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

Lisa M. Jaremka¹, Ronald Glaser^{1,2,3,4}, Timothy J. Loving⁵,
William B. Malarkey^{1,2,4}, Jeffrey R. Stowell⁶, and
Janice K. Kiecolt-Glaser^{1,4,7}

¹Institute for Behavioral Medicine Research, The Ohio State University College of Medicine; ²Department of Internal Medicine, The Ohio State University College of Medicine; ³Department of Molecular Virology, Immunology and Medical Genetics, The Ohio State University College of Medicine; ⁴Comprehensive Cancer Center, The Ohio State University College of Medicine; ⁵Department of Human Development and Family Sciences, The University of Texas at Austin; ⁶Psychology Department, Eastern Illinois University; and ⁷Department of Psychiatry, The Ohio State University College of Medicine

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Abstract

Although evidence suggests that attachment anxiety may increase risk for health problems, the mechanisms underlying these effects are not well understood. In the current study, married couples ($N = 85$) provided saliva samples over 3 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 participants with lower attachment anxiety. Higher cortisol levels were also related to fewer numbers of CD3⁺, CD45⁺, CD3⁺CD4⁺, and CD3⁺CD8⁺ cells, which is consistent with research showing that cortisol alters the cellular immune response. These data suggest that attachment anxiety may have physiological costs, and they provide a glimpse into the pathways through which social relationships affect health. The current study also extends attachment theory in an important new direction by demonstrating the utility of a psychoneuroimmunological approach to the study of attachment anxiety, stress, and health.

Keywords

attachment theory, attachment anxiety, marriage, chronic stress, cortisol, psychoneuroimmunology, psychoneuroendocrinology, interpersonal relationships, individual differences, neuroendocrinology, health

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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 with high attachment avoidance are excessively self-reliant and uncomfortable with closeness and intimacy. Those with high attachment anxiety intensely fear rejection, are dependent on others, and worry about their close relationships.

Convergent evidence suggests that people with high attachment anxiety may have increased risks for health problems. For example, among a national probability sample of adults, people who were more anxiously attached had a greater incidence of strokes and heart attacks and were more likely to be diagnosed with high blood pressure and ulcers than people who were 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

Corresponding Author:

Lisa M. Jaremka, The Ohio State University College of Medicine, Institute for Behavioral Medicine Research, 460 Medical Center Dr., Columbus, OH 43210
E-mail: lisa.jaremka@osumc.edu

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 3-year period. Adolescents who were more anxiously attached also had greater arterial stiffness than those who were less anxiously attached (Midei & Matthews, 2009). The links between attachment avoidance and health problems are less consistent, with research suggesting little to no association between the two, 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), which in turn lead those with high attachment anxiety to easily perceive threats in their environment and frequently experience 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 toward social threat, people who are anxiously attached 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, avoidantly attached individuals actively eschew socially threatening information in an attempt to create psychological distance between themselves and others, and they display a “deactivating” emotional style characterized by thought suppression and inattention to negative information.

Taken together, prior theoretical and empirical work suggests that individuals with high attachment anxiety 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 with high attachment avoidance.

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

Hypersensitivity related to attachment anxiety and corresponding alterations in cortisol production may influence immune function; both chronic stress and cortisol can modify cellular immunity (Ashwell, Lu, & Vacchio, 2000; Sommershof et al., 2009). For example, compared with 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

On the basis of prior research relating anxious attachment to hypervigilance and sensitivity to rejection, we assessed the links between attachment anxiety, cortisol production, and multiple cellular immune 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 levels of 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. Immunosenescence, the aging of the immune system that is linked to many age-related diseases, is marked by limited production of new T cells, and large decrements in CD3⁺CD4⁺ helper T cells can lead to immunodeficiency (Dorshkind, Montecino-Rodriguez, & Signer, 2009; Lee et al., 1991). Furthermore, there are fewer CD3⁺CD8⁺ cytotoxic T cells in obese than in nonobese people, and the CD3⁺CD8⁺ cytotoxic T-cell response among the obese has been linked to an impaired vaccine response (O’Rourke et al., 2005; Sheridan et al., 2012).

We hypothesized that, compared with people who have lower attachment anxiety, those who have higher attachment anxiety would exhibit (a) higher levels of cortisol and (b) 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 associated with stress-related health outcomes and is not characterized by vigilance toward social threat or a hyperactivating emotional style, we predicted that attachment avoidance would be unrelated to daily cortisol levels and cellular immunity (although see the Discussion section for an examination of attachment avoidance and acute stress reactivity).

