A Preliminary Investigation of Attachment Style and Inflammation in African American Young Adults

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Abstract

Individuals’ social experiences are associated with their mental health, physical health, and even mortality. Over the last 30 years, researchers have examined the ways in which these social experiences might be associated with chronic inflammation—a component underlying many of the chronic diseases of aging. Little research, however, has examined the role of adults’ attachment style as a specific social component that might be associated with inflammation. In the present study, we utilized data from a sample of 59 African American adults from the Maryland Adolescent Development in Context Study (MADICS) to examine the links between attachment avoidance and attachment anxiety and C-reactive protein (CRP) and interleukin (IL)-6. After controlling for demographic characteristics, body mass index, and depressive symptoms, attachment avoidance and anxiety were associated with IL-6 but not CRP. This study adds to the growing body of research identifying the wide range of social experiences associated with inflammation and further suggests that attachment relationship experiences may have implications for biological processes relevant to many chronic diseases of aging.

Keywords

attachment avoidance; attachment anxiety; inflammation; depressive symptoms

Across the lifespan, the quality of individuals’ social experiences have far-reaching links to individuals’ mental health (Whisman, 2017), physical health (Cohen, 2004; see Robles, Slatcher, Trombello, & McGinn, 2014 for a meta-analysis on romantic relationship quality...
and health), and even mortality (Holt-Lunstad, Smith, & Layton, 2010). In particular, compared to individuals with poor quality social relationships, individuals who are more socially integrated and who have better quality relationships have fewer physical health symptoms and lower rates of chronic disease (Cohen, 2004; Robles et al., 2014). Further, in a comprehensive meta-analysis of 148 studies, Holt-Lunstad et al. (2010) found that individuals with stronger social relationships have a 50% increased likelihood of survival over the study period (mean study period = 9 years), relative to their more isolated peers. The magnitude of the impact of poor social relationships on negative physical health outcomes has been compared to the effect of many health behaviors, including smoking, unhealthy diets, and sedentary lifestyles (Holt-Lunstad, Robles, & Sbarra, 2017; Holt-Lunstad et al., 2010).

In an effort to better understand how social experiences may “get under the skin” to influence physical health and mortality risk, researchers have been studying the ways in which social experiences are associated with biological mechanisms, with a particular interest in markers of inflammation (Ehrlich, Miller, & Chen, 2016; Kiecolt-Glaser, Gouin, & Hantsoo, 2010). Inflammation occurs when cells of the innate immune system gather at the site of an infection or injury in an attempt to eliminate pathogens, rid the body of infected tissue, repair damage, and begin the process of healing. This response is essential to survival, but it must be carefully regulated so that inflammation does not become persistent, as chronic inflammation has been implicated in many chronic diseases of aging (Danesh, Collins, Appleby, & Peto, 2000; Libby, Ridker, & Hansson, 2009; Ridker, 2007). The process of inflammation is orchestrated by signaling molecules known as cytokines, which are proteins that are released by immune cells and the damaged tissue. Researchers often examine levels of cytokines circulating in the blood, including interleukin (IL)-1, IL-6, and tumor necrosis factor alpha (TNF-α), as rough estimates of ongoing inflammatory activity. In addition, researchers often measure C-reactive protein (CRP), a molecule produced by the liver during inflammation, as an index of low-grade inflammation.

Stressful experiences are thought to alter inflammatory processes via the autonomic nervous system. Fibers from the sympathetic nervous system connect brain regions involved in emotion processing and regulating to the lymphoid organs, where immune responses take place. Evidence from animal studies suggests that stressful experiences can increase the density of these fibers (Sloan et al., 2007), which release neurotransmitters that alter white blood cell activity. In addition, hormonal pathways influence immune processes. Immune cells respond to cortisol, oxytocin, and other molecules whose expression can be affected by stress (e.g., Fries, Ziegler, Kurian, Jacoris, & Pollak, 2005; Miller et al., 2009).

