UC Irvine

UC Irvine Previously Published Works

Title

Perceptions of parental secure base support in African American adolescents and young adults

Permalink

https://escholarship.org/uc/item/7k55q3v1

Journal

Journal of Social and Personal Relationships, 34(8)

ISSN

0265-4075

Authors

Jones, Jason D

Ehrlich, Katherine B

Brett, Bonnie E

et al.

Publication Date

2017-12-01

DOI

10.1177/0265407516670532

Peer reviewed

eScholarship.org Powered by the California Digital Library
University of California

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

HHS Public Access

Author manuscript

*J Soc Pers Relat*. Author manuscript; available in PMC 2023 January 27.

Published in final edited form as:

*J Soc Pers Relat*. 2017 December ; 34(8): 1168–1185. doi:10.1177/0265407516670532.

# Perceptions of parental secure base support in African American adolescents and young adults: A preliminary study of predictive links to adult C-reactive protein

**Jason D. Jones**[1], **Katherine B. Ehrlich**[2], **Bonnie E. Brett**[1], **Jacquelyn T. Gross**[1], **Jonathan J. Mohr**[1], **Elizabeth A. Hopper**[3], **Julie V. Dinh**[4], **Oksana Malanchuk**[5], **Stephen C. Peck**[5], **Amanda B. Brodish**[5], **Emma K. Adam**[2], **Jacquelynne S. Eccles**[5], **Margaret E. Kemeny**[4], **Jude Cassidy**[1]

[1]University of Maryland, College Park, USA

[2]Northwestern University, USA

[3]University of California, Santa Barbara, USA

[4]University of California, San Francisco, USA

[5]University of Michigan, USA

## Abstract

Within the field of relationship science there is increasing interest in the connections between close relationships and physical health. In the present study, we examined whether adolescents' (~12 years old) and young adults' (~20 years old) perceptions of their parents as a secure base prospectively predict C-reactive protein (CRP), a commonly used marker of inflammatory activity, at age 32 in a well-characterized sample of African Americans. We utilized existing data collected as part of the Maryland Adolescent Development in Context Study (MADICS) to construct measures of perceptions of parental secure base support (SBS), general parental support, and peer support in early adolescence and early adulthood. In the present study, SBS was operationalized as the perceived ability to depend on parents in times of need. Fifty-nine African American MADICS participants who reported on perceived support in early adolescence and early adulthood participated in a follow-up home visit at age 32 during which serum CRP was measured via a blood draw. After controlling for inflammation-related confounds (e.g., tobacco use, body mass index), adolescents' perceptions of parental SBS, but not peer support or general parental support, predicted lower CRP values at age 32 ($b = -.92$, $SE = .34$, $p < .05$). None of the support variables in early adulthood predicted CRP at 32 years. This study adds to a growing literature on relationships and health-related outcomes and provides the first evidence for a link between parental SBS in adolescence and a marker of inflammatory activity in adulthood.

**Corresponding author:** Jason D. Jones, Department of Psychiatry, University of Pennsylvania, 10 Gates, HUP, Philadelphia, PA 19104, USA. jasjones@upenn.edu.

Authors' note

Jason D. Jones is now at University of Pennsylvania, Department of Psychiatry. Katherine B. Ehrlich is now at University of Georgia, Department of Psychology and Center for Family Research. Jacquelynne S. Eccles is now at the University of California, Irvine, School of Education. Julie V. Dinh is now at Rice University, Department of Psychology.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

**Keywords**

Adolescence; attachment; C-reactive protein; inflammation; relationships and health; secure base

Mounting evidence suggests that chronic inflammatory activity plays an important role in the development of many diseases of aging and is a risk factor for early mortality (Ridker, 2007). Although acute inflammatory responses are critical to the body's defense against infection and tissue damage, persistent inflammatory activity contributes to coronary heart disease, stroke, and diabetes (Chung et al., 2009)—conditions with morbidity and mortality rates that are higher in African Americans relative to Whites (Centers for Disease Control and Prevention [CDC], 2013). Researchers commonly use serum levels of C-reactive protein (CRP) as an indicator of ongoing inflammatory activity. CRP is a protein produced by the liver that is more stable in blood than other common measures of inflammation, such as cytokines. CRP provides a reliable index of low-grade inflammatory activity and is frequently used as a clinical indicator of cardiovascular disease risk (Ridker, 2003). In fact, both the CDC and the American Heart Association have endorsed measuring CRP as part of risk assessment for heart disease (Pearson et al., 2003).

Given the well-documented links between inflammatory activity and adult health outcomes, research aimed at identifying predictors of inflammatory markers has substantial public health relevance. A large body of evidence has demonstrated that *adult* exposure to acute or chronic stressful life experiences can be associated with concurrent elevation in measures of inflammatory activity (e.g., Dickerson, Gable, Irwin, Aziz, & Kemeny, 2009). For example, evidence suggests that low marital quality is associated with elevated levels of inflammatory markers, particularly among women (Donoho, Crimmins, & Seeman, 2013; Whisman & Sbarra, 2012). Further, mounting evidence suggests that *early* life experiences also have an influence on inflammatory processes in adulthood (see Ehrlich, Miller, & Chen, 2016, and Miller, Chen, & Parker, 2011, for reviews). For example, growing up in a low socioeconomic status (SES) environment predicted higher CRP in adulthood, independent of adult SES (Pollitt et al., 2007). Further, evidence suggests that early experiences with caregivers predict inflammatory markers in adulthood. For instance, negative experiences with parents (e.g., maternal rejection, physical abuse) during childhood were associated with elevated CRP in adulthood (Danese, Pariante, Caspi, Taylor, & Poulton, 2007). Moreover, Chen, Miller, Kobor, and Cole (2011) found, in a sample of adults who grew up in low SES environment, that individuals who experienced high maternal warmth during childhood (retrospectively reported) exhibited leukocyte gene expression patterns suggestive of reduced pro-inflammatory signaling relative to adults who reported low maternal warmth. Despite these intriguing initial findings, further developmental research is needed to better understand the types of early experiences that could influence adult inflammatory activity. In this study, we examine how adolescents' and young adults' perceptions of their parents as a secure base relate to adult inflammatory activity.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

## Access to a secure base

Theory and research about the role of close relationships in development indicate that the availability of a responsive and dependable caregiver is critical to healthy child functioning (e.g., Shonkoff & Phillips, 2000). Within the context of attachment theory, a caregiver on whom a child can depend in times of need or distress is referred to as *a secure base*, and the ability to rely on one's parent(s) as a secure base is the defining feature of attachment security (Ainsworth, 1967; Bowlby, 1988). Bowlby (1988) emphasized the importance of a secure base for child development: "No concept within the attachment framework is more central to developmental psychiatry than that of the secure base" (p. 163–164). Several decades of research demonstrating that a child's access to a secure base early in life is related to functioning at the behavioral, socio-emotional, and physiological levels support Bowlby's proposition (see Cassidy & Shaver, 2016, for reviews).

