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Attachment Anxiety is Linked to Alterations in Cortisol Production and Cellular Immunity

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Abstract

Although evidence suggests that attachment anxiety may increase risk for health problems, the mechanisms are not well understood. Married couples ($N = 85$, $M_{age} = 38.67$) provided saliva samples over three days and blood samples on two occasions. Participants with higher attachment anxiety produced more cortisol and had fewer numbers of $CD3^+$ T-cells, $CD45^+$ T-cells, $CD3^+CD4^+$ helper T-cells, and $CD3^+CD8^+$ cytotoxic T-cells than those with lower attachment anxiety. Higher cortisol was also related to fewer numbers of $CD3^+$, $CD45^+$, $CD3^+CD4^+$, and $CD3^+CD8^+$, which is mechanistically consistent with research showing that cortisol alters the cellular immune response. These data suggest that attachment anxiety may have physiological costs and provide a glimpse into the pathways through which social relationships impact health. The current study also extends attachment theory in an important new direction by utilizing a psychoneuroimmunological approach to the study of attachment anxiety, stress, and health.

Keywords

Attachment theory; attachment anxiety; marriage; chronic stress; cortisol; psychoneuroimmunology; psychoneuroendocrinology

Close and caring relationships are essential to mental and physical well-being (Robles & Kiecolt-Glaser, 2003; Uchino, 2009). Attachment theory provides a framework for understanding the links among interpersonal relationships, stress, and health (Bowlby, 1973; Maunder & Hunter, 2001; Pietromonaco, Uchino, & Dunkel Schetter, in press). According to attachment theory, people differ in the extent to which they believe close others will be supportive and available during times of need. These individual differences are rooted in early life experiences with care providers and can be conceptualized along two dimensions, avoidance and anxiety (Mikulincer, Shaver, & Pereg, 2003). People who are high in attachment avoidance are excessively self-reliant and uncomfortable with closeness and

intimacy. Those who are high in attachment anxiety intensely fear rejection, are dependent on others, and worry about close relationships.

Convergent evidence suggests that people high in attachment anxiety may have an enhanced risk for health problems. For example, among a national probability sample of adults, more anxiously attached people had a greater incidence of strokes and heart attacks and were more likely to be diagnosed with high blood pressure and ulcers than those less anxiously attached (McWilliams & Bailey, 2010). These health differences remained after controlling for important sociodemographic and psychological risk factors. In other research, people with higher attachment anxiety reported worse overall physical health, more stress-related physical symptoms, and a greater frequency of illness and sick days than those with lower attachment anxiety (Ciechanowski, Walker, Katon, & Russo, 2002; Hazan & Shaver, 1990; Maunder, Hunter, & Lancee, 2011; Oliveira & Costa, 2009). Higher attachment anxiety among adolescents was marginally related to abdominal fat increases over a three year period. More anxiously attached adolescents also had greater arterial stiffness than those less anxiously attached (Midei & Matthews, 2009). The links between attachment avoidance and health problems are less consistent, suggesting little to no association, and are thus not the primary focus of the current study (Hazan & Shaver, 1990; Maunder et al., 2011; McWilliams & Bailey, 2010).

The pathways through which attachment anxiety influences health are not well understood. One possibility is that attachment anxiety functions as a chronic social stressor. Fear of rejection and worries about relationships are hallmarks of attachment anxiety (Collins, Guichard, Ford, & Feeney, 2004). Accordingly, attachment anxiety is characterized by constant vigilance and hypersensitivity to cues of rejection (Hazan & Shaver, 1994). This vigilance and hypersensitivity leads those high in attachment anxiety to easily perceive threats in their environment and experience most social interactions as stressful and worrisome (Collins et al., 2004; Mikulincer et al., 2003; Shaver & Mikulincer, 2002).