In addition, mounting evidence suggests that various social experiences are related to markers of systemic inflammation (e.g., Fagundes, Bennett, Derry, & Kiecolt-Glaser, 2011; Gouin et al., 2009; Kiecolt-Glaser et al., 2005). For example, one study found that married couples who were rated as high in hostility during an observed conflict discussion had greater increases in circulating IL-6 and TNF-α the morning after the conflict compared to less hostile couples (Kiecolt-Glaser et al., 2005). Similarly, using data from the Midlife Development in the United States (MIDUS) study, Yang and colleagues (2014) found that support and strain across relationships were associated with inflammation ten years later,
although social strain appeared to have stronger links to inflammation compared to social support. These findings are in line with a recent meta-analysis of 41 studies, which found that social support and integration were related to lower levels of inflammation (Uchino et al., 2018).

Despite this surge in interest in how various aspects of social relationships are associated with inflammation, few studies have examined links between systemic inflammation and individuals’ attachment style. Attachment style in adulthood reflects individuals’ expectations, emotion regulation strategies, and comfort in close relationships (Brennan, Clark, & Shaver, 1998; Shaver & Mikulincer, 2002). Individual differences in adult attachment style have been defined along two dimensions. Attachment avoidance captures individuals’ discomfort with intimacy and avoidance of relying on close relationship partners for support. In contrast, attachment anxiety reflects individuals’ fears of rejection and abandonment and their desires for closeness in relationships. To date, research on the correlates of adults’ attachment style has focused principally on individuals’ thoughts, feelings, and behavior related to others (for a review, see Mikulincer & Shaver, 2016), relationship quality (e.g., Collins & Feeney, 2000), emotion regulation and coping strategies (Mikulincer & Florian, 2004), and mental health (e.g., Shaver, Schachner, & Mikulincer, 2005).

Despite the paucity of research on links between attachment style and the immune system, there are reasons to expect that attachment style may have important links to systemic inflammation, and there have been calls over the last decade to consider the ways in which adult attachment may be linked to markers of inflammation (Ehrlich, 2019; Pietromonaco & Beck, 2019; Pietromonaco, Uchino, & Dunkel Schetter, 2013). For instance, as Simpson and Rholes (2010) have noted, the patterns of emotion regulation and coping strategies that attachment styles reflect might shape physiology in ways that could increase risk for poor physical health. A robust literature documents the links between stressful experiences and inflammation (e.g., Miller, Chen, & Parker, 2011; Slavich & Irwin, 2014). We have argued previously (Ehrlich, Miller, Jones, & Cassidy, 2016) that securely attached individuals may be buffered from heightened inflammation because of their capacity to engage in more effective coping strategies. In contrast, insecure individuals report more stress and conflict in their close relationships (Campbell, Simpson, Boldry, & Kashy, 2005), and they handle these situations less effectively than secure individuals (Simpson, Rholes, & Phillips, 1996; see Pietromonaco, Greenwood, & Barrett, 2004, for a review). Further, insecure individuals are less skilled at seeking and using social support to cope with stress (Mikulincer & Florian, 1998). Collectively, these behaviors could translate into greater exposure to chronic stress and risk for heightened inflammation.

To date, only a handful of studies have examined associations between self-reported attachment style and inflammation. In one study, couples participated in a laboratory discussion in which they were instructed to resolve conflict in their relationship (Gouin et al., 2009). Attachment avoidance, but not anxiety, was positively associated with production of IL-6 following the conflict discussion. Additional evidence linking adult attachment style to inflammatory responses comes from a sample of patients undergoing coronary artery bypass graft surgery (Kidd et al., 2014). Patients reported on their attachment style prior to
surgery, and researchers measured markers of circulating inflammation pre- and post-
surgery. Although attachment style was unrelated to post-surgery levels of two markers of
inflammation (CRP and TNF-α), attachment anxiety (but not avoidance) was related to
higher levels of IL-6 after surgery when controlling for pre-surgery levels. When considering
the implications of this study’s findings, it is important to note that it is circulating
inflammation that is examined; although inflammation in the relevant tissue following an
invasive surgery could be viewed as adaptive for recovery, circulating inflammation may not
confer the same healing benefits, as Kiecolt-Glaser et al. (2005) have shown. For additional
research examining links between inflammation in adulthood and attachment-related
constructs, such as perceptions of the parent as a secure base and maternal nurtance, see

