

## Short Communication

## Anger and fear responses to stress have different biological profiles

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## ABSTRACT

In contrast to a general model of stress, a functional model suggests that emotions may regulate stress responses in specific adaptive ways. The current study examined whether anger and fear during a challenging stress task (Trier Social Stress Task) were differentially associated with cortisol and proinflammatory cytokine responses to an acute stressor. Baseline anger and fear were related to greater cortisol and proinflammatory cytokines. However, anger reactions to the stressor were associated with greater stress-related increases in cortisol over time but not proinflammatory cytokines. In contrast, fear reactions to the stressor were associated with increases in stress-related proinflammatory cytokines over time and a decrease in cortisol. Results are consistent with the functional perspective that distinct emotional experiences appear to trigger temporally-patterned adaptive biological processes to mobilize energy in response to anger and to promote withdrawal in response to fear. Discussion focuses on the role of the HPA axis to increase available metabolic fuel and proinflammatory cytokines to prompt behavioral withdrawal.

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## 1. Introduction

A colleague aggressively berates you. Are you angry? Are you afraid? Experiences of stress can engage any of several negative emotions, such as anger or fear. A functional perspective suggests that different emotions trigger distinct cognitive, behavioral, and biological processes that facilitate dealing with the stressor at hand (Frijda, 1986; Lazarus, 1991; Tooby and Cosmides, 1990). However, such flexibility in dealing with specific stressors is difficult to explain with a general model of stress that posits a set of fixed biological responses to all stressors (e.g., Selye, 1956).

An overarching assumption in much of the research on stress is that physiological responses to threats or stressful events are generalized and share very similar, if not identical, profiles in response to all stressors. However, a functional model of stress is emerging which maintains that stress responses are adaptively tailored to deal with particular stressors (see Kemeny and Shestyuk, 2008; Weiner, 1992). Emotions are one possible means by which stress responses may be tailored to specific stressors. Distinct emotions may differentially affect stress responses by eliciting biological processes that facilitate responding to different types of stressors, such as anger-eliciting or fear-eliciting stressors. The current research examined whether two negative but distinct emotions, anger and fear, are associated with different biological responses to an acute stressor.

Stress initiates several biological adaptations, including activation of the hypothalamic–pituitary–adrenocortical (HPA) axis. When released into the system, cortisol increases available glucose, boosting the metabolic fuel expended in energy-consuming activities. In addition to such neuroendocrine responses, acute stress can also activate immune system responses. The immune system not only repairs bodily damage and fights off infection, but it can also trigger behavioral changes that minimize physical damage from injury or infection. Thus, in addition to coordinating peripheral inflammatory responses to contaminants such as bacteria and viruses, proinflammatory cytokines also signal the brain to induce “sickness behaviors”, which can include reduced eating and drinking, reduced exploratory behavior, and general social withdrawal to promote recovery and recuperation from illness or infection (Hart, 1988; Kent et al., 1992; Maier and Watkins, 1998). Because these same behavioral changes (e.g., social withdrawal) can be useful for dealing with certain psychological stressors for which withdrawal rather than confrontation may be adaptive, the immune system may have been co-opted over time to promote withdrawal in response to certain psychological stressors (see Kemeny, 2007; Maier and Watkins, 1998). Thus, emotions may coordinate neuroendocrine and immune responses to stressors in functional ways.

The idea of specific biological consequences of distinct emotions is consistent with a functional view of emotions (Kemeny, 2007; Kemeny and Shestyuk, 2008), in that cortisol and proinflammatory cytokines appear to be related to only some negative emotions. For example, variation in anger throughout the day has been linked to variation in cortisol levels throughout the day, but cortisol shows

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no such association with sadness (Adam et al., 2006). Anger is a negative emotion associated with appraisals of certainty, low risk, and relative strength (Lerner and Keltner, 2001; Mackie et al., 2000; Smith and Ellsworth, 1985), as well as a motivation to approach with a tendency to aggress (Harmon-Jones and Sigelman, 2001; Lazarus, 1991). In short, anger can be characterized as motivating confrontational behavior. Angry individuals who initiate confrontations, whether verbal or physical, would likely benefit from the metabolic fuel that the HPA axis makes available (Kemeny and Shestyuk, 2008).