In addition to exhibiting an intense fear of rejection and vigilance towards social threat, people who are highly anxious often display a “hyperactivating” emotional style, which involves excessive attention to and rumination about psychologically distressing experiences (Burnette, Davis, Green, Worthington, & Bradfield, 2009; Shaver & Mikulincer, 2002). In contrast, highly avoidant individuals actively avoid socially threatening information in an attempt to create psychological distance between themselves and others and display a “deactivating” emotional style, characterized by thought suppression and inattention to negative information.

Taken together, prior theoretical and empirical work suggests that highly anxious individuals readily perceive social threats in their environment, strongly react to stressful experiences, and dwell on negative aspects of an experience after it is over. These cognitive and emotional patterns are not evident among people high in attachment avoidance.

The social stress experienced by people with high attachment anxiety may have physiological costs. For example, more anxiously attached people had higher levels of resting cortisol (a key stress hormone) than those less anxiously attached (Gordon et al., 2008). Similarly, individuals higher in attachment anxiety produced more cortisol during a 4-7 day period of separation from their romantic partner than those lower in attachment anxiety (Diamond, Hicks, & Otter-Henderson, 2008). Neither study found relationships between attachment avoidance and daily cortisol levels.

Attachment anxiety-related hypersensitivity and corresponding alterations in cortisol production may influence immune function; both chronic stress and cortisol can modify cellular immunity (e.g., T-cells; Ashwell, Lu, & Vacchio, 2000; Sommershof et al., 2009).

For example, compared to married women, those who were recently divorced had fewer $CD3^+CD4^+$ helper T-cells, which activate immune cells and respond to cells infected with a pathogen. In addition, more recent divorces were related to fewer $CD3^+CD8^+$ cytotoxic T-cells, which suppress immune reactive T-cells and kill pathogen infected cells (Kiecolt-Glaser et al., 1987).

The Current Study

Based on prior research relating anxious attachment to hypervigilance and sensitivity to rejection, we assessed the links between attachment anxiety, cortisol production, and multiple cellular markers. We selected $CD3^+$ T-cells, $CD45^+$ T-cells, $CD3^+CD4^+$ helper T-cells, and $CD3^+CD8^+$ cytotoxic T-cells because previous studies have shown that they are modulated by chronic stress and high cortisol (Ashwell et al., 2000; Kiecolt-Glaser et al., 1987; Sommershof et al., 2009). In addition, stress-related immune dysregulation is strongly linked to health. For example, the immune system's ability to mount an effective response to pathogens largely depends on T-cells; they are integral to numerous parts of the immune response (e.g., B cell activation) and fewer numbers of T-cells essentially limit the number of pathogens that the immune system can defend against. Immunosenesence, the aging of the immune system that is linked to many age-related diseases, is marked by limited new T-cell production, and large decrements in $CD3^+CD4^+$ helper T-cells can lead to immunodeficiency (Dorshkind, Montecino-Rodriguez, & Signer, 2009; Lee et al., 1991). Furthermore, CD3+CD8+ cytotoxic T-cells are lower in obese than non-obese people, and CD3+CD8+ among the obese is linked to an impaired vaccine response (O'Rourke et al., 2005; Sheridan et al., 2011).

We hypothesized that, compared to people with lower attachment anxiety, those with higher attachment anxiety would exhibit (1) higher levels of cortisol and (2) dysregulated cellular immunity as indexed by fewer numbers of $CD3^+$ T-cells, $CD45^+$ T-cells, $CD3^+CD4^+$ helper T-cells, and $CD3^+CD8^+$ cytotoxic T-cells. Because attachment avoidance is not consistently related to stress-related health outcomes and is not characterized by vigilance towards social threat or a hyperactivating emotional style, we predicted that attachment avoidance would be unrelated to daily cortisol and cellular immunity (although see the discussion section for an examination of attachment avoidance and acute stress reactivity).

People in longer-term romantic relationships are likely to experience attachment concerns (e.g., fear of rejection) on a regular basis. Therefore, our sample of married couples provided an opportune way to investigate attachment-related physiological dysregulation.

