0.22, 0.41]),[1] suggesting that how much parental support was provided on stressor days varied from youth to youth. Accordingly, we extracted this coefficient for each youth, which ranged from -0.37 to 0.75, such that more positive coefficients indicated that parental support tended to increase on stressor days (relative to non-stressor days), and thus representing more responsive parental support.

1.2.3. Low-grade inflammation

Low-grade inflammation was assessed using six biomarkers, including interleukin (IL)-6, IL-8, IL-10, tumor necrosis factor (TNF)-α, C-reactive protein (CRP), and soluble urokinase-type plasminogen activator receptor (suPAR). Fasting morning blood was collected in serum separator tubes and centrifuged at 1200 RCF for 10 min within 1–2 h of blood draw. Serum was then harvested, aliquoted, and stored at −20 Celsius until assays. CRP was measured in duplicate by high sensitivity immunoturbidimetric assay on a Roche/Hitachi cobas c502 instrument. Cytokines IL-6, IL-8, IL-10, and TNF-α were measured in triplicate by four-plex immunoassay on a microfluidic platform (Simple Plex; Protein Simple). Finally, suPAR was measured in duplicate by immunoassay (Human Quantikine ELISA; R&D Systems). Intra-assay coefficients of variation (CV) ranged from 1.6 % − 5.0 % and inter-assay CV ranged from 13 % to 19 %. To adjust for right skew in distribution, biomarkers were natural log transformed, except suPAR, which exhibited a normal distribution. A composite was then created by averaging the standardized values such that higher scores indicated higher level of low-grade inflammation (Cronbach's *alpha* = 0.68). This composite was used in primary analyses, but Supplementary Tables S3 and S5 report findings by each biomarker as well as findings using empirically informed composites.

1.2.4. Metabolic syndrome

Six components of metabolic syndrome were assessed, including waist circumference, blood pressure, cholesterol, triglycerides, fasting glucose, and insulin resistance. Waist circumference was measured at the midpoint between the upper iliac crest and lower costal margin. Resting blood pressure was assessed continuously for 10 min using a CNAP Monitor 500 (CNSystems). Systolic and diastolic blood pressure readings were averaged across the recording period. Fasting high-density lipoprotein cholesterol and triglycerides were assayed in serum, which was harvested, aliquoted, and stored as above until shipped to the NorthShore University HealthSystem laboratory for analysis. Fasting glucose was assayed photometrically from serum using a Roche/Hitachi Cobas c502 instrument. This assay has a dynamic range of 2 – 750 mg/dL and an intra-assay coefficient of variation (CV) of 0.7 %. Insulin was assessed in duplicate with an an electrochemiluminescent immunoassay (K15164C; MesoScale Discovery) on a SECTOR Imager 2400A (MesoScale Discovery). This assay has a lower limit of detection of 25 pg/ml and the intra-assay CV of 3.8 %. Insulin resistance was computed using the homeostatic model assessment equation: fasting glucose mmol/L × fasting insulin mIU/L divided by 22.5, with higher values reflecting higher insulin resistance.

According to the International Diabetes Federation (IDF), youth 11–16 years old meet criteria for MetS diagnosis if they have WC >/= 90th percentile for their age, sex at birth, and race/ethnicity and have at least two of the following: HDL cholesterol < 40 mg/dL, triglycerides >/= 150 mg/dL, fasting glucose >/= 100 mg/dL, and SBP >/= 130 mm/Hg and/or DBP >/= 85 mm (Zimmet et al., 2007). Because evidence suggests that homeostatic model assessment (HOMA) insulin resistance is more sensitive than fasting glucose in predicting metabolic syndrome among youth (Turchiano et al., 2012), one modification was made to the IDF criteria such that HOMA insulin resistance >/= 3.99 was used instead of fasting glucose. Only 29 adolescents (12 %) in our sample met criteria for MetS diagnosis as physical health problems were an exclusion criterion. Given the low prevalence of MetS, and concerns about the validity of this diagnosis in adolescence (Goodman et al., 2004; Goodman Elizabeth et al., 2007), our primary outcomes were (1) a count of the total number of metabolic syndrome components for which participant met clinical cutoffs (e.g., Miller, Lachman, et al., 2011) and (2) a continuous composite that averaged the standardized values of each of the MetS components (e.g., Levine et al., 2019; Miller et al., 2017, 2020). This composite resulted in four outliers (defined as +/-3 *SD* from the mean), which were excluded from primary analyses (Supplementary Table S2 and Fig. S1 present findings when outliers were winsorized to the next lowest/highest values, rather than excluded).

1.2.5. Covariates

We included sex at birth, race/ethnicity (hereafter, race), socioeconomic status, average daily stress, and average daily parental support as covariates. Participants self-reported sex at birth. Race was coded as Black, non-Black participants of color, and White. Multi-racial participants who identified as Black and of another category were coded as Black. Participants who identified as any other non-White race were coded as non-Black participants of color. White participants were coded as such only if they did not identify as any other race. We controlled for race because there are known racial disparities in stress and inflammation that resulted from experiences, policies, and practices that have systemically disadvantaged subgroups of individuals based on skin color (Bryant et al., 2022; Gravlee, 2009; Kuzawa and Gravlee, 2016). Race is used as a covariate to proxy for differences in these lived experiences. Similarly, both SES and inflammation are patterned by socioeconomic status (Stepanikova et al., 2017), thus was included as a covariate. Socioeconomic status was assessed using parent/caregiver reports of

[1] In multilevel models, the random effect of the slope parameter reflects person-to-person variability in the strength of the association predictor and outcome, which in this case are stress exposure and parental support (Raudenbush, 2009). Here, we report the random effect of the slopes in standard deviation units for ease of interpretation (nlme R package).