Fear, in contrast, is associated with appraisals of uncertainty, risk, and relative weakness (Lerner and Keltner, 2001; Mackie et al., 2000; Smith and Ellsworth, 1985). Fear can motivate avoidance and withdrawal (Blanchard and Blanchard, 1984; Lazarus, 1991), because fearful individuals may believe they cannot overcome stressful events. In response to fear-inducing stressors, people may benefit from increases in proinflammatory cytokines that are tied to submissive withdrawal. For example, the avoidance-oriented emotion of shame has been linked to increases in proinflammatory cytokines (Dickerson et al., 2004). Similarly, a fear-driven increase in proinflammatory cytokine levels would likely increase, over time, adaptive withdrawal that can often follow fear.

Participants' baseline levels of anger, fear, cortisol, the proinflammatory cytokine interleukin-6 (IL-6), and markers of the proinflammatory cytokine tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ) were assessed at the beginning of a laboratory stress session. Participants then participated in the Trier Social Stress Test which reliably produces neuroendocrine stress responses (Kirschbaum et al., 1993) and can produce either anger or fear. Participants' post-stressor anger and fear were assessed and used to predict cortisol, IL-6, and sTNF $\alpha$ RII responses to the stressor. We predicted a differentiated effect of emotions in response to the stressor, reflecting the functional role of emotions, such that anger would be associated with increased cortisol, but only fear would be associated with increased IL-6 and sTNF $\alpha$ RII.

## 2. Method

### 2.1. Participants

One hundred eighty three students and employees (71 men, 112 women) at a large university participated in exchange for \$120. Participants with the following conditions were excluded: mental or physical health problems, use of medications affecting cardiovascular or endocrine function, current treatment from a mental health professional, diagnosis of PTSD, current use of mental health medications or oral contraceptives. Women who were pregnant or lactating were also excluded.

### 2.2. Procedure

Participants reported to the university's General Clinical Research Center between 1:30 and 4:30 in the afternoon to control for diurnal variation in cortisol (Van Cauter et al., 1996). Participants completed health questionnaires that assessed their general health and health-related behaviors. Specifically, participants described their health using a 5-point scale ranging from 1 (excellent) to 5 (poor). Participants also reported the number of servings of caffeinated beverages they consumed in the past hour, on the day of the experiment, and for the prior 7 days on average as well as the number of cigarettes they smoked and the number of alcoholic beverages they drank the day of the experiment and for the prior 7 days on average.

Ten minutes after arrival, participants provided baseline samples of oral mucosal transudate (OMT), to assess markers of im-

mune system activation reliably (Nishanian et al., 1998). An Orasure collective device (Epitope, Beaverton, OR) was placed between the lower cheek and gum to attain the OMT sample. Using a passive drool method, saliva was collected in 2.0 ml Corning<sup>®</sup> cryovials (Corning, Inc., Coning, NY) to assess cortisol. Approximately 30 min later, participants supplied a second saliva sample. Cortisol levels from the first and second saliva sample were averaged to create a measure of baseline cortisol. Using five point scales from 1 (not at all) to 5 (extremely) participants reported how angry and hostile they felt, averaged into a measure of baseline anger ( $r = .58$ ), and afraid and scared, averaged into a measure of baseline fear ( $r = .78$ ).

Each participant then completed the Trier Social Stress Task (TSST), a commonly used stress challenge that reliably elicits biological stress responses (Kirschbaum et al., 1993). Participants prepared and delivered a speech on why they would be a good administrative assistant to either no visible audience, a disapproving audience, or an approving audience. Because this audience manipulation was not relevant to the current research question, it was statistically controlled in analyses.<sup>1</sup> After delivering the speech, participants completed a mental arithmetic task in which they counted backward by 7s and by 13s from 2395 aloud while the experimenter urged them to go faster.