Methods

Participants

Participants were 85 couples married an average of 12.26 years (SD = 11.37, range 2-52). Their average age was 38.67 years (SD = 12.84, range 22-77) and the majority of participants were White (N = 155). Couples were recruited as part of a larger study on marital distress and wound healing through flyers, newspaper and radio ads, and participant referrals (Kiecolt-Glaser et al., 2005).

Couples were ineligible if they were married less than 2 years. We excluded individuals who were pregnant, drank excessive amounts of alcohol or caffeine, smoked, used blood pressure medication, had health problems that had endocrinological or immunological consequences (e.g., cancer, diabetes), or were taking related medications. Due to the nature of the larger study, individuals were excluded if they had a medical condition that influenced wound

healing processes. The project was approved by The Ohio State University Institutional Review Board; participants provided written informed consent before participating.

Procedure

Day 1 - CRC visit—Couples were admitted to the Clinical Research Center (CRC), a hospital research facility, at 7:00 a.m. for a 24-hour visit. Visits were scheduled during the follicular phase of the female's menstrual cycle. Couples fasted for 12 hours prior to the start of their visit and were fed standardized meals throughout their stay. Both partners were in the same room and completed the same tasks, ensuring similar activity levels across individuals.

Participants provided their first saliva and blood samples shortly after arrival and their second saliva sample at 7:45 a.m. During the remainder of the visit, couples underwent a wound induction procedure and engaged in a marital problem solving task which are discussed elsewhere and are not the focus of the current study (Gouin et al., 2009; Kiecolt-Glaser et al., 2005).

Days 2 and 3 – After the visit—On day 2, couples were awakened in the CRC at 7:00 a.m. for blood and saliva samples. Participants provided a second saliva sample 30 minutes later, ate a standard breakfast, and were discharged from the CRC. Two additional saliva samples were collected by participants at 12:00 p.m. and 8:30 p.m. At home on day 3, participants provided saliva samples at awakening, 30 minutes later, 12:00 p.m., and 8:30 p.m., and then returned them to the lab the following day. Participants refrained from brushing their teeth within 30 minutes before sample collection.

Materials

Questionnaires—The Experiences in Close Relationships scale, a widely used measure of adult attachment (ECR; Brennan, Clark, & Shaver, 1998), contains separate attachment anxiety and avoidance subscales ($\alpha = .88$, $\alpha = .91$ respectively). Participants responded using a 1 to 7 scale and the final composite reflected an average of the items. The current sample range was 1.00 to 5.39 ($M = 2.45$, $SD = .91$) for attachment anxiety and 1.00 to 5.94 ($M = 2.15$, $SD = .95$) for avoidance.

The Beck Anxiety Inventory assesses general anxiety symptoms (BAI; Beck, Epstein, Brown, & Steer, 1988). The BAI can discriminate between clinically anxious and non-clinically anxious people and has good test-retest reliability and internal consistency. The BAI provided a way to disentangle general anxiety from attachment anxiety.

The Pittsburgh Sleep Quality Index evaluates overall sleep quality and has good psychometric properties (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). Sleep influences cortisol production and immune function and more anxiously attached people report worse sleep quality than those less anxiously attached (Faraut, Boudjeltia, Vanhamme, & Kerkhofs, 2012; Maunder et al., 2011). Accordingly, we included the PSQI to account for covariation between sleep and attachment anxiety, cortisol, and immune function.

Cortisol—Saliva was collected using a salivette (Sarstedt, Newton, North Carolina), an untreated sterile cotton roll that was placed in the subject's mouth for ~2 min. Each subject's samples were frozen after collection and analyzed within the same assay using the Cortisol Coat-A-Count RIA (Siemens Medical Solutions Diagnostics, Los Angeles, CA).

Cellular immune markers—We assayed four T-cell markers, $CD3^+$, $CD45^+$, $CD3^+CD4^+$, and $CD3^+CD8^+$. The use of the $CD3^+$ and $CD45^+$ monoclonal antibodies provided data about the total number of T lymphocytes. The $CD3^+CD4^+$ and $CD3^+CD8^+$ monoclonal antibodies provided information about the number of helper T-cells and cytotoxic T-cells respectively.