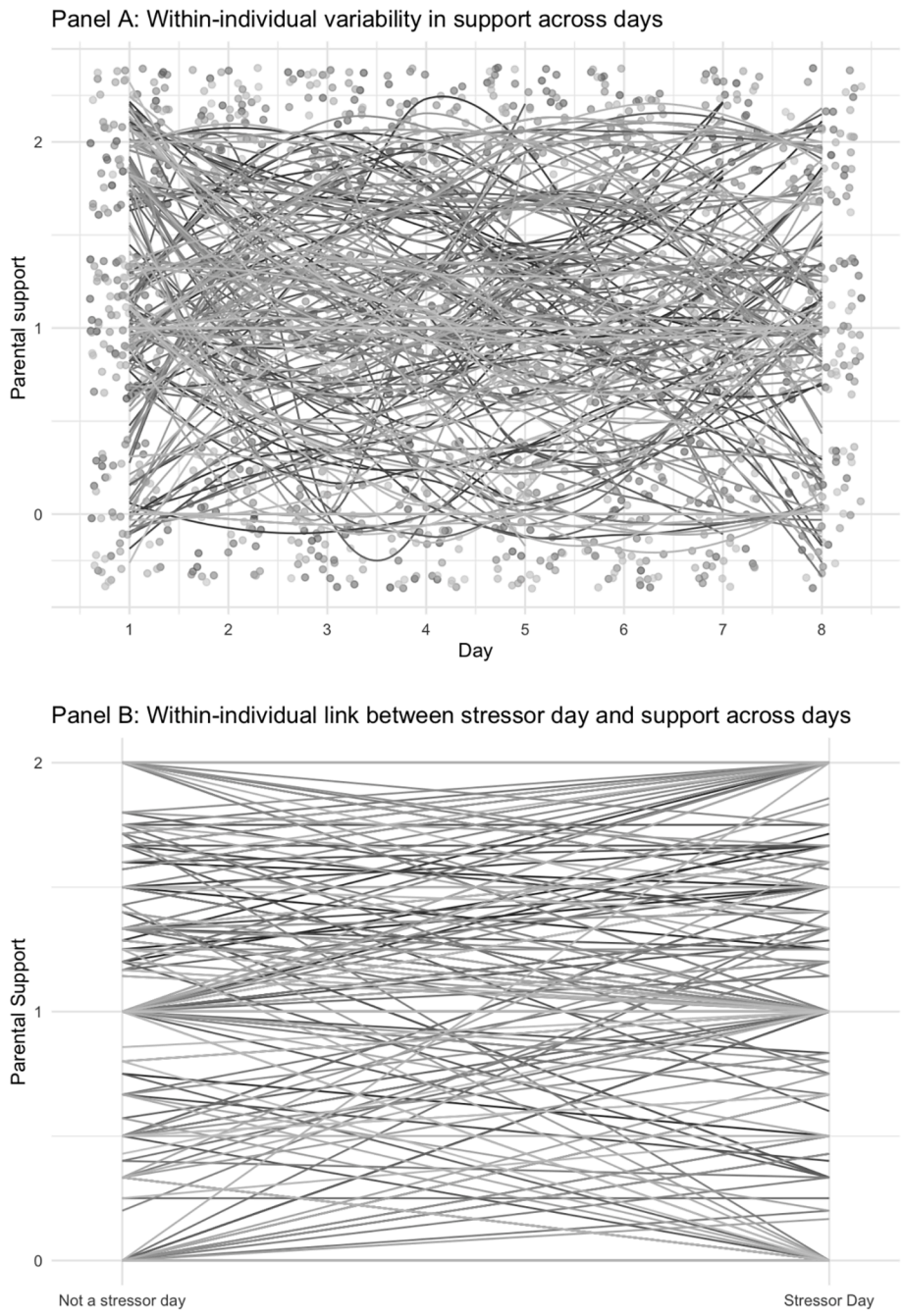

Fig. 1. Panel A displays the within-individual variability in support across days. Points were jittered and reflects each diary entry (N=1859). Each line reflects each youth's support level across days. Substantial within-individual day-to-day variability was observed as reflected by the fluctuations of lines; substantial between-individual variability was observed as reflected by variability from line to line. The intraclass correlation of parental support was 0.46 (i.e., 46% of the total variance of parental support level varied between youth and about 54% of the total variance varied day-to-day). Panel B displays within-individual links between stressor day (vs. not) and parental support across 8 days of diaries. More positive slopes indicate that parental support was more likely to co-occur on stressor days, thus more responsive parental support. As depicted, substantial variability was observed, suggesting that how much parental support was provided on stressor days varied from youth to youth.

family income reported on a 9-point scale: 1 = less than $5,000, 2 = $5,000 to $19,999, 3 = $20,000 to $34,999, 4 = $35,000 to $49,999, 5 = $50,000 to $74,999, 6 = $75,000 to $99,999, 7 = $100,000 to $149,999, 8 = $150,000 to $199,999, and 9 = more than $200,000. Average daily stress was assessed by averaging the binary variable representing stressor day vs. not across the 8-day period, which represents the proportion of stressor (vs. not stressor) days. Average daily parental support was assessed by averaging the daily parental support composite across the 8-day period. Proportion of stressor days and average parental support are considered covariates because (1) previous research have linked daily stress and parental support to cardiometabolic outcomes (e.g., Gouin et al., 2012); (2) responsive support was operationalized as the within-individual slope between stress and support, such that a slope score of zero can result from constant support (or the lack thereof) regardless of stress exposure as well as constant stress exposure (or the lack thereof) regardless of parental support (Supplementary Box 1). Although both scenarios are conceptually considered as less responsive parental support, controlling for average

stress and average support help ensure that results were not driven by differences these scenarios may have on inflammation and metabolic syndrome.

1.3. Analytical approach

First, to examine whether responsive parental support would confer protection against chronic stress, regressions were conducted predicting circulating inflammation and metabolic syndrome from (1) covariates – sex at birth, race/ethnicity, socioeconomic status, averaged daily stress, and averaged daily support, (2) chronic stress and responsive parental support, and (3) in a separate block, the interaction effect between chronic stress and responsive parental support. When examining metabolic syndrome as the outcome, Poisson regression was used for the count variable and linear regression was used for the continuous variable. Significant interactions were decomposed by computing the simple slopes between chronic stress and outcome at low (-1 *SD* from mean) and high (+1 *SD* from mean) responsive parental support. Next, we conducted follow-up analyses to probe the temporal window during which support needs to be offered after stress exposure for buffering effects to occur. Specifically, we estimated the lagged within-individual links between daily stress exposure and parental support provided 1, 2, or 3 days after the exposure and then repeated the above regression analyses to examine whether buffering effects would emerge.

We then conducted four sets of sensitivity analyses. First, we examined whether the buffering effects of responsive support remained significant when additionally controlling for the interaction between chronic stress and averaged daily support across the 8 days of diary reporting. Second, as smoking is associated with both inflammation and MetS, we repeated analyses excluding four youth who reported having smoked cigarettes in the past year. Furthermore, we did not include body mass index (BMI) as a covariate in inflammation models because adiposity is one of MetS components. As such, controlling for BMI would be controlling for an outcome of interest. However, to be transparent, we conducted sensitivity analyses for the inflammation model that additionally controlled for BMI. Third, although including proportion of stressor days and average parental support as covariates should account for the impacts of having constantly high or low support or stress on the current findings, we further examined whether the buffering effects of responsive support would remain when youth who reported 0 stressor days (n = 37), 8 stressor days (n = 9), 0 support across days (n = 22), and max support across days (n = 13) were removed from analyses. Fourth, to ensure that results were not driven by the averaging of standardized inflammation marker values, we reconducted analyses (1) predicting each biomarker in separate regressions; (2) using a mixed model that predicted each participant's standardized inflammation marker value from the same predictors as above, while also controlling for marker type and accounting for within-individual clustering. This approach allowed us to examine all six biomarkers wholistically without aggregation; (3) using unit-weighted and loadings-weighted composites informed by factor analyses.