In the present study, we seek to build on this small body of research by examining links
between attachment style and two markers of inflammation, IL-6 and CRP. To test our
hypotheses that both attachment avoidance and anxiety would be associated with
inflammation, we utilized data collected as part of the Maryland Adolescent Development in
Context Study (MADICS; principal investigators: Jacquelynne S. Eccles and Arnold J.
Sameroff). This preliminary study focused on African Americans—and important
population to study given that, relative to Whites, African Americans have higher rates of
morbidity and mortality due to chronic diseases of aging that have an underlying
inflammatory component, including coronary heart disease, stroke, and diabetes (Centers for
Disease Control and Prevention, 2013; Chung et al., 2009). Both IL-6 and CRP reflect
systemic inflammation, and high CRP levels increase risk for hypertension (Sesso et al.,
2003). Although IL-6 is less strongly predictive of hypertension risk (Sesso, Wang, Buring,
Ridker, & Gaziano, 2007), it stimulates production of CRP by the liver and is therefore an
important cytokine of interest. As such, we included both markers as outcomes in the present
study.

We included depressive symptoms as a covariate in our models to rule out possible
alternative explanations for any associations between attachment style and inflammation.
Given meta-analytic support for associations between depression and CRP and IL-6 in both
community and patient populations (Horn et al., 2018; Howren, Lamkin, & Suls, 2009), we
expected that depressive symptoms would be positively associated with CRP and IL-6 in the
present study.

**Method**

MADICS is a longitudinal study of normative development among 1,482 adolescents
residing in Prince George’s County, Maryland that began in 1991 when adolescents were in
7th grade (approximately 12 years old). Approximately 20 years after the Time 1 visit, at
age 32 (Time 8), 59 African American 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 $50), participants were instructed to refrain from engaging in
strenuous exercise, drinking alcohol, smoking, or taking non-prescription medication during
the two hours before the appointment, and from drinking caffeine, brushing their teeth, or
eating a meal at least one hour 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-minute 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 and attachment style. The procedures for the present study were approved by the (omitted for blind review) 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 described below ($M_{age} = 31.9$ years; $SD = .45$; 63% female). Some MADICS participants were ineligible due to one of the following: (a) major chronic illness or active infection; (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; 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 Time 8 was between $65,000 and $69,000.

Measures

**Adult attachment style.**—Attachment avoidance and anxiety were measured using the 12-item short form of Brennan, Clark, & Shaver’s Experiences in Close Relationships Scale (ECR-S; Brennan et al., 1998; Wei, Russell, Mallinckrodt, & Vogel, 2007). The avoidance subscale (6 items; $\alpha = .81$) measures the extent to which a person is uncomfortable with closeness, intimacy, and emotional disclosure in close relationships (e.g., “I try to avoid getting too close to my partner”). The anxiety subscale (6 items; $\alpha = .76$) measures the extent to which a person worries about being rejected, abandoned, or unloved (e.g., “I find that my partner[s] don’t want to get as close as I would like”). For each item, participants rated the extent to which they agree with the statement using a 7-point Likert-type scale, with responses ranging from 1 (disagree strongly) to 7 (agree strongly). Three items on the avoidance subscale were reverse-scored, and one item on the anxiety scale was reverse-scored. After four items were reverse-scored, items were averaged for each scale, such that higher scores reflect greater attachment anxiety and avoidance in close relationships. The ECR-S has demonstrated excellent psychometric properties including internal consistency, test-retest reliability, and construct validity (Wei et al., 2007).

**Depressive symptoms.**—Participants completed the Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996), a 21-item measure that reflects average depressive symptoms (e.g., sadness, loss of interest in activities) over the past two weeks ($\alpha = .90$). Participants rated each item on a 0 to 3 scale, with higher scores reflecting greater symptomatology. Based on the cutoff criteria provided by Beck, Steer, and Carbin (1988), 57.6% of our sample had minimal depression, 27.1% had mild depression, 11.9% had moderate depression, and 3.4% were severely depressed.
Inflammatory markers.—We used serum IL-6 and high sensitivity CRP as measures of systemic inflammation at age 32 (Time 8). Blood samples from the home visit venipuncture were transported to SeraCare in Gaithersburg, MD within two hours of the blood draw. Serum was then harvested by centrifugation and frozen. After all samples were obtained, CRP and IL-6 were measured using high-sensitivity chemiluminescence. Unfortunately, due to budget constraints, assays were run in singlet.