To measure total T-cells, 100ul of heparin treated whole blood were incubated at room temperature for 15 minutes in the dark with CD3 Cychrome, CD4 Fitc, CD8 PE, and CD45 APC antibody (BD BioSciences PharMingen, San Diego, CA). Cells were also stained for the appropriate isotype control. Complete blood counts allowed us to determine the absolute number of cells.

Data Analysis

The distributions of the cortisol and T-cell data were checked for normality and the presence of outliers. Following established procedures, four participants whose cortisol values were more than 4 standard deviations from the mean were dropped from the cortisol analyses (Diamond et al., 2008). The scores for attachment anxiety, avoidance, trait anxiety, cortisol, and the T-cell measures were highly skewed. Accordingly, each measure was $\log_{10}$ transformed prior to analyses.

The .26 intra-class correlation between wives' and husbands' attachment anxiety scores indicated dependency within dyads. Accordingly, the data were analyzed with mixed models as previously described and were structured with time as a repeated measure and person nested within couple (Kenny, Kashy, & Cook, 2006).

Every analysis included the following control variables: body mass index (BMI: kg/m^2), age, gender, attachment avoidance, and general anxiety symptoms. We did not include time since waking because we tightly controlled participants' wake-up time on two of the three days. Attachment avoidance was included to account for covariation between attachment avoidance and anxiety (Griffin & Bartholomew, 1994), which provided a way to restrict our conclusions to the unique contribution of attachment anxiety, independent of attachment avoidance. Inclusion of general anxiety symptoms allowed us to focus our results on people who were particularly anxious about their close relationships. On average, women were more anxiously attached than men, $p = .020$. Accordingly, we initially included the gender by attachment anxiety interaction in each model. Unless otherwise noted, these interactions were non-significant and the interaction term was omitted from all analyses. Supplemental analyses investigated whether the relationships among attachment anxiety, cortisol, and cellular immunity remained after accounting for sleep quality and other health behaviors.

Results

The statistics for attachment anxiety and avoidance predicting each outcome are reported in Table 1 and Table 2 respectively. All reported coefficients are unstandardized.

Attachment Anxiety and Cortisol

First, we examined the hypothesis that higher attachment anxiety is linked to higher cortisol levels (Kenny et al., 2006). There were different assessment times and numbers of samples on the day of the visit compared to the following two days. Accordingly, we analyzed day 1 separately from days 2 and 3.

To analyze the visit data, we used a mixed model that included the main effects of attachment anxiety and time of assessment (first vs. second) and the interaction between the two. More anxiously attached participants had marginally higher cortisol levels than those

less anxiously attached. The non-significant interaction between attachment anxiety and time indicated that the strength of the relationship between attachment anxiety and cortisol was the same at both visit assessments and the slope of cortisol from the first to the second assessment did not differ by attachment anxiety.

To analyze days 2 and 3, we first examined the area under the curve with respect to ground (AUC_G), which provided a summary index of total cortisol concentration throughout the day (Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003)[1]. Our mixed model included the main effects of attachment anxiety and day of assessment (two vs. three) and the interaction between the two. As expected, participants with higher attachment anxiety produced significantly more cortisol than those with lower attachment anxiety. The non-significant interaction between attachment anxiety and day indicated that the strength of the relationship between attachment anxiety and AUC_G did not differ by day of assessment.

To estimate the AUC_G cortisol difference between those lower and higher in attachment anxiety, we used the covariate adjusted means at one standard deviation above and below the mean of attachment anxiety. Participants with higher attachment anxiety (+1 SD) had 11% more cortisol than those with lower attachment anxiety (−1 SD).

We followed-up on these analyses by separately investigating the morning rise in cortisol and the decline following the morning rise. Examining these patterns individually allowed us to determine whether attachment anxiety-related differences in cortisol were specific to a particular time of day. Consistent with the AUC_G analyses, participants with higher attachment anxiety produced more cortisol during both the morning rise and the recovery from the morning rise than those with lower attachment anxiety. The interaction between attachment anxiety and time was non-significant for both time frames, indicating that the slope of cortisol over time did not differ by attachment anxiety.