People in long-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.

Method

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. Because of the larger study's nature, individuals were also 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 beginning the study.

Procedure

Day 1: Clinical Research Center (CRC) visit. Couples were admitted to The Ohio State University CRC, a hospital research facility, at 7:00 a.m. for a 24-hr visit. Visits were scheduled during the follicular phase of the female partner's menstrual cycle. Couples fasted for 12 hr 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, which ensured 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 (Gouin et al., 2009; Kiecolt-Glaser et al., 2005) and are not the focus of the current study.

Days 2 and 3: after the CRC 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 min later, ate a standardized 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 min 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 for 30 min before sample collection.

Materials

Questionnaires. The Experiences in Close Relationships (ECR; Brennan, Clark, & Shaver, 1998) scale, a widely used measure of adult attachment, contains separate subscales measuring attachment anxiety and attachment avoidance ($\alpha = .88$ and $.91$, respectively). Participants responded to all ECR questions using a scale from 1 to 7, and the final composite for each subscale reflected an average of the items. The range of responses was 1.00 to 5.39 ($M = 2.45$, $SD = 0.91$) for attachment anxiety and 1.00 to 5.94 ($M = 2.15$, $SD = 0.95$) for attachment avoidance, with higher numbers reflecting more attachment anxiety and attachment avoidance, respectively.

The Beck Anxiety Inventory (BAI; Beck, Epstein, Brown, & Steer, 1988) assesses general anxiety symptoms. The BAI can discriminate between people who are clinically anxious and people who are not, and it 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 (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989) assesses overall sleep quality and has good psychometric properties. Sleep influences cortisol production and immune function, and people who are more anxiously attached report worse sleep quality than those who are 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, NC), an untreated sterile cotton roll that was placed in the subject's mouth for approximately 2 min. Each subject's samples were frozen after collection and analyzed within the same assay using the Cortisol Coat-A-Count radioimmunoassay (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, 100 μ l of heparin-treated whole blood were incubated at room temperature for 15 min in the dark with CD3 Cy-Chrome, CD4 Fitc, CD8 PE, and CD45 APC antibodies (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, we dropped 4 participants from the cortisol analyses because their cortisol values were more than 4 standard deviations from the sample mean (Diamond et al., 2008). The distributions of the attachment anxiety, attachment avoidance, general anxiety, cortisol, and T-cell data were highly skewed. Accordingly, each measure was log-10 transformed prior to analyses.

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

Every analysis included body mass index (BMI; measured in kg/m²), age, gender, attachment avoidance, and general anxiety symptoms as control variables. We did not include time since waking because we tightly controlled participants' wake-up time on 2 of the 3 study days. Attachment avoidance was included to account for covariation between attachment avoidance and attachment 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 interaction between gender and attachment anxiety in each model. Unless otherwise noted, these interactions were nonsignificant, and the interaction term was omitted from all analyses. In supplemental analyses, we investigated whether the relationships among attachment anxiety, cortisol, and cellular immunity remained after accounting for sleep quality and other health behaviors.

Results

Tables 1 and 2 provide results for attachment anxiety and attachment avoidance, respectively, as predictors of each outcome. 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 CRC visit than on the following 2 days. Accordingly, we analyzed the data from the CRC (Day 1) separately from Days 2 and 3.

To analyze the CRC 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. Participants who were more anxiously attached had marginally higher cortisol levels than those who were less anxiously attached. The nonsignificant interaction between attachment anxiety and time of assessment indicated that the strength of the relationship between attachment anxiety and cortisol was the same at both assessments and that the slope of cortisol from the first to the second assessment did not differ as a function of attachment anxiety.

To analyze the data from Days 2 and 3, we first examined the area under the curve (AUC) 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 (2 vs. 3) and the interaction between the two. As expected, participants with higher attachment anxiety produced significantly more cortisol than those with lower attachment anxiety. The nonsignificant interaction between attachment anxiety and day of assessment indicated that the strength of the relationship between attachment anxiety and AUC_g did not differ as a function of day of assessment.

To estimate the magnitude of the AUC_g cortisol difference between participants lower and higher in attachment anxiety, we used the covariate-adjusted means at 1 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).

