

## Stress and Specificity: Reply to Miller (2009)

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T. F. Denson, M. Spanovic, and N. Miller (2009) meta-analytically tested the hypotheses that specific appraisals and emotions would predict cortisol and immune responses to laboratory stressors and emotion inductions. Although the cortisol data supported the integrated specificity hypothesis, G. E. Miller (2009) raised questions concerning the extent to which the immunity data supported specificity. The authors respond to these concerns.

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In this issue of *Psychological Bulletin*, we presented a meta-analytic review designed to test the hypotheses that specific cognitive appraisals and emotions would influence cortisol and immune responses to laboratory stressors in what would have been a primarily adaptive manner for our ancestors (Denson, Spanovic, & Miller, 2009). In this test of the integrated specificity hypothesis (Weiner, 1992), we demonstrated numerous specificity effects for cortisol. As we acknowledged in our article, the immunity data were less clear. Miller (2009) raised a number of important issues regarding immunity and specificity. Although Miller largely concurred with the specificity effects we obtained for cortisol, he did question the effects of rumination on cortisol. We first discuss the rumination issue and subsequently respond to Miller's primary concerns regarding the immunity data.

### Rumination and Cortisol

Although the majority of his commentary focused on specificity and immunity, Miller also questioned the extent to which individuals in the studies we analyzed were capable of experiencing rumination during laboratory stressors. First, Miller (2009) noted that "almost by definition, rumination is a cognitive process that happens after a stressor has ended, not during it" (p. 856). Scientific definitions do not place this temporal constraint on rumination. A popular definition of rumination describes the construct as "conscious thinking directed toward a given object for an extended period of time" (Martin & Tesser, 1989, p. 306), and the core process underlying rumination, perseverative cognition, has been defined as "the repeated or chronic activation of the cognitive representation of one or more psychological stressors" (Brosschot, Gerin, & Thayer, 2006, p. 114; for a review on repetitive thought, see Watkins, 2008). Rumination can be experienced prior to a stressor, as during the preparation period for a speech task. More important, rumination is possible even during brief laboratory

stressors. Such "online" rumination might include intrusive self-monitoring thoughts (e.g., "Am I performing well enough?" "What does the experimenter think of me?") or aversive self-awareness (e.g., "I am really upset right now"; "I should stop being so upset"). Such rumination is in contrast to stressful tasks that proceed without such parallel self-conscious thought. Indeed, the absence of such self-aware, ruminative thought processes is a prominent feature of "flow" (Csikszentmihályi, 1990). The point here is that the components of rumination that characterize its defining features as it occurs after the cessation of a stressor are highly isomorphic with those induced prior to or during the stressor. Indeed, a recent experiment found that self-reported rumination experienced just 10 min following a public speech task predicted exacerbated cortisol reactivity (Zoccola, Dickerson, & Zaldivar, 2008). Although speech tasks were the most common stress manipulation included in our meta-analytic review, other manipulations, such as viewing emotionally evocative films or listening to music, certainly left sufficient time for rumination during the task itself.

Overall, we are in partial agreement with Miller (2009), concurring that in our review the perseverative aspects of rumination (i.e., the task features that make it persist over long durations) were undoubtedly precluded from observation by the fact that the preponderance of studies contained short stressors, along with non-delayed measurement procedures. But contrary to Miller, we argue that this state of affairs might have led to an underestimation of the effects of rumination on cortisol reactivity and not a misspecification of its role.

### Specificity and Immunity

Miller's (2009) remaining concerns were related to the immunity data. In particular, Miller questioned our analytic approach, the interpretation of immune cell counts, and the lack of autonomic nervous system (ANS) indicators in our review. We address each of these matters in turn. With regard to our analytic approach, Miller questioned the number of statistical tests conducted for each outcome. Our analytic approach provided a compromise between Type I and Type II error. First, we conducted post hoc tests only (a) after controlling for four variables likely to affect immune responses (see Equation 7 on p. 834; Denson et al., 2009) and (b) in the presence of a subsequent significant variance component,

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which indicated that variance remained to be explained. Furthermore, our approach was conservative in that we allowed control variables to remain in the model at a liberal threshold ( $p < .10$ ). It is true that we did not correct for familywise error once these criteria were met. Given that the number of manipulations for any given immune outcome ranged from 13 to 28, we reasoned that correcting for familywise error in addition to our already fairly conservative criteria (i.e., control variables and subsequent significant variance component) would have resulted in an unnecessarily inflated Type II error rate.

Most of the immunity studies included in our meta-analytic review assessed immune cell counts rather than functional assays. It is the latter that provide direct information about immune system function. This omission of functional assays was not for lack of trying or interest. With the exception of studies of natural killer cell cytotoxicity, there were simply too few social stress or emotion induction studies with a wide enough variety of manipulations at the time we conducted our literature search with which to examine specificity effects on functional outcomes. We agree that the interpretation of immune cell counts is problematic, as discussed in some detail in our article:

It is unclear to what extent transient stress-induced changes to immune cell numbers are broadly indicative of adaptive or maladaptive immune system functioning if at all. Thus, although researchers often regard the up-regulation of immunity as synonymous with increased cell counts, and vice versa for down-regulation, the bottom line is that the current state of the literature allows speculation only about the veracity of this assumption. (Denson et al., 2009, p. 846)

Furthermore, we noted that “without clear answers to these questions, it is difficult to come to firm conclusions regarding the role of specific emotions and cognitive appraisals influencing immune system function” (Denson et al., 2009, p. 846).

Thus, although cell counts might not allow for a definitive test of specificity, they are also limited in providing support for generality. Alternatively, one could envision complementary effects such that generality might best explain changes in the simple redistribution of cell numbers, whereas specific emotions and appraisals might best account for changes in immune system function. We believe that as the number of studies utilizing functional outcomes increases, so will our understanding of the effects of emotions and appraisals on the immune system.

Miller (2009) bemoaned the lack of ANS indicators in our review. We agree that a high-inference meta-analytic review of that literature would be worthwhile and informative. Indeed, we touched on this topic in our article with regard to sympathetic nervous system hormones (Denson et al., 2009, p. 846). A number of meta-analyses have already demonstrated specificity effects of individual differences in depression, anger, and hostility on cardiovascular outcomes (Barth, Schumacher, & Herrmann-Lingen, 2004; Friedman & Booth-Kewley, 1987; Suls & Wan, 1993; Suls, Wan, & Costa, 1995; Van der Kooy et al., 2007). Thus, ANS indicators are worthy candidates for investigation. Although we limited our review to cortisol and immunity, we do not believe that the lack of ANS indicators diminishes the support for specificity observed in our meta-analysis. Certainly, there are a number of interesting physiological variables worthy of investigation; however, including all of them in a single meta-analysis would have

left little room in the pages of *Psychological Bulletin* for other articles. We look forward to future developments in this area.

## Conclusion

Should we throw out the specificity hypothesis or “narrow its scope to cortisol” as Miller (2009, p. 855) suggested? Our answer to that question is “not just yet.” In light of our meta-analytic support for specificity effects on cortisol, it would be unusual indeed if other systems were unaffected by specific appraisals and emotions. As ongoing and future research utilizing a diverse array of experimental manipulations and physiological outcomes (including functional immunity) continues to be conducted, we are confident that so too will evidence in support of specificity continue to accumulate. We look forward to future research investigating appraisals, emotions, and physiology.

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