Participants with higher attachment avoidance produced significantly less cortisol at the beginning of the visit than those with lower attachment avoidance. However, on days 2 and 3, attachment avoidance was unrelated to AUC_G, cortisol levels during the morning rise and the subsequent decline, and the slope of cortisol throughout the day.

Attachment Anxiety and Cellular Immunity

To analyze the hypothesis that higher attachment anxiety is related to fewer T-cells, we used a separate mixed model for each immune marker that included the main effects of attachment anxiety and day of assessment and the interaction between the two. More anxiously attached participants had significantly fewer numbers of $CD3^+$ T-cells, $CD45^+$ T-cells, $CD3^+CD4^+$ helper T-cells, and $CD3^+CD8^+$ cytotoxic T-cells than those less anxiously attached. The non-significant anxiety by day interaction terms in each model indicated that the strength of the relationships between attachment anxiety and the immune parameters did not differ by day of assessment. Furthermore, participants with higher attachment anxiety (+1 SD) had 19%, 15%, 11%, and 22% fewer $CD3^+$ T-cells, $CD45^+$ T-cells, $CD3^+CD4^+$ helper T-cells, and $CD3^+CD8^+$ cytotoxic T-cells respectively than those with lower attachment anxiety (−1 SD).

The relationships between attachment anxiety and two of the immune markers ($CD45^+$ and $CD3^+CD4^+$) were moderated by gender, $F(1, 203) = 4.38$, $p = .038$ and $F(1,209) = 5.42$, $p = .021$ respectively. Simple slopes analyses revealed that women with higher attachment

[1]We had information about participants' wake-up time on day 2 but not 3. To compute AUC_G on day 3, we used participants' typical wake-up time as an estimate of their actual wake-up time. We had the actual wake-up time on one of the two days so we were able to statistically examine whether the results differed by day of assessment.

anxiety had lower $CD45^+$ and $CD3^+CD4^+$ than women with lower attachment anxiety, $b = -.30$, $F(1,170) = 17.05$, $p \leq .001$ and $b = -.27$, $F(1,170) = 11.30$, $p = .001$. Attachment anxiety for men was unrelated to $CD45^+$ and $CD3^+CD4^+$, $b = -.08$, $F(1,162) = 0.90$, $p = .344$ and $b = -.01$, $F(1,164) = 0.02$, $p = .881$. Attachment avoidance was unrelated to the immune parameters.

Relationships between Cortisol and Cellular Immunity

Samples collected at the same time (7:00 a.m.) on two days provided a way to address the relationships between cortisol and cellular immunity. A series of mixed models with the main effects of cortisol and day of assessment and the interaction between the two were used to analyze each immune marker. Higher levels of cortisol were significantly related to fewer numbers of $CD3^+$ T-cells [$b = -.08$, $F(1,247) = 5.05$, $p = .026$], $CD45^+$ T-cells [$b = -.09$, $F(1,247) = 7.50$, $p = .007$], and $CD3^+CD4^+$ helper T-cells [$b = -.08$, $F(1,246) = 5.47$, $p = .020$], and marginally related to fewer $CD3^+CD8^+$ cytotoxic T-cells [$b = -.08$, $F(1,245) = 3.32$, $p = .070$]. The non-significant cortisol by day interaction terms indicated that the strength of the relationships between cortisol and the immune parameters did not differ by day of assessment.

Health Behaviors

We used a series of mixed models to examine whether the relationships among attachment anxiety, cortisol, and cellular immunity held after controlling for physical activity, sleep quality, and birth control, the most common type of medication in our sample (N = 18). Participants with higher attachment anxiety produced more cortisol and had fewer numbers of $CD3^+$ T-cells, $CD45^+$ T-cells, $CD3^+CD4^+$ helper T-cells, and $CD3^+CD8^+$ cytotoxic T-cells than those with lower attachment anxiety after accounting for these health behaviors.