Inflammation-related confounds.—The health questionnaire at age 32 assessed a number of health-related confounds, including the following which are used in the present study: sex, 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. The majority of the sample reported being a non-smoker (78.9%). Participants also reported minimal alcohol use: 76.8% of the sample reported consuming two or fewer drinks per week. Body Mass Index (BMI) was a composite of measurements of height and weight. BMI varied substantially in the sample: 21.1% of the sample was considered normal weight (18.5 to 25), 33% were considered overweight (25.0 to 29.9), and 43.9% were considered obese (BMI of 30 or above).

Results

Missing Data and Outliers

Following common practice, participants with CRP values greater than 10 mg/L (n = 4) were excluded from analyses because values this high are an indication of acute inflammation due to infection or injury (Pearson et al., 2003). IL-6 values that were greater than three standard deviations from the mean were also excluded from the analysis (n = 2).

Descriptive Statistics

Means, standard deviations, and correlations of study variables are presented in Table 1. We first examined correlations among health-related confounds, CRP, and IL-6. CRP and IL-6 were not correlated with cigarette use, alcohol use, oral contraceptives, exercise, caffeine, or NSAID pain reliever use; as such, these covariates were not included in subsequent models.

Principal Analyses

Given the considerable overlap between attachment avoidance and anxiety (r = .40, p < .01), we conducted separate regression models to examine the associations among attachment avoidance, attachment anxiety, and inflammation (see Stanton et al., 2017 for a similar approach).

Predicting IL-6.—Table 2 presents the regression models for IL-6. After controlling for participant gender, BMI, and depressive symptoms, both attachment avoidance and attachment anxiety were positively associated with IL-6. The links between attachment styles and IL-6 remained significant when depressive symptoms were removed from the models.
Predicting CRP.—Table 3 presents the regression models for CRP. Participants’ depressive symptoms were positively associated with CRP. Neither attachment avoidance nor attachment anxiety was associated with CRP, however.

Discussion

The present study provides the first examination of attachment style and inflammation specifically in African Americans, a population often underrepresented in attachment research, yet overrepresented in prevalence of inflammation-related health problems (Yusuf, Reddy, Ôunpuu, & Anand, 2001). Both attachment avoidance and anxiety were concurrently associated with higher IL-6, but not CRP. Associations between adult attachment style and IL-6 were observed over and above BMI (which was strongly associated with both IL-6 and CRP) and depressive symptoms, which were correlated with attachment avoidance and marginally associated with attachment anxiety. As in previous studies, BMI was a significant predictor of inflammation (e.g., Yudkin et al., 2000), whereas depressive symptoms emerged as a significant predictor of CRP but not IL-6.

These findings are largely consistent with the two previous studies to examine direct links between attachment style and inflammation. Kidd et al. (2014) found that attachment anxiety (but not avoidance) predicted IL-6—and not CRP—among heart surgery patients. Gouin and colleagues (2009) found that attachment avoidance (but not anxiety) predicted married couples’ IL-6 levels following a conflict task (CRP was not measured). Whereas these previous studies examined attachment and inflammation in majority White samples experiencing acute stressors (heart surgery, conflict), the present study extends findings to a community sample of African Americans. The research to date suggests that adult attachment style predicts IL-6 levels, although the unique contributions of avoidance and anxiety may vary depending on the context or characteristics of the sample.

Why did attachment style predict IL-6 but not CRP, especially given that these measures were correlated? It is possible that a single assessment of CRP does not provide a reliable assessment of inflammation, so studies with repeated measurements of CRP will be important. It is notable that a similar pattern (i.e., attachment style predicting IL-6 but not CRP) was also observed in the Kidd et al. (2014) study of heart surgery patients. Thus, the evidence to date suggests that there may be specificity in the direct link between attachment and inflammatory markers. Other evidence suggests that central adiposity may alter the strength of proposed associations between stressors and inflammation (Chiang, Bower, Irwin, Taylor, & Fuligni, 2017). Chiang and colleagues (2017) found that central adiposity and BMI moderated the link between several stressors and inflammatory reactivity in a sample of adolescents, wherein the hypothesized links were significant for individuals with high waist circumference but not those with smaller waists. They further proposed that stress may stimulate adipose tissue to release cytokines—a hypothesis that warrants further examination in future studies. Finally, it is possible that attachment style predicts CRP only later in development (e.g., in middle age), with insecurity and related physiological and behavioral responses to stress contributing cumulatively to CRP over time. Three main study limitations merit consideration and point to avenues for further research. First, although the present small sample was intentionally focused on African Americans, future studies should...
seek to replicate findings with larger and more representative samples. It is possible that our observed effect sizes are inflated due to the small sample size (Ioannidis, 2005, 2008). With a larger and more representative sample, researchers would then be able to examine race as a moderator so that direct comparisons can be made between African Americans and non-African Americans within same study. Although we have no specific predictions about why these effects would differ as a function of race, it remains an open question. In addition, researchers could expand the scope of measures of the immune system by including assessments of adaptive immunity (see Picardi et al., 2007).