Table 1. Mixed-Model Results: Attachment Anxiety as a Predictor of Each Outcome

Outcome and time of assessment	b^a	F	p
Cortisol: Day 1	0.20	$F(1, 309) = 3.26$.072
Cortisol: Days 2 and 3 AUC_g	3.77	$F(1, 269) = 8.46$.004
Cortisol: Days 2 and 3 morning rise	0.19	$F(1, 579) = 5.57$.019
Cortisol: Days 2 and 3 postrise decline	0.22	$F(1, 894) = 10.05$.002
CD3 ⁺ T cells: Days 1 and 2	−0.26	$F(1, 260) = 15.49$	< .001
CD45 ⁺ T cells: Days 1 and 2	−0.22	$F(1, 267) = 12.56$	< .001
CD3 ⁺ CD4 ⁺ helper T cells: Days 1 and 2	−0.17	$F(1, 274) = 6.32$.013
CD3 ⁺ CD8 ⁺ cytotoxic T cells: Days 1 and 2	−0.27	$F(1, 268) = 10.26$.002

Note: Day 1 was the day of the visit to the Clinical Research Center; Days 2 and 3 were the following 2 days. AUC_g = area under the curve with respect to ground.

^aThese unstandardized coefficients were derived from log-transformed scores.

Table 2. Mixed-Model Results: Attachment Avoidance as a Predictor of Each Outcome

Outcome and time of assessment	<i>b</i> ^a	<i>F</i>	<i>p</i>
Cortisol: Day 1	-0.21	<i>F</i> (1, 283) = 5.44	.020
Cortisol: Days 2 and 3 <i>AUC_g</i>	-0.20	<i>F</i> (1, 265) = 0.03	.855
Cortisol: Days 2 and 3 morning rise	-0.01	<i>F</i> (1, 595) = 0.01	.939
Cortisol: Days 2 and 3 postrise decline	-0.02	<i>F</i> (1, 884) = 0.08	.774
CD3 ⁺ T cells: Days 1 and 2	0.08	<i>F</i> (1, 226) = 2.36	.126
CD45 ⁺ T cells: Days 1 and 2	0.05	<i>F</i> (1, 229) = 0.97	.325
CD3 ⁺ CD4 ⁺ helper T cells: Days 1 and 2	0.08	<i>F</i> (1, 238) = 1.98	.161
CD3 ⁺ CD8 ⁺ cytotoxic T cells: Days 1 and 2	0.04	<i>F</i> (1, 231) = 0.41	.525

Note: Day 1 was the day of the visit to the Clinical Research Center; Days 2 and 3 were the following 2 days. *AUC_g* = area under the curve with respect to ground.

^aThese unstandardized coefficients were derived from log-transformed scores.

We followed-up on the *AUC_g* analyses by separately investigating the morning rise in cortisol (7:00 a.m.–7:45 a.m.) and the decline following the morning rise (7:45a.m.–8:30 p.m.). Examining these patterns individually allowed us to determine whether attachment-anxiety-related differences in cortisol levels were specific to a particular time of day. Consistent with the *AUC_g* analyses, findings revealed that participants with higher attachment anxiety produced more cortisol during both the morning rise and the decline following the morning rise than those with lower attachment anxiety. The interaction between attachment anxiety and time of assessment was non-significant for both time frames, which indicated that the slope of cortisol over time did not differ as a function of 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. Each model included the main effects of attachment anxiety and day of assessment and the interaction between the two. Participants who were more anxiously attached had significantly fewer numbers of CD3⁺ T cells, CD45⁺ T cells, CD3⁺CD4⁺ helper T cells, and CD3⁺CD8⁺ cytotoxic T cells than those who were less anxiously attached. The nonsignificant interaction between anxiety and day of assessment in each model indicated that the strength of the relationships between attachment anxiety and the immune parameters did not differ as a function of day of assessment. Furthermore, participants with higher attachment anxiety (+1 *SD*) had 19% fewer CD3⁺ T cells, 15% fewer CD45⁺ T cells, 11% fewer CD3⁺CD4⁺ helper T cells, and 22% fewer

CD3⁺CD8⁺ cytotoxic T cells than those with lower attachment anxiety (–1 *SD*).