Discussion

In accord with psychological data demonstrating that anxiety about relationships is a potent stressor, people with higher attachment anxiety produced more cortisol and had fewer numbers of $CD3^+$ T-cells, $CD45^+$ T-cells, $CD3^+CD4^+$ helper T-cells, and $CD3^+CD8^+$ cytotoxic T-cells than those with lower attachment anxiety, independent of their general anxiety levels. Attachment avoidance was inconsistently related to cortisol and was unrelated to the immune markers. The current results are consistent with theoretical speculation that anxiety about close relationships enhances risk for mental and physical health problems (Feeney, 2000; Maunder & Hunter, 2001).

Both theory and research suggest that attachment anxiety increases perceived stress and alters the stress response (Maunder & Hunter, 2001; Mikulincer et al., 2003). Our data support this perspective and demonstrate that attachment anxiety has physiological costs. In particular, the current study suggests that attachment anxiety may function as a chronic social stressor that is related to alterations in cortisol and cellular immunity. These results extend attachment theory in an important new direction by utilizing a psychoneuroimmunological (PNI) approach to the study of attachment anxiety, stress, and health.

In line with prior work, the current study demonstrated that attachment anxiety is linked to the overproduction of cortisol. The present data expand upon these findings by revealing that attachment anxiety-related increases in cortisol production were linked to cellular immune dysregulation. Specifically higher cortisol was related to fewer numbers of $CD3^+$ T-cells, $CD45^+$ T-cells, and $CD3^+CD4^+$ helper T-cells, and marginally related to fewer $CD3^+CD8^+$ cytotoxic T-cells. These results are mechanistically consistent with research

showing that cortisol alters immune function, particularly cellular immunity (Ashwell et al., 2000).

The current data suggest that attachment anxiety may influence health through alterations in cortisol production and cellular immunity; the T-cell response is critical to the effective resolution of viral and bacterial infections, among other functions. The magnitude of attachment anxiety-related T-cell differences, which ranged from 11% in $CD3^+CD4^+$ helper T-cells to a 22% in $CD3^+CD8^+$ cytotoxic T-cells in our sample, supports the health implications of the current results. For instance, in one study, obese (BMI≥35) people had 9% fewer CD3+CD8+ cytotoxic T-cells than those who were non-obese (BMI≤25), and the CD3+CD8+ cytotoxic T-cell response among obese people is linked to an impaired vaccine response (O'Rourke et al., 2005; Sheridan et al., 2011). Furthermore, reductions in naïve CD3+CD8+ cells, a subset of CD3+CD8+ T-cells, are a hallmark of the aging immune system (Dorshkind et al., 2009).

Other mechanisms may work independently or in tandem with changes in cortisol and cellular immunity to influence health (Maunder & Hunter, 2001). For example, compared to less anxiously attached people, those more anxiously attached reported poorer sleep quality, a strong predictor of negative health outcomes (Maunder et al., 2011; Strine & Chapman, 2005). The current data demonstrate that people with higher attachment anxiety produced more cortisol and fewer numbers of $CD3^+$ T-cells, $CD45^+$ T-cells, $CD3^+CD4^+$ helper T-cells, and $CD3^+CD8^+$ cytotoxic T-cells than those with lower attachment anxiety, regardless of their sleep quality. These data suggest that sleep quality does not explain attachment-related cortisol and immune alterations.

According to attachment theory, the social vigilance and hyperactivating emotional style exhibited by those high in attachment anxiety were likely forged during early life experiences with inconsistent care providers (Diamond & Fagundes, 2008). A number of theoretical perspectives further propose that early adverse experiences have the potential to re-calibrate the HPA axis and other physiological systems, effectively altering the way these systems function in adulthood (Repetti, Taylor, & Seeman, 2002). Thus, the cortisol and cellular immune dysregulation exhibited by anxiously attached individuals in the current study may be a result of early life changes that cascaded forward, current social stress and hypervigilance, or a combination of the two.