Second, in addition to race, future investigations should examine other moderators, such as marital status, socioeconomic status (SES), sex, and age. For example, it is possible that influence of attachment on inflammation becomes greater in old age, with increasing inflammation-related health risks (e.g., Hamer, Berman, Albers, Brown, & Silver, 1999) as well as increasing dependence on close others; that is, having a reliable secure base may be especially important for managing everyday stressors among the elderly (see Fagundes et al., 2011). With regard to SES, secure attachment style may buffer against effects of poverty on inflammation, in line with the social buffering of stress hypothesis (e.g., Cohen & Wills, 1985; Hostinar, 2015), whereas insecure attachment style may exacerbate the negative health effects of stressors associated with poverty.

Third, the study’s design was correlational and analyses examined concurrent associations, so we are unable to draw conclusions about longitudinal associations or causality. Future research should track both attachment and inflammation over time to examine whether attachment style in early adulthood predicts inflammation later in adulthood, whether attachment style predicts trajectories of inflammation over time, and whether changes in attachment style precede and predict corresponding changes in inflammation. One promising avenue for testing causal connections is to examine whether interventions that promote adult attachment security (see Slade, 2016) also demonstrate anti-inflammatory effects.

In addition to these questions, future research should investigate what mechanisms link attachment style and inflammation, testing mediators at the biological, psychological, and behavioral levels. For example, research has shown that insecure adults show greater physiological reactivity to stress (e.g., Feeney & Kirkpatrick, 1996), employ more negative cognitive strategies for coping with stressors (e.g., Mikulincer & Florian, 1995), have lower quality relationships (Nofile & Shaver, 2006), and engage in unhealthier behaviors such as smoking and drinking (Huntsinger & Luecken, 2004). These factors, in turn, have been linked to inflammation (O’Connor et al., 2009). Investigating mechanisms may also uncover potentially unique pathways linking attachment avoidance vs. anxiety to inflammation and disease. For example, some research suggests that attachment anxiety, but not avoidance, is related to self-reported sleep difficulties (Carmichael & Reis, 2005), which in turn have been linked to increased inflammation (e.g., Friedman, 2011; Irwin, Wang, Campomayor, Collado-Hidalgo, & Cole, 2006).

Finally, future research should examine attachment style in combination with other factors in the social environment relevant to stress and health, such as social network size, perceived discrimination, neighborhood violence, marital satisfaction, and parenting stress, to better
understand the cumulative and interactive contributions of social relationships at multiple levels of analysis.

In sum, evidence points to a link between insecure attachment style—including dimensions of anxiety about rejection/abandonment and avoidance of emotional intimacy—and the inflammatory marker IL-6 among African American young adults. Although further research is needed to illuminate the precise developmental pathways and mechanisms of this association, the results contribute to a growing literature demonstrating the importance of social relationships in understanding the origins of physical health and disease (e.g., Holt-Lunstad et al., 2017). Together, this exciting body of work suggests that investing in programs and policies to nurture secure, supportive human relationships may be a key component in public health efforts to promote both mental and physical health.