The relationships between attachment anxiety and two of the immune markers were moderated by gender, CD45⁺: *F*(1, 203) = 4.38, *p* = .038; CD3⁺CD4⁺: *F*(1, 209) = 5.42, *p* = .021. Simple-slopes analyses revealed that women with higher attachment anxiety had fewer CD45⁺ T cells, *b* = –0.30, *F*(1, 170) = 17.05, *p* < .001, and CD3⁺CD4⁺ helper T cells, *b* = –0.27, *F*(1, 170) = 11.30, *p* = .001, than women with lower attachment anxiety. Attachment anxiety for men was unrelated to CD45⁺ T cells, *b* = –0.08, *F*(1, 162) = 0.90, *p* = .344, and CD3⁺CD4⁺ helper T cells, *b* = –0.01, *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 2 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* = –0.08, *F*(1, 247) = 5.05, *p* = .026, CD45⁺ T cells, *b* = –0.09, *F*(1, 247) = 7.50, *p* = .007, and CD3⁺CD4⁺ helper T cells, *b* = –0.08, *F*(1, 246) = 5.47, *p* = .020, and marginally related to fewer CD3⁺CD8⁺ cytotoxic T cells, *b* = –0.08, *F*(1, 245) = 3.32, *p* = .070. The nonsignificant interaction between cortisol and day of assessment indicated that the strength of the relationships between cortisol and the immune parameters did not differ as a function of 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 whether participants used birth-control medication, the most common type of medication taken in our sample ($n = 18$). We found that even after accounting for these health behaviors, 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.

Discussion

In accord with psychological data demonstrating that anxiety about relationships is a potent stressor, the results of the current study demonstrated that 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 levels and was unrelated to the immune markers. The current results are thus 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 levels and cellular immunity. These results extend attachment theory in an important new direction by demonstrating the utility of a psychoneuroimmunological approach to the study of attachment anxiety, stress, and health.

In line with prior work, the current results demonstrated that attachment anxiety is linked to the overproduction of cortisol. The present data expand on these findings by revealing that attachment-anxiety-related increases in cortisol production were linked to cellular immune dysregulation. Specifically, higher cortisol levels were related to fewer numbers of CD3⁺ T cells, CD45⁺ T cells, and CD3⁺CD4⁺ helper T cells, and were marginally related to fewer CD3⁺CD8⁺ cytotoxic T cells. These results are 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 22% in CD3⁺CD8⁺ cytotoxic T cells in our sample, supports the health implications of the current results. For instance, in one study, obese people (BMI > 35 kg/m²) had 9% fewer CD3⁺CD8⁺ cytotoxic T cells than those who were nonobese (BMI < 25 kg/m²), and the CD3⁺CD8⁺ cytotoxic T-cell

response among obese people was linked to an impaired vaccine response (O'Rourke et al., 2005; Sheridan et al., 2012). Furthermore, reductions in naive 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 levels and cellular immunity to influence health (Maunder & Hunter, 2001). For example, compared with people who were less anxiously attached, people who were 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 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, regardless of their sleep quality. These data suggest that sleep quality does not explain attachment-anxiety-related cortisol and immune alterations.

According to attachment theory, the social vigilance and hyperactivating emotional style exhibited by people with high 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 recalibrate the hypothalamic-pituitary-adrenal 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 has 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). In the current study, 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 anticipatory stress. However, participants with higher attachment anxiety produced more cortisol than those with lower attachment anxiety during the 2 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, attachment avoidance, and reactivity to an acute stressor. For example, men with higher attachment anxiety and women with higher attachment avoidance had stronger cortisol responses to a relationship-conflict discussion than men with lower attachment anxiety and women with lower attachment avoidance, respectively (Powers, Pietromonaco, Gunlicks, & Sayer, 2006). Higher attachment avoidance was related to greater interleukin-6 (a measure of inflammation) reactivity during a marital-conflict

discussion compared with a social-support discussion (Gouin et al., 2009). 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 individuals with relatively high attachment avoidance actively stay away from socially threatening situations (Shaver & Mikulincer, 2002). In contrast, 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 attachment avoidance in understanding acute stress reactivity. Utilizing a psychoneuro-immunological 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 their attachment-anxiety scores were on the low end of the spectrum. Thus, the attachment-anxiety-related differences in T-cell counts evident in this sample likely underrepresent the magnitude of 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 affect health and well-being.

Declaration of Conflicting Interests

The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

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Note

1. We had information about participants' wake-up time on Day 2 but not on Day 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 1 of the 2 days, so we were able to statistically examine whether the results differed as a function of day of assessment.

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