Finally, we conducted exploratory analyses to examine whether buffering effects of responsive parental support would vary by race or sex at birth. Specifically, we tested the three-way interactions: chronic stress × responsive parental support × Black (vs. White), chronic stress × responsive parental support × non-Black youth of color (vs. White), and chronic stress × responsive parental support × female (vs. male).

2. Results

As summarized in Table 1, simple correlations suggest that adolescents with more chronic stress had higher levels of low-grade inflammation. In addition, non-Black adolescents of color, adolescents with lower socioeconomic status, and adolescents with more chronic stress had more metabolic syndrome components meeting clinical cutoffs. No other associations were observed between predictors and inflammation/metabolic syndrome.

2.1. Low-grade inflammation

First, we examined whether responsive support would buffer the link between chronic stress and low-grade inflammation. As summarized in Table 2, adjusting for age, sex at birth, race/ethnicity, socioeconomic status, averaged daily stress, and averaged daily support, more chronic stress was associated with higher levels of inflammation ($b = 0.24$, $SE = 0.07$, $p = .001$) and responsive support was not associated with inflammation. Furthermore, as depicted in Fig. 2A, a chronic stress by responsive support interaction emerged ($b = -0.82$, $SE = 0.36$, $p = .023$), such that every 1 *SD* increase in chronic stress was associated 0.37 *SD* increase in inflammation only among adolescents who received less responsive support ($\beta = 0.37$, $SE = 0.09$, $p < .01$), but not among those who received more responsive support ($\beta = 0.12$, $SE = 0.09$, $p = .22$).

In follow-up analyses, we examined whether similar buffering patterns would be observed if parental support was presented 1, 2, or 3 days after stressor exposure, rather than the day of stress exposure. For all three scenarios, we did not observe significant buffering patterns (p's > 0.33).

2.2. Metabolic syndrome

Next, we examined whether responsive support would buffer the link between chronic stress and metabolic syndrome. As summarized in Table 2, adjusting for covariates, more chronic stress was associated with metabolic syndrome as assessed with the continuous standardized composite ($b = 1.14$, $SE = 0.32$, $p = .001$) and the count of components above clinical cutoffs ($b = 0.36$, $SE = 0.12$, $p = .003$). Responsive support was not associated with metabolic syndrome. In addition, as depicted in Fig. 2B, a chronic stress by responsive support interaction emerged ($b = -1.66$, $SE = 0.68$, $p = .015$), such that among adolescents who received less responsive support, every 1 *SD* increase in chronic stress was

Table 1
Descriptive statistics and simple correlations among study variables (N = 242).

	Descriptive statistics	1	2	3	4	5	6	7	8	9
1. Female (vs. male)	63 %	1								
2. Black (vs. White)	38 %	0.00	1							
3. Non-Black participants of color (vs. White)	34 %	0.00	-0.56*	1						
4. Socioeconomic status	5.38 (2.24)	-0.08	-0.36*	-0.19*	1					
5. Proportion of stressor days	0.40 (0.29)	-0.03	-0.11	0.02	0.20*	1				
6. Average parental support	1.05 (0.55)	0.06	-0.12*	-0.09	0.22*	-0.03	1			
7. Chronic stress	2.25 (0.65)	-0.01	0.25*	0.13*	-0.54*	-0.02	-0.30*	1		
8. Responsive support	0.00 (0.15)	-0.06	0.01	0.02	0.03	0.09	-0.07	0.07	1	
9. Circulating inflammation	0.00 (0.62)	-0.05	-0.09	0.05	-0.03	-0.05	0.00	0.17*	0.07	1
10. Metabolic syndrome (count of symptoms)	0.94 (1.2)	-0.05	-0.02	0.17*	-0.18*	-0.04	-0.09	0.24*	0.04	0.33*

Note. *$p < .05$. Descriptive statistics are presented as means and standard deviations in parentheses for continuous variables and percentage of endorsed category for binary variables.

Table 2
Chronic stress by responsive support interaction on circulating inflammation and metabolic syndrome (N = 242).

	Unstandardized regression coefficient, CI_{95} [lower bound, upper bound], p-value		
	Circulating inflammation	Metabolic Syndrome (continuous variable)	Metabolic Syndrome (count variable)
Female (vs. Male)	-0.09 [-0.24, 0.07], p=.278	−1.48 [-2.18, -0.79], p=<0.001*	-0.15 [-0.42, 0.12], p=.263
Black (vs. White)	-0.17 [-0.39, 0.05], p=.135	-0.49 [-1.48, 0.5], p=.327	0.00 [-0.42, 0.43], p=.997
Non-Black participants of color (vs. White)	-0.09 [-0.31, 0.12], p=.394	0.08 [-0.9, 1.06], p=.872	0.32 [-0.08, 0.73], p=.120
Socioeconomic status	0.02 [-0.03, 0.06], p=.418	0.05 [-0.15, 0.25], p=.617	-0.02 [-0.1, 0.06], p=.604
Proportion of stressor days	-0.21 [-0.47, 0.05], p=.118	-0.4 [-1.57, 0.77], p=.503	-0.13 [-0.6, 0.34], p=.598
Average support	0.04 [-0.11, 0.18], p=.615	-0.35 [-1, 0.29], p=.277	-0.07 [-0.32, 0.19], p=.601
Chronic stress	0.24 [0.09, 0.38], p=.001*	1.14 [0.52, 1.77], p=<0.001*	0.36 [0.12, 0.59], p=.003*
Responsive support	0.26 [-0.23, 0.75], p=.300	0.16 [-2.02, 2.33], p=.887	0.15 [-0.74, 1.01], p=.744
Chronic stress × responsive support	-0.82 [-1.53, -0.11], p=.023*	−3.00 [-6.08, 0.07], p=.055†	−1.66 [-3.01, -0.33], p=.015*

Note. *p <.05. Interaction effects were entered as a separate block (i.e., the presented main effects are not lower ordered). Linear regressions were conducted for models involving circulating inflammation and the continuous standardized composite of metabolic syndrome. Poisson regression was conducted for the model involving count of metabolic syndrome components that met clinical cutoffs.

associated with an increase of 1.8 times the rate of having greater number of metabolic syndrome components over clinical cutoffs (*Incidence Rate Ratio [IRR]* = 1.86, b = 0.62, SE = 0.16, p <.01). However, this association was not apparent among those who received more responsive support (*IRR* = 1.12, b = 0.12, SE = 0.16, p =.46).