Approximately 30 min after beginning the TSST, participants provided a second OMT sample from which post-stressor IL-6 and sTNF $\alpha$ RII were assessed and a third saliva sample. Participants then completed post-stressor questionnaires including how angry and hostile they felt, averaged to create post-stressor anger ( $r = .59$ ), and afraid and scared, averaged to create post-stressor fear ( $r = .77$ ). Approximately 10 min later, participants provided a fourth saliva sample. Cortisol levels from the third and fourth saliva samples were averaged as a measure of post-stressor cortisol.<sup>2</sup> Participants were then debriefed and dismissed.

Saliva samples were shipped for overnight delivery on dry ice to Salimetrics (State College, PA) where cortisol assays were conducted. Salivary cortisol levels were determined from a 25- $\mu$ l sample, which was assayed in duplicate by radioimmunoassay using the HS-cortisol High Sensitivity Salivary Cortisol Enzyme Immunoassay Kit (Salimetrics LLC, State College, PA). The HS-cortisol assay allows for robust results when saliva samples have a pH within the range of 3.5–9.0. All samples were within this pH range.

The proinflammatory cytokine assays were conducted at the Center for Interdisciplinary Research in Immunology and Disease (CIRID) at the University of California, Los Angeles. IL-6 was measured using the IMx automated microparticle enzyme immunoassay system (Abbot, Abbott Park, IL). We assessed the soluble receptor for TNF- $\alpha$  (sTNF $\alpha$ RII) because it is more reliably measured than TNF- $\alpha$  itself (Diez-Ruiz et al., 1995). sTNF $\alpha$ RII was measured with Quantikine Human sTNF-RII enzyme immunoassay kit manufactured by R&D Systems (Minneapolis, MN). Protein in oral fluids was quantified by the Bradford method using the Bio-Rad protein assay kit with bovine plasma albumin as the standard. All IL-6 results are reported using analyte-to-protein ratios, because this measure controls for individual differences in salivary flow rate, and is more reliable than the analyte values alone (Dickerson et al., 2004).

<sup>1</sup> The audience condition did not differentially affect fear or anger reactions to the stressor. Following assumptions for the use of covariates, we verified that the audience manipulation did not interact with any predictor variables in predicting any dependent variables.

<sup>2</sup> Cortisol levels of saliva samples 3 and 4 were combined because, based on the established time course of cortisol (Kirschbaum et al., 1993), both samples reflect responses to the stressor. The same pattern of results emerges if samples 3 and 4 are analyzed separately.

**Table 1**  
Baseline and post-stressor mean values of cortisol, IL-6, and sTNF $\alpha$ RII.

Variables	Baseline	Post-stressor
Cortisol (pg/ml)	.15 <sup>a</sup>	.24 <sup>b</sup>
IL-6 (pg/ml)	.33 <sup>a</sup>	.45 <sup>b</sup>
sTNF $\alpha$ RII (pg/ml)	2.63 <sup>a</sup>	2.76 <sup>b</sup>

Note: Values within rows not sharing similar superscripts are significantly different from each other at  $p < .05$ .

### 3. Results

Table 1 presents the mean values for baseline and post-stressor levels of cortisol, IL-6, and sTNF $\alpha$ RII. In all cases, baseline mean values are significantly lower than post-stressor levels of cortisol, IL-6, and sTNF $\alpha$ RII.

#### 3.1. Simple correlations

Table 2 presents the correlations among baseline measures. Baseline anger and fear were positively associated with baseline cortisol. Baseline fear was positively associated with IL-6, whereas baseline anger was only marginally positively associated with IL-6. A similar, but weaker, pattern emerged for sTNF $\alpha$ RII such that baseline fear was marginally positively associated with sTNF $\alpha$ RII, whereas anger showed no association with sTNF $\alpha$ RII. Baseline cortisol was not associated with baseline levels of IL-6 or sTNF $\alpha$ RII.

Table 3 presents the correlations among post-stressor measures. Post-stressor anger, but not fear, was marginally associated with post-stressor cortisol. In contrast, post-stressor fear, but not anger, was associated with IL-6. Neither post-stressor anger nor post-stressor fear were associated with sTNF $\alpha$ RII. Post-stressor cortisol was negatively associated with post-stressor IL-6, but not associated with sTNF $\alpha$ RII.