Although the original focus of research on the secure base phenomenon revolved around young children, it is now widely accepted that adolescents and young adults continue to depend on their parents in times of need and that having a parental secure base remains important to adjustment at these later stages of development (Allen & Tan, 2016; Rosenthal & Kobak, 2010). Importantly, however, in adolescence and young adulthood, the physical presence of the secure base becomes less important than the individual's confidence in the availability of the parental secure base should a situation arise in which he or she needs help or support. Individuals who have had access to a secure base, particularly under conditions of threat or stress, are thought to develop mental representations or perceptions of parents as available and responsive when needed. These representations of secure base availability in times of threat/stress instill a sense of competence that allows individuals to handle difficult situations on their own or seek assistance when needed (Bowlby, 1973). Relatedly, individuals who have had a consistent secure base (i.e., available and responsive *most* of the time) are better able to cope with stressors and regulate their emotions relative to individuals who did not have a consistent secure base (Cassidy, 1994; Mikulincer & Shaver, 2016). Given that stress can be associated with elevated levels of inflammatory markers (Glaser & Kiecolt-Glaser, 2005), the enhanced emotion regulation and coping skills that having a secure base promotes may result in lower levels of inflammatory markers in adulthood.

Recently, evidence has emerged suggesting that having a secure base prospectively predicts positive health outcomes. For example, adults who had been securely attached to their mothers during infancy (i.e., were able to rely on their mothers as a secure base) reported fewer inflammation-related illnesses at age 32 compared to adults who had been insecurely attached (Puig, Englund, Collins, & Simpson, 2013). Moreover, access to a consistent secure base in toddlerhood was negatively associated with the risk of obesity in adolescence (Anderson, Gooze, Lemeshow, & Whitaker, 2012)—a condition that can also be associated with heightened inflammatory activity (Hotamisligil, 2006). These intriguing findings raise an important question: What is the underlying biological mechanism by which early secure base experiences affect later health outcomes? It has been proposed that inflammatory activity is a key biological pathway through which early social experiences influence later physical health (e.g., Miller et al., 2011). However, no study to date has examined directly whether secure base support (SBS) prospectively predicts biological processes central to

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

inflammation and inflammatory disease in adulthood. The present study aims to address this gap.

## The present study

The present study involved secondary data analysis of three waves of data collected as part of the Maryland Adolescent Development in Context Study (MADICS; principal investigators: Jacquelynne S. Eccles and Arnold J. Sameroff). We utilized these data to conduct a preliminary investigation of whether African American adolescents' (Time 1 [T1]; ~12 years old) and young adults' (T6; ~20 years old) perceptions of their parents as a secure base (i.e., perceived ability to depend on parents in times of need) prospectively predict CRP at age 32 (T8). To establish the unique role of perceived parental SBS in predicting adult CRP, we included measures of perceived general parental and peer support in our analyses to examine the relative influence of each of these variables on adult CRP. We focused on predictors at T1 and T6 because (a) the two time points included similar measures of perceived SBS and (b) the time points span two developmental periods (adolescence and early adulthood), allowing us to test developmental differences in the links between perceived SBS and CRP.

At the outset, we acknowledge the methodological limitations of using existing data to create measures of constructs that were not the central focus of the original study. The measure construction process and methodological limitations are discussed in detail in the "Method" and "Study limitations" sections, respectively. Given the methodological limitations, we have framed the present study as a preliminary investigation.

Given the focus of the larger follow-up study on racial discrimination and health outcomes, recruitment for the T8 CRP measurement was limited to African American MADICS participants. It is particularly important to examine predictors of adult inflammatory activity in African Americans because they tend to have higher CRP than Whites (Khera et al., 2005) and because several inflammation-related diseases disproportionately affect African Americans (CDC, 2013). Also, MADICS participants were sampled from a largely middle-class county in Maryland, thus reducing the confounding of race and SES in the prediction of health outcomes (LaVeist, 2005).

We predicted that adolescents' (T1) perceptions of their parents as a secure base, but not perceptions of their parents' general support or peer support would predict lower CRP at 32 years. In addition to prior evidence linking SBS specifically to inflammation-related health outcomes (e.g., Puig et al., 2013), our differential prediction rests on the notion that parental SBS, but not the other forms of support examined here, serves a stress regulation function that should predict later CRP (see Glaser & Kiecolt-Glaser, 2005, for a review of the links between stress and inflammatory activity). This differential prediction is also supported by prior empirical work that did not find significant associations between measures of general family and peer support and CRP (Yang, Schorp, & Harris, 2014).

Although there is likely some overlap between parental SBS specifically and more general support from parents, we argue that these are two distinct constructs. General parental

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

support, such as praising accomplishments or reminding adolescents to do homework, is certainly important for healthy development, but this is a very different type of experience from being able to depend on parents in times of need or distress (i.e., SBS). Prior research supports conceptualizing these two types of support as distinct constructs and has revealed that the two constructs differentially relate to child outcomes. For example, Leerkes, Blankson, and O'Brien (2009) found that, although maternal supportive responses to child distress and non-distress were significantly correlated, only supportive responses to child distress predicted child outcomes (i.e., social competence and behavioral problems). Therefore, we predicted that adolescents' perceived ability to depend on parents when faced with a problem would be more strongly related to adult CRP compared to more general support from their parents in non-distress contexts.

Furthermore, although adolescence is characterized by increased autonomy from parents and more time spent with peers (Steinberg, 2008), parents continue to be the principal secure base for most adolescents (Rosenthal & Kobak, 2010). In fact, evidence suggests that relying mainly on peers as a secure base in adolescence is linked to negative outcomes, such as greater internalizing and externalizing problems and more risk-taking behavior (Kobak, Herres, Gaskins, & Laurenceau, 2012; Rosenthal & Kobak, 2010). Therefore, we predicted that T1 parental SBS, but not peer support, will predict lower CRP in adulthood.