The interaction between chronic stress and responsive support on metabolic syndrome using the continuous standardized composite approached the conventional significance threshold (b = -3.00, SE = 1.56, p =.055). As depicted in Fig. 2C, the pattern of results was similar, such that every 1 *SD* increase in chronic stress was associated with about 0.37 *SD* increase in metabolic syndrome only among adolescents who received less responsive support (β = 0.37, SE = 0.09, p <.01), but not among adolescents who received more responsive support (β = 0.16, SE = 0.09, p =.10).

We then examined whether similar buffering patterns would occur if parental support was presented 1, 2, or 3 days after daily stressor exposure. Again, we did not observe significant buffering for one-day and three-days lagged parental support for both metabolic syndrome assessments (p's > 0.47). There were significant buffering patterns when parental support was presented two days after stress exposure (metabolic syndrome count variable: b = -3.60, SE = 1.0, p <.001; metabolic syndrome continuous variable: b = -6.24, SE = 2.5, p =.012); however, these findings should be considered as preliminary as the number of tests conducted may have inflated type I error.

2.3. Sensitivity analyses

We conducted four sets of sensitivity analyses. First, we examined whether responsive parental support remained a significant moderator when additionally controlling for the interaction between chronic stress and *average* level of support. Results suggest that even when adjusting for the average level of support, responsive parental support remained as a significant moderator, attenuating the link between chronic stress and low-grade inflammation (b = -0.79, SE = 0.36, p =.030). Similarly, responsive parental support remained a significant moderator for the link between chronic stress and count of metabolic syndrome components (b = -1.39, SE = 0.69, p =.044). These findings suggest that buffering by responsive parental support occurred above and beyond any protective benefits of parental support level.

Second, the buffering patterns remained significant when youth who reported smoking were excluded (chronic stress × responsive support in inflammation model: b = -0.87, SE = 0.36, p =.015; in metabolic syndrome model: b = -1.64, SE = 0.69, p =.017). Furthermore, when BMI was included as a covariate in the inflammation model, the pattern of results was similar, such that chronic stress was associated with inflammation only among youth with lower responsive support (b = 0.27, SE = 0.09, p <.001), but not among youth with higher support (b = 0.07, SE = 0.08, p =.43); however, the interaction was not significant, b = -0.67, SE = 0.35, p =.055. We did not control for BMI in metabolic syndrome model as adiposity is a key component of this outcome.

Third, we reconducted analyses excluding youth who receive constant support, or the lack thereof, on all 8 days as well as youth who experience stress, or the lack thereof, on all 8 days. Responsive support continued to confer protection (interaction b = -0.90, SE = 0.36, p =.015, N = 167), such that chronic stress was associated with inflammation only among youth with lower responsive support (b = 0.41, SE = 0.10, p <.001), but not among youth with higher support (b = 0.10, SE = 0.10, p =.330). The chronic stress by responsive support interaction on metabolic syndrome, however, dropped out of significance partly because of the smaller sample (interaction b = -1.31, SE = 0.70, p =.062, N = 177). However the magnitude of the interaction and its pattern were similar, such that chronic stress was linked with metabolic syndrome among youth with lower responsive support (IRR = 1.86, b = 0.62, SE = 0.18, p <.001), but not among youth with higher responsive support (IRR = 1.18, b = 0.17, SE = 0.18, p =.35). These findings suggest that constantly high/low stress or constantly high/low support did not drive the buffering patterns associated with timely support.

We also reconducted analyses excluding only youth who received no support across all 8 days or had constantly high or no stress across all 8 days, such that a score of 0 would include youth who received high support for all 8 days. Again, the pattern of results was the same. The link between chronic stress and inflammation was most apparent at low responsive support (simple slope at −1 SD: ß = 0.44), followed by some attenuation at 0 score (simple slope at 0: ß = 0.26), and then the most at high responsive support (simple slope at + 1 SD: ß = 0.08). This was similarly observed for metabolic syndrome (simple slope at −1 SD: IRR = 1.84; at 0: IRR = 1.46; at + 1 SD: IRR = 1.15). These findings suggest that relative to low responsive support, constantly high support (score of 0) may still attenuate the negative impacts of chronic stress, but that this attenuation was not as strong as high responsive support.

Fourth, we examined chronic stress by responsive support interaction with each inflammation marker, rather than a single composite. As summarized in Supplementary Table S3 and depicted in Supplementary Fig. S2, buffering patterns emerged for CRP (b = -1.24, SE = 0.61, p =.044), IL-6 (b = -1.38, SE = 0.59, p =.020), and IL-10 (b = -1.45, SE = 0.60, p =.016). The direction of the interaction was similar for TNF-a, but not significant (b = -0.98, SE = 0.59, p =.096). Interactions were not observed for IL-8 (b = 0.21, SE = 0.61, p =.735) and suPAR (b = -0.08, SE = 0.60, p =.894). Next, we repeated analyses using a mixed model to predict overall inflammation instead of a composite averaging the standardized value of each biomarker. Controlling for marker type, the chronic stress by responsive support interaction remained significant (b = -0.82, SE = 0.35, p =.021). Finally, we conducted a factor analysis with the six biomarkers as indicators. Results are summarized in Supplementary Table S4. Based on the final factor analysis model, a unit-weighted composite and a loading-weighted composite were computed using CRP, IL-6, IL-10, TNF- α, and suPAR. As summarized in Supplementary Table S5, responsive support remained a significant moderator for both the unit-weighted composite (b = -1.03, SE = 0.41, p =.012) and the loading-weighted composite (b = -1.34, SE = 0.50, p