#### 3.2. Regression analyses

To examine the associations between emotions and cortisol in response to the stressor, a regression analysis predicting post-stressor cortisol was conducted that controlled for the audience manipulation, health questionnaire items (i.e., general health, caffeine intake, alcohol consumption, and smoking), baseline emotions, and baseline cortisol levels.<sup>3</sup> As displayed in Table 4 and Fig. 1, when post-stressor anger and post-stressor fear were entered as simultaneous predictors of post-stressor cortisol, anger was significantly positively associated with cortisol,  $p = .018$ , whereas fear was significantly negatively associated with cortisol,  $p = .042$ .<sup>4</sup>

To examine the hypothesized association between emotions and IL-6 and sTNF $\alpha$ RII, identical regression models that controlled for baseline IL-6 and sTNF $\alpha$ RII, respectively, were conducted.<sup>5</sup> As displayed in Table 4, fear was significantly positively associated with IL-6,  $p = .003$ , as expected, whereas anger was not associated with levels of IL-6,  $p = .93$ . A similar pattern emerged for sTNF $\alpha$ RII, although the positive relationship between fear and sTNF $\alpha$ RII was marginally significant,  $p = .076$ ; there was no association between

<sup>3</sup> No results interacted with the audience manipulation. Two dummy coded variables were created to control for the audience manipulation in all regression analyses.

<sup>4</sup> The correlations of post-stressor cortisol with post-stressor anger and post-stressor fear remained significant when baseline IL-6 and baseline sTNF $\alpha$ RII were included as covariates.

<sup>5</sup> IL-6 and sTNF $\alpha$ RII data were log-transformed to normalize distribution of residuals. Three outliers were excluded for producing post-stressor IL-6 scores more than three standard deviations from the mean. The correlation of post-stressor IL-6 with post-stressor fear remained significant when baseline cortisol was included as a covariate.

**Table 2**  
Correlations among baseline anger, fear, cortisol, IL-6, and sTNF $\alpha$ RII.

	Baseline anger	Baseline fear	Baseline cortisol	Baseline IL-6	Baseline sTNF $\alpha$ RII
Baseline Anger	–	.326 <sup>***</sup>	.166 <sup>†</sup>	.143 <sup>†</sup>	.066
Baseline Fear		–	.202 <sup>**</sup>	.195 <sup>**</sup>	.120 <sup>†</sup>
Baseline Cortisol			–	.073	.061
Baseline IL-6				–	.548 <sup>***</sup>
Baseline sTNF $\alpha$ RII					–

<sup>†</sup>  $p < .10$ .

<sup>\*</sup>  $p < .05$ .

<sup>\*\*</sup>  $p < .01$ .

<sup>\*\*\*</sup>  $p < .001$ .

anger and sTNF $\alpha$ RII,  $p = .68$ . None of the reported effects were moderated by participant gender.

### 4. Discussion

We explored whether anger and fear are associated with different, theoretically adaptive patterns of biological responses to stressors. Greater anger in response to the stressor was associated with higher post-stressor cortisol levels, as predicted. Greater fear in response to the stressor was associated with lower post-stressor cortisol levels. As hypothesized, greater fear was associated with higher levels of the proinflammatory cytokine IL-6, whereas anger was not. Similarly, greater fear was marginally associated with higher levels of sTNF $\alpha$ RII, whereas anger was not. These findings are consistent with the idea that distinct emotions tailor stress responses to an acute stressor.

Post-stressor fear was negatively correlated with post-stressor cortisol levels. Because cortisol suppresses the production of proinflammatory cytokines (Robles et al., 2005), a reduction in cortisol levels facilitates the proliferation of proinflammatory cytokines. Consistent with this point, the post-stressor cortisol and post-stressor IL-6 were negatively correlated. Because fear-driven reduction of cortisol would presumably enhance the biological and behavioral consequences of fear-driven IL-6 production, both the increase in IL-6 and the decrease in cortisol are consistent with the idea that fear coordinates a functional response to a fear-eliciting stressor.