Exploratory analyses were conducted on links between early adulthood (T6) support and CRP at age 32. On the one hand, many young adults continue to rely on parents, rather than peers, as their principal secure base (Rosenthal & Kobak, 2010). Thus, it is possible that young adults' perceptions of parental SBS, rather than perceived peer support will predict later inflammatory markers. On the other hand, by early adulthood, many individuals have left home and have less direct contact with their parents. Thus, it is possible that in early adulthood, perceived peer support, rather than parental SBS, will predict later inflammatory markers. Further, it is possible that young adults' perceptions of parental SBS and peer support will each have unique effects on CRP at age 32. Alternatively, it is possible that neither parent support nor peer support during early adulthood will predict CRP at age 32: Current theory conceptualizes adolescence as a second sensitive period (Steinberg, 2005) and it is therefore possible that only support experienced during adolescence, but not support later in development (i.e. early adulthood), will predict CRP at age 32.

## Method

### Procedures

We utilized existing data collected as part of MADICS to construct measures of perceived parental SBS, general parental support, and peer support in early adolescence and early adulthood. MADICS is a longitudinal study of normative development among adolescents residing in Prince George's County, Maryland, that began in 1991. When the adolescents were in seventh grade (~12 years old; T1), they and their parent(s) completed a battery of questionnaires during an in-home visit. Three years after high school (~20 years old; T6), participants completed questionnaires via mail. Questionnaires from T1 and T6 assessed a variety of social context variables, including SES, neighborhood quality, peer characteristics, family socialization processes and relationships, transition into work and college, romantic

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

relationships, and perceptions of parents (see Maryland Adolescent Development in Context Study, 2013, for a complete list of measures).

Importantly, MADICS was not designed to assess secure base processes specifically or to test the specific hypotheses we propose in this study. However, the 20-year longitudinal design of MADICS and the collection of inflammatory biomarkers in adulthood provide a unique opportunity to conduct a preliminary investigation of links between parental SBS and adult inflammatory activity. Therefore, parental and peer support measures were created via an a priori process whereby four of the authors (JDJ, KBE, BEB, and JC) independently searched the available MADICS data set and selected items reflecting the constructs of interest. Each author presented his or her selected items at group meetings. Final items were selected through extensive group discussion, and disagreements were resolved through consensus (see “Measures” section below). An important component of this approach is that item selection was determined before any data analyses were performed.

Approximately 20 years after the T1 visit, at age 32 (T8), participants were contacted via telephone and asked to take part in the home visit and blood draw. After agreeing to participate, participants were scheduled for an in-home visit with a research assistant and a phlebotomist. Before the visit (for which they were paid US$50), participants were instructed to refrain from engaging in strenuous exercise, drinking alcohol, smoking, or taking nonprescription medication during the 2 hr before the appointment and from drinking caffeine, brushing their teeth, or eating a meal at least 1 hr before. During the visit, participants first provided written consent and then completed a health information questionnaire and other questionnaires about their emotions and relationships. Then, after a 20-min rest period, they provided 60 cubic centimeters of venous blood via antecubital venipuncture. Participants were then measured for height, weight, and waist–hip ratio. Finally, they completed a questionnaire assessing depressive symptoms. The procedures for the present study were approved by the University of Maryland, College Park, University of California, San Francisco, and Northwestern University Institutional Review Boards.

## Participants

Following receipt of funds to collect data on inflammatory activity from up to 60 African American participants from the original MADICS sample, we enrolled the first individuals who remained in the region, were able to be contacted, agreed to participate, and met none of the exclusionary criteria (mean age = 31.9 years, $SD$ = .45, 63% female, $n$ = 59 because of a scheduling conflict). Exclusionary criteria included (a) major chronic illnesses or active infections; (b) factors that could influence cardiovascular functioning, such as pregnancy; (c) factors that could influence endocrine functioning, such as recent administration of anesthesia; (d) factors that could affect the immune system, such as the use of corticosteroids or presence of an inflammatory disease (e.g., Crohn’s Disease); and (e) anxiety related to venipuncture (i.e., a rating of greater than 4 on a 7-point scale). Median family income of participants in the present study at T8 was between $65,000 and $69,000.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

## Measures

**Adolescent and early adult parental secure base support.—**To capture adolescents' (T1) perceptions of parental secure base support, we selected the following item from the MADICS data set: "When you have a social/personal problem at school how often can you depend on your parent(s) to help you out?" Response choices included: 1 (*almost never*), 2 (*not too often*), 3 (*about half the time*), 4 (*fairly often*), or 5 (*almost always*). At T6 (early adulthood), participants completed a nearly identical item: "When you have a social/personal problem, how often can you depend on your parent(s) to help you out?" These questions are the only items available in the MADICS data set that assess precisely what it means to have a secure base as defined within the core tenets of attachment theory (i.e., ability to consistently depend on attachment figure(s) during times of need; Bowlby, 1969/1982). Because of the conceptual difference between having a secure base consistently (i.e., *almost always*) versus inconsistently (i.e., *almost never to fairly often*), we chose to dichotomize the secure base items to reflect either having a consistent secure base (a rating of 5, dummy coded as 1) or not (a rating of 1–4, dummy coded as 0).

**Adolescent and early adult general support from parents.—**At T1, we selected 8 Likert-type items to create a scale of perceived general parental support ($\alpha$ = .75; e.g. "My parent(s) praise me for doing well," and "My parent(s) encourage me to do my best at everything I do"), each rated from 1 (*almost never*) to 5 (*almost always*). At T6, we selected 6 Likert-type items to create a scale of perceived general parental support (3 items related to maternal support and the same 3 items related to paternal support; $\alpha$ = .87). Sample items include, "During the past month, how often did your father help you to feel good about yourself?" and "During the past month, how often did your mother help you do something that's important to you?" rated from 1 (*never*) to 5 (*almost every day*).