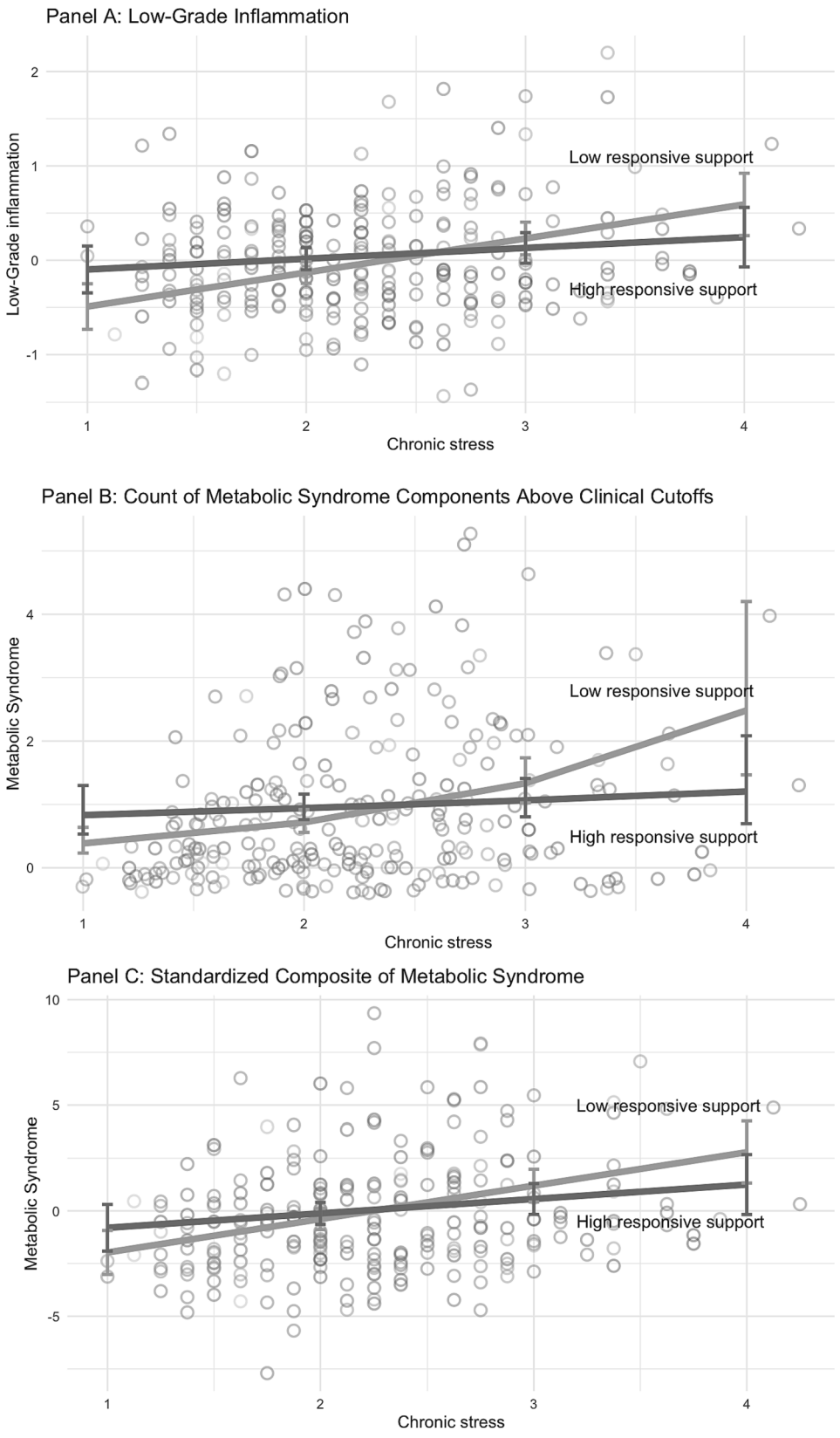

Fig. 2. The interaction between chronic stress and responsive support on low-grade inflammation (Panel A), count of metabolic syndrome components above clinical cutoffs (Panel B), standardized composite of metabolic syndrome (Panel C). Raw points are displayed and predicted slopes at low (-1 *SD* from mean) and high (+1 *SD* from mean) responsive parental support. Bars are 95% confidence intervals. Estimated simple slopes for count of metabolic syndrome components above clinical cutoffs depicted in Panel B are not linear because this outcome was modeled using Poisson regression.

=.007).

2.4. Exploratory analyses

We explored whether the magnitude of buffering associated with responsive support would vary by race and by sex at birth. As summarized in Supplementary Tables S6 and S7, buffering patterns did not vary by race (chronic stress × support × Black [vs. White] interaction b = -1.00 [-3.18, 1.17], p =.364 in inflammation model and −1.23 [-5.65, 3.29], p =.589 in metabolic syndrome model; chronic stress × support × non-Black people of color [vs. White] interaction b = -0.53 [-1.94, 3.01], p =.671 in inflammation model and b = 0.58 [-3.79, 5.18], p =.802 in metabolic syndrome model) nor by sex at birth (chronic stress × support × female [vs. male] interaction b = 0.15 [-1.34, 1.63], p =.846 in inflammation model and b = -1.51 [-4.31, 1.23], p =.285 in metabolic syndrome model).

3. Discussion

While emerging evidence suggests that parental support can attenuate the negative biological impacts of chronic stress among youth, studies have not examined whether the timing of *when* parental support is presented has implications for its protective effect. The current findings suggest that timely parental support buffered the link between chronic stress and cardiometabolic risk, as assessed with low-grade inflammation and metabolic syndrome. Specifically, more chronic stress was linked with greater cardiometabolic risk only when parental support was not aligned with daily exposure (i.e., not received on days adolescents had stressors or received on days without stressors), but this association was not apparent among adolescents who received timely parental support. Notably, these patterns were observed above and beyond average level of parental support, highlighting the importance of considering *timing* of support in addition to *level* of support. In addition, buffering patterns were evident above and beyond the proportion of stressor days, suggesting that protection was not due to lower stress exposure among those with high responsive support.

How may timely parental support to *daily* stressors buffer the impacts of *chronic* stress on cardiometabolic risk? Chronic stress is generally comprised of multiple events that occur across shorter timescales. These events are postulated to contribute to longer-term health problems because they trigger acute behavioral and biological responses that accumulate in organs over time. By attenuating these responses as they are unfolding, timely parental support would be able to minimize or prevent this accumulation. For example, an adolescent who is experiencing chronic interpersonal stress with peers may be exposed to daily stressors, such as being made fun of or having arguments with friends. If on days when these stressors occur, the parent provides support, such as providing comfort, a safe space to express, or help in problem-solving, the adolescent may perceive these events as less threatening or may feel an increased sense of efficacy in coping. In turn, this may lower the typical biological responses (e.g., cardiovascular reactivity; Kamarck and Lovallo, 2003) and behavioral responses (e.g., shorter or poorer sleep Kalmbach et al., 2018). Over time, these attenuated stress responses may accumulate to confer protection against cardiometabolic risk. However, unlike this example, our measures of chronic stress and daily stress are not exact matches in terms of the domains in which they are occurring, so future research would benefit from examining whether results would replicate if same-domain chronic and daily stressors were assessed.

Other explanations for the current findings may be methodological in nature. For example, values at the negative end of our responsive support measure could reflect support provided on days without stressors, which may be perceived as not respecting the adolescent's personal space. However, as the support items were framed positively, buffering patterns likely did not emerge solely because of the negative pole of our scale. Future research will benefit from directly assessing youth's perceived valence of parental support. Furthermore, although a negative score and a positive score on the current responsive support measure are readily interpretable, a score of zero is more ambiguous. A series of sensitivity analyses suggest that constantly low or high stress or support did not drive the current results. However, future research is necessary to examine whether the protective value responsive support has against chronic stress would vary based on trait level of support. Finally, although we interpret our timing of parental support measure as how responsive parents were to adolescents' needs, it may in fact be capturing how likely adolescents were to *seek* support from parents on days with stressors. As such, it may be adolescents' timely support-seeking, rather than parents' responsive support, that buffered the link between chronic stress and cardiovascular risk. Future research would benefit from administering diaries to both parents and adolescents to tease apart adolescents' support-seeking behaviors vs. parents' support-offering behaviors.