Previous research demonstrated that people with higher attachment anxiety were more concerned about their ability to handle pain than those with lower attachment anxiety (Meredith, Strong, & Feeney, 2006). As noted in the methods section, the latter portion of the day 1 visit was devoted to a wound induction procedure. Thus, the day 1 cortisol data could be interpreted in terms of acute stress reactivity. However, participants higher in attachment anxiety produced more cortisol than those lower in attachment anxiety during the two days following the visit. These results are consistent with the argument that attachment anxiety is a chronic social stressor that has implications for endocrine and immune function in everyday life.

A distinct but related body of work has addressed the relationships between attachment anxiety and avoidance and reactivity to an acute stressor. For example, more anxiously attached men and more avoidantly attached women had stronger cortisol responses to a relationship conflict discussion than less anxiously attached men and less avoidantly attached women, respectively (Powers, Pietromonaco, Gunlicks, & Sayer, 2006). Higher attachment avoidance was related to greater interleukin-6 (IL-6; a measure of inflammation) reactivity during a marital conflict discussion compared to a social support discussion (Gouin et al., 2009). Importantly, chronic and acute stress reactivity are differentially related

to physiological and psychological states (Segerstrom & Miller, 2004). Furthermore, people have some degree of control over the social situations they encounter in daily life, and avoidantly attached individuals actively stay away from socially threatening situations (Shaver & Mikulincer, 2002). On the other hand, acute laboratory stressors force people to confront the situation at hand. The current study suggests that attachment anxiety may serve as a chronic social stressor and prior work points to the importance of attachment anxiety and avoidance in understanding acute stress reactivity. Utilizing a PNI approach is critical to fully understanding the links among attachment anxiety, stress, and health.

In sum, higher attachment anxiety was linked to alterations in cortisol production and cellular immunity. These results are particularly notable in light of the sample under investigation; only healthy people were allowed to participate and the attachment anxiety scores were on the lower end of the spectrum. Thus, the attachment anxiety-related differences in T-cell counts evident in this sample likely underestimate the true effects. The current study demonstrates that attachment anxiety has physiological costs and provides a glimpse into the pathways through which social relationships can impact health and well-being.

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Table 1
Summary of mixed model results for attachment anxiety predicting each outcome

Outcome	Time of Assessment	Unstandardized Slope Coefficient	F	p
Cortisol	Day 1	.20	3.26	.072
	Days 2 and 3 AUC_G	3.77	8.46	.004
	Days 2 and 3 Morning Rise	.19	5.57	.019
	Days 2 and 3 Post-rise Decline	.22	10.05	.002
$CD3^+$ T-cells	Days 1 and 2	−.26	15.49	<.001
$CD45^+$ T-cells	Days 1 and 2	−.22	12.56	<.001
$CD3^+CD4^+$ helper T-cells	Days 1 and 2	−.17	6.32	.013
$CD3^+CD8^+$ cytotoxic T-cells	Days 1 and 2	−.27	10.26	.002

Note. Day 1 = day of the visit, Day 2 = first day after the visit, and Day 3 = second day after the visit. AUC_G = area under the curve with respect to ground. The unstandardized coefficients were derived from log transformed scores.

Table 2
Summary of mixed model results for attachment avoidance predicting each outcome

Outcome	Time of Assessment	Unstandardized Slope Coefficient	F	p
Cortisol	Day 1	−.21	5.44	.020
	Days 2 and 3 AUC_G	−.20	.03	.855
	Days 2 and 3 Morning Rise	−.01	.01	.939
	Days 2 and 3 Post-rise Decline	−.02	.08	.774
$CD3^+$ T-cells	Days 1 and 2	.08	2.36	.126
$CD45^+$ T-cells	Days 1 and 2	.05	.97	.325
$CD3^+CD4^+$ helper T-cells	Days 1 and 2	.08	1.98	.161
$CD3^+CD8^+$ cytotoxic T-cells	Days 1 and 2	.04	.41	.525

Note. Day 1 = day of the visit, Day 2 = first day after the visit, and Day 3 = second day after the visit. AUC_G = area under the curve with respect to ground. The unstandardized coefficients were derived from log transformed scores.