**Adolescent and early adult peer support.—**At T1, we selected two Likert-type items to capture perceived peer support ($\alpha$ = .70), one of which directly parallels the parental SBS item (i.e., "When you have a social/personal problem at school, how often can you depend on friends to help you out?" and "When you're having trouble on schoolwork, how often do you go to your friends for help?"). Adolescents responded on a scale ranging from 1 (*almost never*) to 5 (*almost always*). At T6, we selected 5 Likert-type scale items with various rating scales to measure perceived peer support ($\alpha$ = .84). These items asked about "the friends that you spend most of your time with" (e.g., "How often do the friends that you spend most of your time with help you do something that's important to you?" and "How close do you feel to these friends?").

**Inflammatory marker.—**We used serum CRP as our biomarker of inflammatory activity at age 32 (T8). Serum from the home visit venipuncture was harvested by centrifugation and frozen. After all samples were obtained, CRP was measured using high-sensitivity chemiluminescence.

## Inflammation-related confounds

**Health information.—**The health questionnaire at age 32 assessed a number of health-related confounds, including the following which are used in the present study: sex,

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

use of anti-inflammatory medication or birth control pills within the past week; alcohol consumption within the past week; and same-day exercise, number of cigarettes smoked, and caffeine consumption. Body mass index (BMI) was a composite of direct measurements of height and weight. Depressive symptoms were measured with the widely used 21-item Beck Depression Inventory-II (BDI-II; α = .90; Beck, Steer, & Brown, 1996).

**T1 family SES.—**T1 SES was measured using the composite created for use in MADICS (see Brodish et al., 2011). This composite includes parent-reported variables from the T1 questionnaires: family income, highest education level completed by either parent, and highest occupational status of either parent.

## Results

### Missing data and outliers

We had complete data at T1 for 57 participants. At T6, we had complete data for 45 participants. At T8, we had complete data for 58 participants. Following common practice, CRP values greater than 10 mg/L ($n = 4$) were treated as outliers and excluded from the principal analyses (Pearson et al., 2003). However, we reran all of our analyses including cases with CRP values greater than 10 mg/L to ensure that the removal of the subjects with high CRP values was not driving the results.

### Descriptive statistics

Means and standard deviations of key study variables are presented in Table 1. CRP values were skewed (skewness = 1.77); therefore, we used a log transformation to improve the distribution of this variable (skewness after transforming = .04). Correlations among key study variables are presented in Table 2. Log CRP was positively correlated with BMI ($r = .61$, $p < .01$). Additionally, log CRP was negatively correlated with parental SBS at T1 ($r_{pb} = -.33$, $p < .05$) and negatively correlated with a concurrent measure of exercise on the same day as the blood draw ($r_{pb} = -.27$, $p < .05$).

### Principal analyses

We performed two separate hierarchical regression analyses to test whether the support variables at T1 and T6, respectively, predicted log CRP at age 32. In Step 1 of each model, we entered concurrent BMI, BDI scores, smoking, alcohol use, caffeine intake, birth control use, anti-inflammatory medication use, exercise, T1 SES, and sex to control for health-related confounds. In Step 2 of each model, we entered our predictor variables: perceived parental SBS, general parental support, and peer support.

### Support in early adolescence (T1) and adulthood CRP

In the first model we tested, the variables in Step 1 explained 58% of the variance in CRP levels in adulthood. The T1 variables entered in Step 2 explained an additional 9% of the variance, $\Delta F(3, 38) = 3.32$, $p < .05$; listwise $n = 52$. Adolescents' perceptions of their parents as a secure base significantly predicted lower adult CRP ($b = -.92$, $SE = .34$ $p < .05$; see Table 3). That is, participants who reported having a consistent parental secure base in early adolescence had lower log CRP in adulthood compared to adolescents who did not perceive

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

their parents as a consistent secure base. In contrast, neither perceived general parental support nor peer support in adolescence significantly predicted adult CRP. Results of the analysis including CRP outliers yielded identical conclusions.

### Support in early adulthood (T6) and adulthood CRP

In the second model we tested, the variables in Step 1 explained 64% of the variance in adulthood CRP. None of the T6 support variables significantly predicted adult log CRP nor did they explain a significant amount of additional variance over and above the control variables (listwise $n = 42$; see Table 3). Results of the analysis including CRP outliers yielded identical conclusions. Additionally, for the T6 analyses, we used multiple imputation (including all analysis variables and theoretically related auxiliary variables in the imputation model) to impute missing data, giving us 20 complete imputed data sets. Results using the imputed data sets with complete data were similar to those obtained using listwise deletion, and the resulting conclusions were identical.

## Discussion

In recent years, attachment researchers have begun to tackle the intriguing question of how attachment relates to physical health outcomes. A small but growing body of evidence suggests that early attachments and the presence of a parental secure base are associated with subsequent physical health (Anderson et al., 2012; Anderson & Whitaker, 2011; Puig et al., 2013). Researchers have proposed that inflammatory activity may be the biological pathway through which early social experiences “get under the skin” to affect adult health (Miller et al., 2011). However, researchers have yet to examine how having a secure base relates to inflammatory markers that are associated with adult health outcomes. In this preliminary study, we utilized data from a 20-year longitudinal study to examine whether adolescents’ and young adults’ perceptions of parental SBS predict adult CRP, a biomarker of inflammatory activity that is associated with a number of chronic diseases of aging.

As predicted, we found that adolescents’ (T1) perceptions of parental SBS predicted lower CRP in adulthood, even after controlling for biobehavioral factors known to influence CRP. Our exploratory analysis of parent and peer support in early adulthood (T6) revealed that none of the support variables in early adulthood predicted CRP at age 32. These findings highlight the potential importance of a parental secure base, the most central construct in attachment theory (Bowlby, 1988), for subsequent physical health.

### Developmental timing

Why might parental SBS in early adolescence, but not in early adulthood, be predictive of CRP at age 32? Given the lack of research in this area, we can only begin to speculate about this pattern of results. Adolescence is increasingly viewed as a second sensitive period during which social, cognitive, and physiological changes have long-lasting influences on development (Steinberg, 2005). It is possible that secure base experiences with caregivers during this period are particularly influential for later physical health and biological processes relevant to health, as they appear to be during early childhood. Another possibility is that parents may play a more important role in their children’s management of stress in

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

adolescence compared to early adulthood, when many children have left home and may have more limited contact with their parents. Continued longitudinal research will help answer questions about the relative timing of protective factors for later inflammation.