3.1. Implications

The current findings are consistent with theories postulating that support may only be beneficial if it is delivered responsively (Feeney and Collins, 2015) and extend these theories by suggesting that timing of support may be an important but unrecognized element of responsiveness. As alluded to above, this may be because achieving autonomy from parents is a common goal during adolescence, and thus parental support may not be well-received if it is provided without stress exposure, whereas timely parental support may be perceived as respectful of adolescents' changing developmental needs and goals. This conceptualization may help explain why parental support does not always confer protective effects during adolescence. For example, studies have found that relative to a stranger's support, parental support promoted more adaptive cortisol responses to standardized laboratory stressors for children, but not for adolescents (Gee et al., 2014; Hostinar et al., 2015; Perry et al., 2021). Together with previous literature, our findings emphasize the need for more nuanced approaches, beyond level of support, to understand when parental support would and would not exert protective effects during adolescence.

While the current study begins to address this question, it also invites the question of what other adolescence-sensitive factors, in addition to timing of support, would change the protective value of parental support during this developmental period (Chen et al., 2017). For instance, autonomy goals during adolescence may render emotional support as the preferred type of support, relative to instrumental and informative support; the decreasing proportion of time spent with parents relative to peers during adolescence may render perceived availability of support more important than actual amount of support delivered; the increase in sensitivity to evaluations during adolescence may render the manner in which support was conveyed important to consider. Future research will benefit from examining how development may shape the type of support and support-delivery approach that would be beneficial for adolescents and how in turn that may change the buffering magnitude of parental support across development. Furthermore, the current findings also invite the questions of whether the observed effects would extend beyond adolescence and beyond parent–child relationships. Future research will benefit from examining whether timing of support would confer protection against stress, above and beyond level of support, in childhood or adulthood, and whether timing of support would be relevant for teacher-child, peer, and romantic relationships.

3.2. Limitations

There are limitations to the current study. First, as we only have relevant measures cross-sectionally, we are unable to establish directionality of these observations, and cannot make inferences about causality for responsive parental support. Second, although the buffering magnitude of responsive support did not vary across racial groups, we

may not be well-powered to detect three-way interactions. Specifically, assuming 12 total predictors in the model, our sample size of 242 can detect a regression coefficient of $\beta = 0.18$ or larger with at least 0.80 power; however, effect sizes for three-way interactions are often much smaller. As there are known cultural differences in family values and interactions (Dayton et al., 2022) as well as in developmental goals (Benito-Gomez et al., 2020), timely parental support may be less important in cultures where autonomy from parents is not as valued during adolescence. Future research will be necessary to examine whether the observed buffering effects of timely parental support would vary across different cultures in well-powered samples. Third, there are limitations to our measurements. Because we have too few numbers of days of diaries, our analyses examining the temporal window during which parental support need to occur after daily stress exposure should be considered preliminary. In addition, we did not examine daily stressors in non-social domains (e.g., academic stress), parse support by types (e.g., emotional vs. instrumental), and examine stressor-specific support. Future research will benefit from having improved daily stress and support assessments as well as examining whether type of support may moderate the observed effects. Fourth, although our current findings point to practical implications, given the alternative explanations mentioned above, conceptual replication and additional empirical work will be necessary before they can be implemented. Of note, that chronic stress was associated with poorer outcomes only among youth less timely support should not be interpreted as reflecting variation in *choices* of parenting behaviors. Rather, parents may not have the opportunity to provide timely support for other reasons, including decreased parent–child interaction time during adolescence, parent's own level of stress, or contextual barriers such as socioeconomic status. Thus, to inform interventions, we must first understand what factors may promote or decrease parents' ability to provide prompt support. For example, would parents' own stress exposures, level of perceived support, and psychological resources predict whether they provide responsive support to the child? Future research would benefit from additionally assessing parents' daily social exposures to gain traction in answering these questions.

3.3. Conclusions

To conclude, youth exposed to chronic stress have increased risk for cardiovascular health problems, and parental support has been found to buffer this association. The current study extends knowledge by demonstrating that timely parental support provided on stressors days buffered the link between chronic stress and cardiovascular risk, as assessed with low-grade inflammation and metabolic syndrome. These findings highlight the importance of taking a developmental approach to better understand the conditions under which parental support can provide health-protective benefits during adolescence.

Funding source

This research was supported by a grant from the National Institutes of Health HL122328 (GEM), HL136676 (EC), F31 HL147509 (PHL).

Financial disclosure

The authors have no financial relationships relevant to this article to disclose.

Conflicts of interest

The authors have no conflicts of interest to disclose.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

The authors do not have permission to share data.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.bbi.2023.11.027.