Acknowledgments

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References


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## Table 1

Descriptive Statistics and Correlations among Principal Variables in the Present Study

<table>
<thead>
<tr>
<th>Variable</th>
<th>M</th>
<th>SD</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Gender (% male)</td>
<td>37.3%</td>
<td>.49</td>
<td>-14</td>
<td>.11</td>
<td>.18</td>
<td>.16</td>
<td>.11</td>
<td>.07</td>
<td></td>
</tr>
<tr>
<td>2. Body Mass Index</td>
<td>29.4</td>
<td>6.13</td>
<td></td>
<td>-.07</td>
<td>.00</td>
<td>-.18</td>
<td>.38</td>
<td>.57</td>
<td>.57</td>
</tr>
<tr>
<td>3. Depressive Symptoms</td>
<td>.47</td>
<td>.41</td>
<td></td>
<td></td>
<td>-.37</td>
<td>-.22</td>
<td>.08</td>
<td>.34</td>
<td></td>
</tr>
<tr>
<td>4. Attachment Avoidance</td>
<td>2.43</td>
<td>1.09</td>
<td></td>
<td></td>
<td>-.40</td>
<td>.27</td>
<td>-.06</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Attachment Anxiety</td>
<td>3.36</td>
<td>1.21</td>
<td></td>
<td></td>
<td>.19</td>
<td>.03</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. IL-6</td>
<td>2.31</td>
<td>1.92</td>
<td></td>
<td></td>
<td>.20</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. C-Reactive Protein</td>
<td>1.88</td>
<td>2.30</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note.

* $p < .10$.

* $p < .05$.

** $p < .01$.

*** $p < .001$.  

$^? = p < .10$. 

$p < .05$. 

$^* = p < .01$. 

$^** = p < .001$. 

$^*** = p < .0001$. 

$^* = p < .05$. 

$^** = p < .01$. 

$^*** = p < .001$. 

$^**** = p < .0001$.
**Table 2**
Attachment Avoidance and Depressive Symptoms as Predictors of Inflammation

<table>
<thead>
<tr>
<th>Predictors</th>
<th>IL-6 β</th>
<th>p</th>
<th>Lower</th>
<th>Upper</th>
<th>CRP β</th>
<th>p</th>
<th>Lower</th>
<th>Upper</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td>.008</td>
<td>.95</td>
<td>-.96</td>
<td>1.02</td>
<td>.044</td>
<td>.689</td>
<td>-.81</td>
<td>1.22</td>
</tr>
<tr>
<td>BMI</td>
<td>.382</td>
<td>.003</td>
<td>.04</td>
<td>.20</td>
<td>.539</td>
<td>&lt;.001</td>
<td>.13</td>
<td>.31</td>
</tr>
<tr>
<td>Depressive symptoms</td>
<td>−.052</td>
<td>.698</td>
<td>−1.49</td>
<td>1.00</td>
<td>.351</td>
<td>.003</td>
<td>.69</td>
<td>3.25</td>
</tr>
<tr>
<td>ECR Avoidance</td>
<td>.286</td>
<td>.038</td>
<td>.03</td>
<td>.97</td>
<td>−.168</td>
<td>.150</td>
<td>−.85</td>
<td>.13</td>
</tr>
</tbody>
</table>

Note. ECR = Experiences in Close Relationships scale. Gender coded as 0 = female, 1 = male.
Table 3
Attachment Anxiety and Depressive Symptoms as Predictors of Inflammation

<table>
<thead>
<tr>
<th>Predictors</th>
<th>β</th>
<th>p</th>
<th>Lower</th>
<th>Upper</th>
<th>β</th>
<th>p</th>
<th>Lower</th>
<th>Upper</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td>.008</td>
<td>.952</td>
<td>−.97</td>
<td>1.03</td>
<td>.003</td>
<td>.977</td>
<td>−1.02</td>
<td>1.05</td>
</tr>
<tr>
<td>BMI</td>
<td>.425</td>
<td>.002</td>
<td>.05</td>
<td>.21</td>
<td>.566</td>
<td>&lt;.001</td>
<td>.14</td>
<td>.33</td>
</tr>
<tr>
<td>Depressive symptoms</td>
<td>−.007</td>
<td>.954</td>
<td>−1.23</td>
<td>1.16</td>
<td>.28</td>
<td>.015</td>
<td>.33</td>
<td>2.84</td>
</tr>
<tr>
<td>ECR Avoidance</td>
<td>.266</td>
<td>.047</td>
<td>.01</td>
<td>.84</td>
<td>.084</td>
<td>.468</td>
<td>−.28</td>
<td>.59</td>
</tr>
</tbody>
</table>

Note. ECR = Experiences in Close Relationships scale. Gender coded as 0 = female, 1 = male.