### Stress regulation as a potential mechanism

As noted in the introduction, one possible mechanism by which parental SBS may affect adult inflammatory activity is through its influence on regulatory systems related to the stress response (see Ehrlich, Miller, Jones, & Cassidy, 2016, for more detail). There is mounting evidence that caregiving experiences influence the calibration and ongoing regulation of the child's hypothalamic–pituitary–adrenal (HPA) axis (Gunnar & Quevedo, 2007), which plays an integral role in inflammatory activity. Future studies should examine HPA axis functioning as a potential mechanism linking early secure base experiences with adult inflammatory activity.

### Family race/ethnicity

Given evidence that African Americans tend to have higher CRP levels than Whites (Khera et al., 2005), and that several inflammation-related diseases disproportionately affect African Americans (CDC, 2013), our focus on African American MADICS participants is a strength. However, it will be important to examine how parental and peer support relate to inflammatory markers in other racial/ethnic groups. Some evidence suggests that early experiences may differentially affect inflammatory functioning in African Americans and Whites. For example, Slopen et al. (2010) found that early life adversity (retrospectively reported) was associated with heightened inflammatory activity in adulthood among African American participants but not among White participants. Furthermore, there is evidence for racial/ethnic differences in the nature of family interactions and peer relationships. For example, relative to Whites, African Americans report helping out family members more often and having more daily contact with family members. However, Whites report providing more support to and having more frequent interactions with friends compared to African Americans (Taylor, Chatters, Woodward, & Brown, 2013). Given these differences in family and peer relationships, it is possible that the links between parental and peer support during adolescence and inflammatory activity in adulthood differ as a function of race/ethnicity. Future studies with racially heterogeneous samples could examine these possibilities.

### Study limitations and additional future directions

It is important to weigh study strengths against the methodological limitation that plagues many studies that take advantage of existing data from a longitudinal study—the use of available data to construct measures and test hypotheses that were not a central focus of the original study. Our construction of measures of perceived parent and peer support using the variables at our disposal (i.e., those collected with other aims in mind) inevitably means that we are using measures lacking established psychometric properties. However, access to 20 years of data on African American youth from the MADICS sample, along with the addition of a blood draw and the assessment of inflammation-related covariates in adulthood, provided a rare opportunity to test novel questions about potential links between parental SBS and a biomarker of adult inflammatory activity. Thus, despite the

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

methodological limitations, we view this preliminary study as an important first step in furthering the understanding of how attachment-related processes in adolescence and early adulthood relate to biological markers of physical health in adulthood.

We now elaborate on several limitations of our measures and offer suggestions for future research. First, the MADICS data set did not include direct assessments of attachment or parental SBS. Therefore, we measured SBS with a single item reflecting whether or not the participants felt they could depend on their parents in times of need or distress. Although we feel that this item captures the most central aspect of the secure base construct, it is unclear how this measure relates to established measures of attachment or secure base processes. Thus, additional longitudinal research employing more comprehensive measures with established psychometric properties is warranted to further examine the conclusions of this preliminary study.

Second, the measures of perceived parental SBS at T1 and T6 were very similar but not identical. The T1 item inquired about social/personal problems at school, whereas the T6 item did not reference a specific environmental context for the social/personal problems. Thus, we cannot rule out the possibility that this minor measurement difference is affecting the pattern of results observed in the present study.

Third, our measures of perceived parental SBS and general support from parents did not distinguish between perceived support from mothers and fathers. Although both mothers and fathers are important contributors to a variety of aspects of children's development (Lamb & Lewis, 2013), evidence suggests that mothers and fathers are not equally likely to serve as the principal attachment figure for adolescents and young adults (Rosenthal & Kobak, 2010). Thus, it is possible that perceptions of SBS from mothers versus fathers could differentially relate to adult inflammatory activity.

Fourth, in this study perceived parent and peer support were assessed with self-report measures. Although self-report rating scales are commonly used in the social and health sciences, they are subject to various reporting biases (e.g., Paulhus & Vazire, 2007; Rosenbaum & Valsiner, 2011). Future studies should include interviews or observational measures of parental and peer support.

In addition, although samples of this size are not uncommon in studies of immune functioning (e.g., Fuligni et al., 2009), our sample size limited the analyses we were able to perform. For example, we were unable to test all T1 and T6 predictors in the same model. Larger sample sizes will allow examination of not only a wider set of predictors in the same model but also of more complex research questions about buffering and exacerbating factors and about mechanisms that may mediate the associations observed in the present study. Furthermore, this prospective study began in early adolescence, and as such, we are unable to test hypotheses about the role of experiences with parents during childhood, whether there are earlier sensitive periods during which experiences with parents exert a particularly large influence on later inflammatory activity, and how these processes unfold over time. Future longitudinal studies including assessments that begin earlier in childhood and extend through adolescence and adulthood could address these issues.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

## Conclusion

Advancing understanding of the mechanisms through which close relationship processes might predict health outcomes is increasingly viewed as a topic of considerable urgency (Miller et al., 2011; Pietromonaco, Uchino, & Dunkel Schetter, 2013). This study is the first to document a link between parental SBS in early adolescence and a biomarker of inflammatory activity in adulthood. We hope this preliminary study spurs future investigations into the links between secure base processes and health that utilize measures with established psychometric properties.

The presence of a secure base may be protective for later health problems (Puig et al., 2013), and the findings from the present study demonstrate one possible biological pathway that could explain how these experiences with caregivers could translate to reduced risk for illnesses in adulthood (i.e., through inflammatory activity). These findings add to a growing literature on the links between social experiences and physical health and further highlight the potential benefits of considering close relationships, alongside diet and exercise, as predictors of physical health outcomes.

## Acknowledgments

We thank Todd Bartko, Elaine Belansky, Heather Bouchey, Nick Butler, Celina Chatman, Courtney Cogburn, Diane Early, Kari Fraser, Thomas Fuller-Rowell, Leslie Gutman, Katie Jodl, Ariel Kalil, Linda Kuhn, Sarah Lord, Karen McCarthy, Alice Michael, Melanie Overby, Robert Roeser, Sherri Steele, Erika Taylor, Janice Templeton, Cindy Winston, and Carol Wong for their support in this project.

### Funding

The authors disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: This research was supported by the MacArthur Network on Successful Adolescent Development in High Risk Settings, Spencer Foundation grant MG #200000275, NICHD grant # R01 HD048970, NIA grant # RC2 AG0367800, NICHD grant # F32 HD076563, and NIDA grant # F31 DA033848.