References

Adler, N.E., Stewart, J., 2010. Health disparities across the lifespan: Meaning, methods, and mechanisms. Ann. N. Y. Acad. Sci. 1186 (1), 5–23.
Bai, S., Reynolds, B.M., Robles, T.F., Repetti, R.L., 2017. Daily links between school problems and youth perceptions of interactions with parents: A diary study of school-to-home spillover. Soc. Dev. 26 (4), 813–830.
Benito-Gomez, M., Williams, K.N., McCurdy, A., Fletcher, A.C., 2020. Autonomy-supportive parenting in adolescence: Cultural variability in the contemporary United States. J. Fam. Theory Rev. 12 (1), 7–26.
Brady, S.S., Matthews, K.A., 2006. Chronic stress influences ambulatory blood pressure in adolescents. Ann. Behav. Med. 31 (1), 80–88.
Brody, G.H., Lei, M.K., Chae, D.H., Yu, T., Kogan, S.M., Beach, S.R.H., 2014. Perceived Discrimination Among African American Adolescents and Allostatic Load: A Longitudinal Analysis With Buffering Effects. Child Dev. 85 (3), 989–1002. https://doi.org/10.1111/cdev.12214.
Brody, G.H., Yu, T., Beach, S.R., Philibert, R.A., 2015. Prevention effects ameliorate the prospective association between nonsupportive parenting and diminished telomere length. Prev. Sci. 16, 171–180.
Brody, G.H., Miller, G.E., Yu, T., Beach, S.R., Chen, E., 2016. Supportive family environments ameliorate the link between racial discrimination and epigenetic aging: A replication across two longitudinal cohorts. Psychol. Sci. 27 (4), 530–541.
Brown, B. B., & Larson, J. (2009). *Peer relationships in adolescence.*.
Bryant, B.E., Jordan, A., Clark, U.S., 2022. Race as a social construct in psychiatry research and practice. JAMA Psychiat. 79 (2), 93–94.
Cameron, C.A., McKay, S., Susman, E.J., Wynne-Edwards, K., Wright, J.M., Weinberg, J., 2017. Cortisol stress response variability in early adolescence: Attachment, affect and sex. J. Youth Adolesc. 46 (1), 104–120.
Chen, E., Brody, G.H., Miller, G.E., 2017. Childhood close family relationships and health. Am. Psychol. 72 (6), 555–566. https://doi.org/10.1037/amp0000067.
Chiang, J.J., Lam, P.H., Chen, E., Miller, G.E., 2022. Psychological stress during childhood and adolescence and its association with inflammation across the lifespan: A critical review and meta-analysis. Psychol. Bull. 148 (1–2), 27.
Cohen, S., Janicki-Deverts, D., Turner, R.B., Doyle, W.J., 2015. Does hugging provide stress-buffering social support? A study of susceptibility to upper respiratory infection and illness. Psychol. Sci. 26 (2), 135–147.
Cohen, S., Wills, T.A., 1985. Stress, social support, and the buffering hypothesis. Psychol. Bull. 98 (2), 310–357.
Collins, N.L., Ford, M.B., 2010. Responding to the needs of others: The caregiving behavioral system in intimate relationships. J. Soc. Pers. Relat. 27 (2), 235–244.
Collins, W.A., Laursen, B., 2004. Parent-adolescent relationships and influences. Handbook Adolescent Psychol. 2, 331–362.
Dayton, A., Aceves-Azuara, I., Rogoff, B., 2022. Collaboration at a microscale: Cultural differences in family interactions. Br. J. Dev. Psychol. 40 (2), 189–213.
DeBoer, M.D., Gurka, M.J., Golden, S.H., Musani, S.K., Sims, M., Vishnu, A., Guo, Y., Pearson, T.A., 2017. Independent associations between metabolic syndrome severity and future coronary heart disease by sex and race. J. Am. Coll. Cardiol. 69 (9), 1204–1205.
Efstathiou, S.P., Skeva, I.I., Zorbala, E., Georgiou, E., Mountokalakis, T.D., 2012. Metabolic Syndrome in Adolescence: The Prediction of Metabolic Syndrome in Adolescence (PREMA) Study. Circulation 125 (7), 902–910.
Elizabeth, G., Daniels, S.R., Meigs, J.B., Dolan, L.M., 2007. Instability in the Diagnosis of Metabolic Syndrome in Adolescents. Circulation 115 (17), 2316–2322. https://doi.org/10.1161/CIRCULATIONAHA.106.669994.
Feeney, B.C., 2004. A secure base: Responsive support of goal strivings and exploration in adult intimate relationships. J. Pers. Soc. Psychol. 87 (5), 631.
Feeney, B.C., Collins, N.L., 2015. A new look at social support: A theoretical perspective on thriving through relationships. Pers. Soc. Psychol. Rev. 19 (2), 113–147.
Galobardes, B., Smith, G.D., Lynch, J.W., 2006. Systematic Review of the Influence of Childhood Socioeconomic Circumstances on Risk for Cardiovascular Disease in Adulthood. Ann. Epidemiol. 16 (2), 91–104. https://doi.org/10.1016/j.annepidem.2005.06.053.
Gee, D.G., Gabard-Durnam, L., Telzer, E.H., Humphreys, K.L., Goff, B., Shapiro, M., Flannery, J., Lumian, D.S., Fareri, D.S., Caldera, C., 2014. Maternal buffering of human amygdala-prefrontal circuitry during childhood but not during adolescence. Psychol. Sci. 25 (11), 2067–2078.
Godoy, L.C., Frankfurter, C., Cooper, M., Lay, C., Maunder, R., Farkouh, M.E., 2021. Association of adverse childhood experiences with cardiovascular disease later in life: A review. JAMA Cardiol. 6 (2), 228–235.

Goodman, E., Daniels, S.R., Morrison, J.A., Huang, B., Dolan, L.M., 2004. Contrasting prevalence of and demographic disparities in the World Health Organization and National Cholesterol Education Program Adult Treatment Panel III definitions of metabolic syndrome among adolescents. J. Pediatr. 145 (4), 445–451. https://doi.org/10.1016/j.jpeds.2004.04.059.

Goodman, E., Daniels, S.R., Dolan, L.M., 2007. Socioeconomic disparities in insulin resistance: Results from the Princeton School District Study. Psychosom. Med. 69 (1), 61–67.

Gouin, J.-P., Glaser, R., Malarkey, W.B., Beversdorf, D., Kiecolt-Glaser, J., 2012. Chronic stress, daily stressors, and circulating inflammatory markers. Health Psychol. 31 (2), 264–268.

Gravlee, C.C., 2009. How race becomes biology: Embodiment of social inequality. Am. J. Phys. Anthropol. 139 (1), 47–57.

Hackman, D.A., Betancourt, L.M., Brodsky, N.L., Kobrin, L., Hurt, H., Farah, M.J., 2013. Selective impact of early parental responsivity on adolescent stress reactivity. PLoS One 8 (3), e58250.

Halfon, N., Verhoef, P.A., Kuo, A.A., 2012. Childhood antecedents to adult cardiovascular disease. Pediatr. Rev. 33 (2), 51–61.

Hammen, C., & Rudolph, K. (1999). UCLA life stress interview for children: Chronic stress and episodic life events. *Manual. University of Illinois*.

Helgeson, V.S., 1993. Two important distinctions in social support: Kind of support and perceived versus received 1. J. Appl. Soc. Psychol. 23 (10), 825–845.

Helgeson, V.S., 2003. Social support and quality of life. Qual. Life Res. 12 (Suppl 1), 25–31.

Herder, C., Schneitler, S., Rathmann, W., Haastert, B., Schneitler, H., Winkler, H., Bredahl, R., Hahnloser, E., Martin, S., 2007. Low-grade inflammation, obesity, and insulin resistance in adolescents. J. Clin. Endocrinol. Metab. 92 (12), 4569–4574.

Hostinar, C.E., Johnson, A.E., Gunnar, M.R., 2015. Parent support is less effective in buffering cortisol stress reactivity for adolescents compared to children. Dev. Sci. 18 (2), 281–297.

Johnson, M.K., Crosnoe, R., Elder Jr, G.H., 2011. Insights on adolescence from a life course perspective. J. Res. Adolesc. 21 (1), 273–280.

Juhola, J., Magnussen, C.G., Viikari, J.S., Kähönen, M., Hutri-Kähönen, N., Jula, A., Lehtimäki, T., Åkerblom, H.K., Pietikäinen, M., Laitinen, T., 2011. Tracking of serum lipid levels, blood pressure, and body mass index from childhood to adulthood: The Cardiovascular Risk in Young Finns Study. J. Pediatr. 159 (4), 584–590.