The negative correlation between fear and cortisol emerged because the regression analysis controlled for anger. When anger was not controlled, fear was not correlated with cortisol. Controlling for the shared negative valence between the two emotions leaves only characteristics of fear that distinguish it from anger (e.g., uncertainty) to correlate with dependent variables like cortisol. Such an approach may reveal effects of distinct emotions that are attributable to distinctive properties of emotions other than their negative valence.

The current findings are correlational and preclude inferences about causation. However, the observed endocrine and immune effects were most likely triggered by central nervous system processes, such as the rapid emotional responses to the stressor. Thus, we speculate that the specific appraisals unique to fear and anger precede the endocrine and immune system responses observed here.

The present study examined only two emotional responses to stressors. Sadness is a reasonable response to some stressors and may similarly promote withdrawal from the situation, perhaps mediated by changes in proinflammatory cytokines. Although sadness is an unlikely emotional response to the stressor in the current study, our findings suggest that the association between distinct emotions and biological processes might potentially be enlightening with regard to other emotional responses to stress.

**Table 3**  
Correlations among post-stressor anger, fear, cortisol, IL-6, and sTNF $\alpha$ RII.

	Post-stressor anger	Post-stressor fear	Post-stressor cortisol	Post-stressor IL-6	Post-stressor sTNF $\alpha$ RII
Post-stressor Anger	–	.432 <sup>***</sup>	.131 <sup>†</sup>	.096 <sub>s</sub>	.057
Post-stressor Fear		–	.010	.156 <sub>s</sub> *	.086
Post-stressor Cortisol			–	–.191	–.099 <sup>***</sup>
Post-stressor IL-6				–	.609 <sup>***</sup>
Post-stressor sTNF $\alpha$ RII					–

<sup>†</sup>  $p < .10$ .  
\*  $p < .05$ .  
\*\*\*  $p < .001$ .

**Table 4**  
Post-stressor anger and fear predicting post-stressor cortisol, IL-6, and sTNF $\alpha$ RII.

Predictor variables	Post-stressor cortisol $\beta$	Post-stressor IL-6 $\beta$	Post-stressor sTNF $\alpha$ RII $\beta$
Post-stressor anger	.293 <sub>s</sub> *	–.007 <sup>**</sup>	–.036
Post-stressor fear	–.253 <sub>s</sub> *	.237 <sup>**</sup>	.159 <sup>†</sup>

<sup>†</sup>  $p < .10$ .  
\*  $p < .05$ .  
\*\*  $p < .01$ .

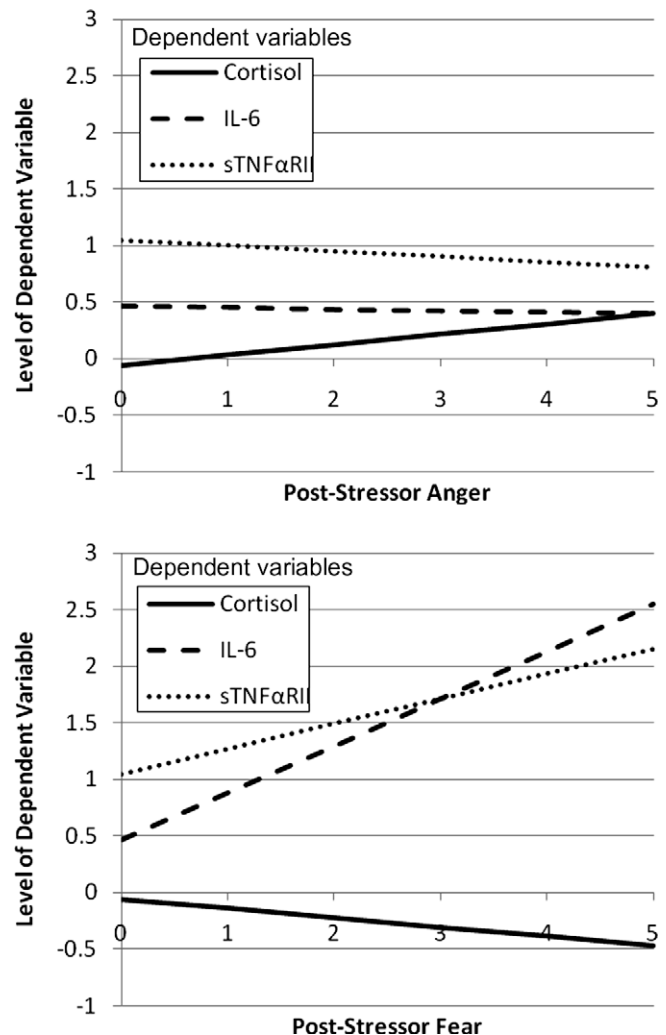
Note: Analyses of post-stressor cortisol, IL-6, and sTNF $\alpha$ RII controlled for baseline levels of cortisol, IL-6, and sTNF $\alpha$ RII, respectively. All three regressions controlled for the audience manipulation, health questionnaire items, and baseline emotions.