## References

Ainsworth MDS (1967). Infancy in Uganda: Infant care and the growth of love. Baltimore, MD: Johns Hopkins University Press.

Allen JP, & Tan JS (2016). The multiple facets of attachment in adolescence. In Cassidy J & Shaver PR (Eds.), Handbook of attachment: Theory, research, and clinical applications (3rd ed., pp. 399–415). New York, NY: Guilford.

Anderson SE, Gooze RA, Lemeshow S, & Whitaker RC (2012). Quality of early maternal-child relationship and risk of adolescent obesity. Pediatrics, 129, 132–140. doi:10.1542/peds.2011-0972 [PubMed: 22201144]

Anderson SE, & Whitaker RC (2011). Attachment security and obesity in US preschool aged children. Archives of Pediatric and Adolescent Medicine, 165, 235–242. doi:10.1001/archpediatrics.2010.292

Beck AT, Steer RA, & Brown GK (1996). Manual for the Beck Depression Inventory-II. San Antonio, TX: Psychological Corporation.

Bowlby J (1969/1982). Attachment and loss: Vol. 1. Attachment. New York, NY: Basic Books.

Bowlby J (1973). Attachment and loss: Vol. 2. Separation. New York, NY: Basic Books.

Bowlby J (1988). A secure base: Parent-child attachment and healthy human development. London, England: Routledge.

Brodish AB, Cogburn CD, Fuller-Rowell TE, Peck S, Malanchuk O, & Eccles JS (2011). Perceived racial discrimination as a predictor of health behaviors: The moderating role of gender. Race and Social Problems, 3, 160–169. doi:10.1007/s12552-011-9050-6 [PubMed: 22844386]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

Cassidy J (1994). Emotion regulation: Influences of attachment relationships. Monographs of the Society for Research in Child Development, 59, 228–283. doi:10.2307/1166148 [PubMed: 7984163]

Cassidy J, & Shaver PR (2016). Handbook of attachment: Theory, research, and clinical applications (3rd ed.). New York, NY: Guilford.

Centers for Disease Control and Prevention. (2013). CDC health disparities and inequalities report – United States, 2013. Morbidity and Mortality Weekly Report, 62, 1–186. [PubMed: 23302815]

Chen E, Miller GE, Kobor MS, & Cole SW (2011). Maternal warmth buffers the effects of low early-life socioeconomic status on pro-inflammatory signaling in adulthood. Molecular Psychiatry, 16, 729–737. doi:10.1038/mp.2010.53 [PubMed: 20479762]

Chung HY, Cesari M, Anton S, Marzetti E, Giovannini S, Seo AY, … Leeuwenburgh C (2009). Molecular inflammation: Underpinnings of aging and age-related diseases. Ageing Research Reviews, 8, 18–30. doi:10.1016/j.arr.2008.07.002 [PubMed: 18692159]

Danese A, Pariante CM, Caspi A, Taylor A, & Poulton R (2007). Childhood maltreatment predicts adult inflammation in a life-course study. Proceedings of the National Academy of Sciences, 104, 1319–1324. doi:10.1073/pnas.0610362104

Dickerson SS, Gable SL, Irwin MR, Aziz N, & Kemeny ME (2009). Social-evaluative threat and proinflammatory cytokine regulation: An experimental laboratory investigation. Psychological Science, 20, 1237–1244. doi:10.1111/j.1467-9280.2009.02437.x [PubMed: 19754527]

Donoho CJ, Crimmins EM, & Seeman TE (2013). Marital quality, gender, and markers of inflammation in the MIDUS cohort. Journal of Marriage and Family, 75, 127–141. doi:10.1111/j.1741-3737.2012.01023.x [PubMed: 24700968]

Ehrlich KB, Miller GE, & Chen E (2016). Childhood adversity and adult physical health. In Cicchetti D, Developmental Psychopathology (3rd ed., Vol. 4, pp. 1–42). Hoboken, NJ: Wiley.

Ehrlich KB, Miller GE, Jones JD, & Cassidy J (2016). Attachment and psychoneuroimmunology. In Cassidy J & Shaver PR (Eds.), Handbook of attachment: Theory, research, and clinical applications (3rd ed., pp. 180–201). New York, NY: Guilford.

Fuligni AJ, Telzer EH, Bower J, Irwin MR, Kiang L, & Cole SW (2009). Daily family assistance and inflammation among adolescents from Latin American and European backgrounds. Brain, Behavior, and Immunity, 23, 803–809. doi:10.1016/j.bbi.2009.02.021 [PubMed: 19275931]

Glaser R, & Kiecolt-Glaser JK (2005). Stress-induced immune dysfunction: Implications for health. Nature Reviews Immunology, 5, 243–251. doi:10.1038/nri1571

Gunnar M, & Quevedo K (2007). The neurobiology of stress and development. Annual Review of Psychology, 58, 145–173. doi:10.1146/annurev.psych.58.110405.085605

Hotamisligil GS (2006). Inflammation and metabolic disorders. Nature, 444, 860–867. doi:10.1038/nature05485 [PubMed: 17167474]

Khera A, McGuire DK, Murphy SA, Stanek HG, Das SR, Vongpatanasin W, … de Lemos JA (2005). Race and gender differences in C-reactive protein levels. Journal of the American College of Cardiology, 46, 464–469. doi:10.1016/j.jacc.2005.04.051 [PubMed: 16053959]

Kobak R, Herres J, Gaskins C, & Laurenceau J (2012). Teacher–student interactions and attachment states of mind as predictors of early romantic involvement and risky sexual behaviors. Attachment & Human Development, 14, 289–303. [PubMed: 22537525]

Lamb ME, & Lewis C (2013). Father-child relationships. In Tamis-LeMonda CS & Cabrera N (Eds.), Handbook of father involvement (2nd ed., pp. 119–134). New York, NY: Psychology Press.