Juonala, M., Magnussen, C.G., Venn, A., Dwyer, T., Burns, T.L., Davis, P.H., Chen, W., Srinivasan, S.R., Daniels, S.R., Kähönen, M., 2010. Influence of age on associations between childhood risk factors and carotid intima-media thickness in adulthood: The Cardiovascular Risk in Young Finns Study, the Childhood Determinants of Adult Health Study, the Bogalusa Heart Study, and the Muscatine Study for the International Childhood Cardiovascular Cohort (i3C) Consortium. Circulation 122 (24), 2514–2520.

Kalmbach, D.A., Anderson, J.R., Drake, C.L., 2018. The impact of stress on sleep: Pathogenic sleep reactivity as a vulnerability to insomnia and circadian disorders. J. Sleep Res. 27 (6), e12710.

Kamarck, T.W., Lovallo, W.R., 2003. Cardiovascular reactivity to psychological challenge: Conceptual and measurement considerations. Psychosom. Med. 65 (1), 9–21.

Klevens, J., Hall, J., 2014. The importance of parental warmth, support, and control in preventing adolescent misbehavior. J. Child Adolescent Behav. 2 (1), 121–129.

Kuhlman, K.R., Chiang, J.J., Horn, S., Bower, J.E., 2017. Developmental psychoneuroendocrine and psychoneuroimmune pathways from childhood adversity to disease. Neurosci. Biobehav. Rev. 80, 166–184.

Kuzawa, C.W., Gravlee, C.C., 2016. Beyond genetic race: Biocultural insights into the causes of racial health disparities. New Direct. Biocultural Anthropol. 89–105.

Lam, P.H., Chiang, J.J., Chen, E., Miller, G.E., 2021. Race, socioeconomic status, and low-grade inflammatory biomarkers across the lifecourse: A pooled analysis of seven studies. Psychoneuroendocrinology 123, 104917.

Levine, C.S., Markus, H.R., Austin, M.K., Chen, E., Miller, G.E., 2019. Students of color show health advantages when they attend schools that emphasize the value of diversity. Proc. Natl. Acad. Sci. 116 (13), 6013–6018.

Maisel, N.C., Gable, S.L., 2009. The paradox of received social support: The importance of responsiveness. Psychol. Sci. 20 (8), 928–932.

Manczak, E.M., Leigh, A.K., Chin, C.-P., Chen, E., 2018. Consistency matters: Consistency in the timing and quality of daily interactions between parents and adolescents predicts production of proinflammatory cytokines in youths. Dev. Psychopathol. 30 (2), 373–382.

Miller, G.E., Chen, E., Parker, K.J., 2011a. Psychological stress in childhood and susceptibility to the chronic diseases of aging: Moving toward a model of behavioral and biological mechanisms. Psychol. Bull. 137 (6), 959.

Miller, G.E., Lachman, M.E., Chen, E., Gruenewald, T.L., Karlamangla, A.S., Seeman, T. E., 2011b. Pathways to resilience: Maternal nurturance as a buffer against the effects of childhood poverty on metabolic syndrome at midlife. Psychol. Sci. 22 (12), 1591–1599.

Miller, G.E., Chen, E., Yu, T., Brody, G.H., 2017. Metabolic syndrome risks following the great recession in rural black young adults. J. Am. Heart Assoc. 6 (9), e006052.

Miller, G.E., Chen, E., Yu, T., Brody, G.H., 2020. Youth who achieve upward socioeconomic mobility display lower psychological distress but higher metabolic syndrome rates as adults: Prospective evidence from Add Health and MIDUS. J. Am. Heart Assoc. 9 (9), e015698.

Murphy, M.L., Janicki-Deverts, D., Cohen, S., 2018. Receiving a hug is associated with the attenuation of negative mood that occurs on days with interpersonal conflict. PLoS One 13 (10), e0203522.

Perry, N., Johnson, A., Hostinar, C., Gunnar, M., 2021. Parental emotional support and social buffering in previously institutionalized and typically developing children and adolescents. Dev. Psychobiol. 63 (5), 1167–1176.

Pervanidou, P., Chrousos, G.P., 2011. Stress and obesity/metabolic syndrome in childhood and adolescence. Int. J. Pediatr. Obes. 6 (sup1), 21–28.

Raudenbush, S.W., 2009. Analyzing effect sizes: Random-effects models. The handbook of research synthesis and meta-analysis 2, 295–316.

Repetti, R.L., Robles, T.F., Reynolds, B.M., Sears, M.S., 2012. A naturalistic approach to the study of parenting. Parenting 12 (2–3), 165–174.

Ridker, P.M., Hennekens, C.H., Buring, J.E., Rifai, N., 2000. C-reactive protein and other markers of inflammation in the prediction of cardiovascular disease in women. N. Engl. J. Med. 342 (12), 836–843.

Stepanikova, I., Bateman, L.B., Oates, G.R., 2017. Systemic Inflammation in Midlife: Race, Socioeconomic Status, and Perceived Discrimination. Am. J. Prev. Med. 52 (1 Suppl 1), S63–S76. https://doi.org/10.1016/j.amepre.2016.09.026.

Stoner, L., Lucero, A.A., Palmer, B.R., Jones, L.M., Young, J.M., Faulkner, J., 2013. Inflammatory biomarkers for predicting cardiovascular disease. Clin. Biochem. 46 (15), 1353–1371.

Suglia, S.F., Ryan, L., Laden, F., Dockery, D., Wright, R.J., 2008. Violence exposure, a chronic psychosocial stressor, and childhood lung function. Psychosom. Med. 70 (2), 160.

Suglia, S.F., Appleton, A.A., Bleil, M.E., Campo, R.A., Dube, S.R., Fagundes, C.P., Heard-Garris, N.J., Johnson, S.B., Slopen, N., Stoney, C.M., 2021. Timing, duration, and differential susceptibility to early life adversities and cardiovascular disease risk across the lifespan: Implications for future research. Prev. Med. 153, 106736.

Tamayo, T., Herder, C., Rathmann, W., 2010. Impact of early psychosocial factors (childhood socioeconomic factors and adversities) on future risk of type 2 diabetes, metabolic disturbances and obesity: A systematic review. BMC Public Health 10 (1), 525. https://doi.org/10.1186/1471-2458-10-525.

Turchiano, M., Sweat, V., Fierman, A., Convit, A., 2012. Obesity, metabolic syndrome, and insulin resistance in urban high school students of minority race/ethnicity. Arch. Pediatr. Adolesc. Med. 166 (11), 1030–1036.

Viner, R. M., Ross, D., Hardy, R., Kuh, D., Power, C., Johnson, A., Wellings, K., McCambridge, J., Cole, T. J., & Kelly, Y. (2015). *Life course epidemiology: Recognising the importance of adolescence*.

Zimmet, P., Alberti, K.G.M., Kaufman, F., Tajima, N., Silink, M., Arslanian, S., Wong, G., Bennett, P., Shaw, J., Caprio, S., 2007. The metabolic syndrome in children and adolescents–an IDF consensus report. Pediatr. Diabetes 8 (5), 299–306.