These results may be revealing as to the time course and coordination of psychological and biological stress responses. Because the production of cortisol and proinflammatory cytokines is slow, relative, for example, to sympathetic activation, it is unlikely that these processes play a central role in the immediate fight or flight response to acute stressors. Other faster processes such as changes in cardiovascular functioning are more likely to mobilize the energy expended in immediate fight or flight responses. Instead, emotion-driven increases in cortisol or proinflammatory cytokines may supplement, over time, the initial generalized reaction to stressors, in more specific, emotion-consistent, and adaptive ways. For example, anger-driven increases in cortisol can supplement available energy for confrontation. In contrast, fear-driven increases in proinflammatory cytokines may motivate withdrawal when retreat may be advantageous without precluding individuals from an initial flight response driven by cardiovascular activity. Thus, emotions may have relatively undifferentiated early fight or flight responses to acute stressors; however, over time more differentiated effects of distinct emotions may emerge and may shape endocrine and immune processes in functional manners. Stress has been found to initiate processes that lead to deferred benefits in other contexts (e.g., production and distribution of leukocytes that prepare the body to mend potential future injury (Dhabhar and McEwen, 1999; Viswanathan et al., 2002); the present findings suggest a potential role for emotions in shaping delayed benefits.

Although energy is required for both the fight response linked to anger and the flight response linked to fear, based on the current findings, the motivation to withdraw from a fear-inducing threat may be more important and adaptive for people feeling afraid than the availability of additional energy through the release of cortisol. Because increased cortisol would inhibit increases in withdrawal-linked proinflammatory cytokine production, it may be that a fearful individual sacrifices the additional energy that would be made available by cortisol in exchange for increases in the motivation to withdraw from fear-inducing situations.

In summary, the present findings provide beginning support for a functional model of responses to stress in which biological processes are associated with specific emotional responses to stress-

ors. As such, emotions appear to be likely mechanisms by which stress responses are modified in adaptive ways. Anger, a confrontative emotion, demonstrated an association with increased HPA axis activity in response to the stressors, consistent with the idea that energy resources are needed following confrontative responses to stress. Fear, by contrast, was associated with enhanced proinflammatory cytokine activity, especially IL-6, effects that are consistent with promoting withdrawal which can often follow states of heightened fear.



**Fig. 1.** Post-stressor anger predicting post-stressor cortisol, IL-6, and sTNF $\alpha$ RII (top panel). Post-stressor fear predicting post-stressor cortisol, IL-6, and sTNF $\alpha$ RII (bottom panel). Analyses of post-stressor cortisol, IL-6, and sTNF $\alpha$ RII controlled for baseline levels of cortisol, IL-6, and sTNF $\alpha$ RII, respectively. All three regressions controlled for the audience manipulation, health questionnaire items, and baseline emotions.