LaVeist TA (2005). Disentangling race and socioeconomic status: A key to understanding health inequalities. Journal of Urban Health, 82, 26–34. doi:10.1093/jurban/jti061

Leerkes EM, Blankson A, & O'Brien M (2009). Differential effects of maternal sensitivity to infant distress and nondistress on social-emotional functioning. Child Development, 80, 762–775. doi:10.1111/j.1467-8624.2009.01296.x [PubMed: 19489902]

Maryland Adolescent Development in Context Study. (2013). Retrieved November 23, 2013, from http://www.rcgd.isr.umich.edu/pgc/home.htm

Mikulincer M, & Shaver PR (2016). Adult attachment and emotion regulation. In Cassidy J & Shaver PR (Eds.), Handbook of attachment: Theory, research, and clinical applications (3nd ed., pp. 507–533). New York, NY: Guilford.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

Miller GE, Chen E, & Parker KJ (2011). Psychological stress in childhood and susceptibility to the chronic diseases of aging: Moving toward a model of behavioral and biological mechanisms. Psychological Bulletin, 137, 959–997. doi:10.1037/a0024768 [PubMed: 21787044]

Paulhus DL, & Vazire S (2007). The self-report method. In Robins RW, Fraley RC, & Krueger RF (Eds.), Handbook of research methods in personality psychology (pp. 224–239). New York, NY: Guilford.

Pearson TA, Mensah GA, Alexander RW, Anderson JL, Cannon RO, Criqui M, … Vinicor F (2003). Markers of inflammation and cardiovascular disease: Application to clinical and public health practice. A statement for healthcare professionals from the Centers for Disease Control and Prevention and the American Heart Association. Circulation, 107, 499–511. doi:10.1161/01.CIR.0000052939.59093.45 [PubMed: 12551878]

Pietromonaco PR, Uchino B, & Dunkel Schetter C (2013). Close relationship processes and health: Implications of attachment theory for health and disease. Health Psychology, 32, 499–513. [PubMed: 23646833]

Pollitt RA, Kaufman JS, Rose KM, Diez-Roux AV, Zeng D, & Hess G (2007). Early-life and adult socioeconomic status and inflammatory risk markers in adulthood. European Journal of Epidemiology, 22, 55–66. doi:10.1007/s10654-006-9082-1 [PubMed: 17225957]

Puig J, Englund MM, Simpson JA, & Collins W (2013). Predicting adult physical illness from infant attachment: A prospective longitudinal study. Health Psychology, 32, 409–417. doi:10.1037/a0028889 [PubMed: 22823067]

Ridker P (2003). Clinical application of C-reactive protein for cardiovascular disease detection and prevention. Circulation, 107, 363–369. doi:10.1161/01.CIR.0000053730.47739.3C [PubMed: 12551853]

Ridker PM (2007). Inflammatory biomarkers and risks of myocardial infarction, stroke, diabetes, and total mortality: Implications for longevity. Nutrition Reviews, 65, S253–S259. doi:10.1111/j.1753-4887.2007.tb00372.x [PubMed: 18240558]

Rosenbaum PJ, & Valsiner J (2011). The un-making of a method: From rating scales to the study of psychological processes. Theory & Psychology, 21, 47–65. doi:10.1177/0959354309352913

Rosenthal NL, & Kobak R (2010). Assessing adolescents' attachment hierarchies: Differences across developmental periods and associations with individual adaptation. Journal of Research on Adolescence, 20, 678–706. doi:10.1111/j.1532-7795.2010.00655.x [PubMed: 22545000]

Shonkoff JP, & Phillips DA (Eds.). (2000). From neurons to neighborhoods. Washington, DC: National Academy Press.

Slopen N, Lewis TT, Gruenewald TL, Mujahid MS, Ryff CD, Albert MA, & Williams DR (2010). Early life adversity and inflammation in African Americans and Whites in the Midlife in the United States Survey. Psychosomatic Medicine, 72, 694–701. doi:10.1097/PSY.0b013e3181e9c16f [PubMed: 20595419]

Steinberg L (2005). Cognitive and affective development in adolescence. Trends In Cognitive Sciences, 9, 69–74. doi:10.1016/j.tics.2004.12.005 [PubMed: 15668099]

Steinberg L (2008). Adolescence (8th ed.). New York, NY: McGraw-Hill.

Taylor RJ, Chatters LM, Woodward AT, & Brown E (2013). Racial and ethnic differences in extended family, friendship, fictive kin, and congregational informal support networks. Family Relations, 62, 609–624. doi:10.1111/fare.12030 [PubMed: 25089067]

Whisman MA, & Sbarra DA (2012). Marital adjustment and interleukin-6 (IL-6). Journal of Family Psychology, 26, 290–295. doi:10.1037/a0026902 [PubMed: 22229879]

Yang YC, Schorpp K, & Harris KM (2014). Social support, social strain and inflammation: Evidence from a national longitudinal study of U.S. adults. Social Science & Medicine, 107, 124–135. doi:10.1016/j.socscimed.2014.02.013 [PubMed: 24607674]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

**Table 1.**

Means, standard deviations, and proportions for key study variables.

| Continuous variables (*N*) | *M* (*SD*) |
|---|---|
| Body mass index (59) | 29.50 (6.08) |
| BDI score (59) | 0.46 (0.41) |
| Cigarettes in past day (59) | 0.64 (1.84) |
| Alcoholic drinks in past week (58) | 3.97 (6.14) |
| Caffeinated drinks in past day (59) | 0.47 (0.72) |
| SES (59) | 0.05 (0.87) |
| GPS—Time 1 (58) | 3.82 (0.75) |
| PS—Time 1 (58) | 3.07 (l.02) |
| GPS-Time 2 (45) | 3.19 (l.l0) |
| PS—Time 2 (46) | 3.92 (0.73) |
| Raw CRP (55)[a] | l.92 (2.29) |
| Log-transformed CRP (55)[a] | −0.02 (l.22) |
| **Dichotomous variables (*N*)** | **% Reported** |
| Sex (59) | 63% female |
| Birth control use (59) | l4% |
| AIM (59) | 7l% |
| Exercise engagement (59) | l0% |
| High SBS—Time 1 (58) | 76% |
| High SBS—Time 2 (46) | 46% |

*Note*. CRP = C-reactive protein; SBS = secure base support; AIM = anti-inflammatory medication use; SES = socioeconomic status; PS = peer support; BDI = Beck Depression Inventory; GPS = general parental support.

[a]Outliers removed.

Author Manuscript

Author Manuscript

Author Manuscript

**Table 2.**

Correlation matrix of key study variables.