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## References

- Adam, E.K., Hawkley, L.C., Kudielka, B.M., Cacioppo, J.T., 2006. Day-to-day dynamics of experience-cortisol associations in a population-based sample of older adults. *Proc. Natl. Acad. Sci. USA* 103, 17058–17063.
- Blanchard, D.C., Blanchard, R.J., 1984. Affect and aggression: an animal model applied to human behavior. *Adv. Stud. Aggress.* 1, 1–62.
- Dhabhar, F.S., McEwen, B.S., 1999. Enhancement of the immunization/sensitization phase of cell-mediated immunity: the role of acute stress and adrenal stress hormones. *Neuroimmunomodulation* 6, 213.
- Dickerson, S.S., Kemeny, M.E., Aziz, N., Kim, K.H., Fahey, J.L., 2004. Immunological effects of induced shame and guilt. *Psychosom. Med.* 66, 124–131.
- Diez-Ruiz, A., Titz, G.P., Zangerle, R., Baier-Bitterlich, G., Wachter, H., Fuchs, D., 1995. Soluble receptors for tumor necrosis factor in clinical laboratory diagnosis. *Eur. J. Haematol.* 54, 1–8.
- Frijda, N.H., 1986. *The Emotions*. Cambridge University Press, Cambridge, England.
- Harmon-Jones, E., Sigelman, J., 2001. State anger and prefrontal brain activity: evidence that insult-related relative left-prefrontal activation is associated with experienced anger and aggression. *J. Pers. Soc. Psychol.* 80, 797–803.
- Hart, B.L., 1988. Biological basis of the behavior of sick animals. *Neurosci. Biobehav. Rev.* 12, 123–137.
- Kemeny, M.E., 2007. Emotions and the immune system. In: Ader, R. (Ed.), *Psychoneuroimmunology*. Academic Press, New York, pp. 619–630.
- Kemeny, M.E., Shestyuk, A., 2008. Emotions, the neuroendocrine and immune systems, and health. In: Lewis, M., Haviland-Jones, J.M., Barrett, L.F. (Eds.), *Handbook of Emotions*. Guilford Press, New York, pp. 661–675.
- Kent, S., Bluthé, R.M., Kelley, K.W., Dantzer, R., 1992. Sickness behavior as a new target for drug development. *Trends Pharmacol. Sci.* 13, 24–28.
- Kirschbaum, C., Pirke, K., Hellhammer, D.H., 1993. The 'Trier Social Stress Test' – a tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology* 28, 76–81.
- Lazarus, R.S., 1991. *Emotion and Adaptation*. Oxford University Press, New York.
- Lerner, J.S., Keltner, D., 2001. Fear, anger, and risk. *J. Pers. Soc. Psychol.* 81, 146–159.
- Mackie, D.M., Devos, T., Smith, E.R., 2000. Intergroup emotions: explaining offensive action tendencies in an intergroup context. *J. Pers. Soc. Psychol.* 79, 602–616.
- Maier, S.F., Watkins, L.R., 1998. Cytokine for psychologists: implications of bidirectional immune-to-brain communication for understanding behavior, mood, and cognition. *Psychol. Rev.* 10, 83–107.
- Nishanian, P., Aziz, N., Chung, J., Detels, R., Fahey, J.L., 1998. Oral fluids as an alternative to serum for measurement of markers of immune activation. *Clin. Diagn. Lab. Immunol.* 5, 507–512.
- Robles, T.F., Glaser, R., Kiecolt-Glaser, J.K., 2005. A new look at chronic stress, depression, and immunity. *Curr. Dir. Psychol. Sci.* 14, 111–115.
- Selye, H., 1956. *The Stress of Life*. McGraw-Hill, New York.
- Smith, C.A., Ellsworth, P.C., 1985. Patterns of cognitive appraisal in emotion. *J. Pers. Soc. Psychol.* 48, 813–838.
- Tooby, J., Cosmides, L., 1990. The pas explains the present: emotional adaptations and the structure of ancestral environments. *Ethol. Sociobiol.* 11, 375–424.
- Van Cauter, E., Leproult, R., Kupfer, D.J., 1996. Effects of gender and age on the levels and circadian rhythmicity of plasma cortisol. *J. Clin. Endocrinol. Metab.* 81, 2468–2473.
- Viswanathan, K., Bilbo, S.D., Dhabhar, F.S., 2002. Acute stress administered before primary immunization or before challenge enhances skin cell mediate immunity to keyhole limpet hemocyanin (KLH). *Brain Behav. Immun.* 16, 222.
- Weiner, H., 1992. *Perturbing The Organism: The Biology of Stressful Experiences*. University of Chicago Press, Chicago.