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. Sex | – | | | | | | | | | | | | | | | | |
| 2. BMI | −.15 | – | | | | | | | | | | | | | | | |
| 3. BDI score | −.11 | .08 | – | | | | | | | | | | | | | | |
| 4. Cigarettes | .31* | −.13 | −.02 | – | | | | | | | | | | | | | |
| 5. Alcohol | .51** | .09 | −.04 | .43** | – | | | | | | | | | | | | |
| 6. Caffeine | −.16 | .15 | .25 | .33* | .15 | – | | | | | | | | | | | |
| 7. SES | .13 | .13 | −.22 | .01 | .18 | .29* | – | | | | | | | | | | |
| 8. BC | −.31* | .18 | −.10 | −.14 | −.22 | −.05 | .09 | – | | | | | | | | | |
| 9. AIM | −.10 | .16 | .01 | −.16 | .05 | .00 | .15 | .19 | – | | | | | | | | |
| 10. Exercise | −.03 | −.23 | −.11 | −.09 | −.01 | −.06 | .14 | .03 | .03 | – | | | | | | | |
| 11. SBS (T1) | .03 | −.02 | .06 | .00 | .03 | −.05 | −.06 | −.01 | .10 | −.07 | – | | | | | | |
| 12. GPS (T1) | .02 | −.07 | −.28* | −.19 | .02 | .18 | .38** | −.14 | −.22 | .27* | .28* | – | | | | | |
| 13. PS (T1) | −.30* | .22 | .02 | −.20 | −.17 | .04 | .12 | −.03 | −.14 | .01 | −.06 | .19 | – | | | | |
| 14. SBS (T6) | .15 | −.20 | .03 | .22 | .26 | −.05 | −.12 | −.10 | −.00 | .10 | .33* | .12 | −.13 | – | | | |
| 15. GPS (T6) | −.08 | −.01 | −.19 | .18 | .18 | −.18 | −.09 | −.10 | .10 | −.02 | .24 | .15 | .20 | .46** | – | | |
| 16. PS (T6) | −.20 | −.11 | −.26 | −.20 | −.15 | −.36* | −.15 | .02 | −.02 | −.02 | .21 | .14 | −.01 | −.05 | .26 | – | |
| 17. Log CRP | −.01 | .61** | .20 | .12 | .06 | .02 | .15 | .14 | −.08 | −.27* | −.33* | −.25 | .01 | −.01 | .03 | −.03 | – |

Note. CRP = C-reactive protein; BMI = body mass index; BDI = Beck Depression Inventory; SES = socioeconomic status; BC = birth control (1 = yes); AIM = anti-inflammatory medication use (1 = no); SBS = secure base support (coded 0 = does not perceive parents as consistent secure base; 1 = does perceive parents as consistent secure base); GPS = general parent support; PS = peer support; T1 = Time 1; T6 = Time 6; Sex (0 = female; 1 = male).

* $p < .05$

** $p < .01$.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

**Table 3.**

Unstandardized regression coefficients and standard errors for variables predicting log CRP.

| | Time 1 | | | | Time 6 | | | |
|---|---|---|---|---|---|---|---|---|
| **Variable** | ***b*** | ***SE*** | **95% CI** | **$\Delta R^2$** | ***b*** | ***SE*** | **95% CI** | **$\Delta R^2$** |
| Step 1 | | | | .58** | | | | .63** |
| Sex | −.12 | .33 | [−.77, .54] | | −.19 | .36 | [−.92, .56] | |
| BMI | .14** | .02 | [.09, .19] | | .18** | .03 | [.11, .24] | |
| BDI score | .95* | .35 | [.23, 1.66] | | .93* | .42 | [.08, 1.78] | |
| Cigarettes | .22* | .08 | [.05, .39] | | .18 | .09 | [−.01, .36] | |
| Alcohol | −.02 | .03 | [−.07, .03] | | −.04 | .04 | [−.11, .04] | |
| Caffeine | −.47* | .23 | [−.93, −.01] | | −.48 | .24 | [−.98, .02] | |
| SES | .39* | .18 | [.03, .75] | | .24 | .22 | [−.22, .69] | |
| Birth control | .47 | .43 | [−.38, 1.33] | | .43 | .45 | [−.49, 1.35] | |
| AIM | −.32 | .29 | [−.90, .26] | | −.30 | .32 | [−.96, .35] | |
| Exercise | −.45 | .42 | [−1.29, .39] | | −.28 | .47 | [−1.24, .68] | |
| Step 2 | | | | .09* | | | | .02 |
| Sex | −.25 | .32 | [−.90, .40] | | −.18 | .41 | [−1.01, .65] | |
| BMI | .14** | .02 | [.09, .18] | | .18** | .03 | [.12, .25] | |
| BDI score | 1.06** | .36 | [.32, 1.80] | | .95* | .44 | [.05, 1.86] | |
| Cigarettes | .22* | .09 | [.04, .40] | | .18 | .10 | [−.02, .38] | |
| Alcohol | −.02 | .02 | [−.07, .03] | | −.05 | .04 | [−.12, .03] | |
| Caffeine | −.57* | .24 | [−1.05, −.09] | | −.47 | .26 | [−.99, .06] | |
| SES | .41* | .17 | [.06, .76] | | .27 | .23 | [−.20, .74] | |
| Birth control | .24 | .43 | [−.62, 1.11] | | .46 | .48 | [−.54, 1.43] | |
| AIM | −.30 | .31 | [−.92, .33] | | −.26 | .34 | [−.95, .43] | |
| Exercise | −.65 | .42 | [−1.50, .20] | | −.28 | .49 | [−1.28, .72] | |
| SBS | −.92* | .34 | [−1.60. −.23] | | .43 | .34 | [−.28, 1.13] | |
| GPS | .11 | .27 | [−.43, .65] | | −.08 | .16 | [−.41, .26] | |
| PS | −.18 | .13 | [−.45, .09] | | .12 | .28 | [−.45, .69] | |

*Notes.* SBS coded 0 = does not perceive parents as consistent secure base; 1 = does perceive parents as consistent secure base. Sex (0 = *female*; 1 = *male*). CRP = C-reactive protein; BMI = body mass index; BDI = Beck Depression Inventory; SES = socioeconomic status; AIM = anti-inflammatory medication use; SBS = secure base support; GPS = general parental support; PS = peer support.

* $p < .05$

** $p < .01$.