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In Search of Integrated Specificity: Comment on Denson, Spanovic, and Miller (2009)

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Abstract

Psychologists have long been interested in the integrated specificity hypothesis, which maintains that stressors elicit fairly distinct behavioral, emotional, and biological responses, molded by selective pressures to meet specific demands from the environment. This issue of *Psychological Bulletin* features a meta-analytic review of the evidence for this proposition by Denson, Spanovic, and Miller (2009). It concludes that the meta-analytic findings support the "core concept behind the integrated specificity model (p. XX)" and reveal that "within the context of a stressful event, organisms produce an integrated and coordinated response at multiple levels (i.e., cognitive, emotional, physiological; p. XX)." In this commentary I argue that conclusions like this are unwarranted given the data. Aside from some effects for cortisol, in fact, there was little evidence of specificity, and most of the significant findings reported would be expected by chance alone. I also contend that Denson et al. fail to consider some important sources of evidence bearing on the specificity hypothesis, particularly how appraisals and emotions couple with autonomic nervous system endpoints and functional indices of immune response. If selective pressures did give rise to an integrated stress response, such pathways almost certainly would have been involved. By omitting such outcomes from the meta-analysis, the authors have overlooked what are probably the most definitive tests of the specificity hypothesis. As a result, the field is back where it started: with a lot of affection for the concept of integrated specificity, but little in the way of definitive evidence to refute or accept it.

Denson, Spanovic, and Miller (2009) present a meta-analytic review of evidence for the integrated specificity hypothesis. Under this hypothesis, stressors elicit fairly distinct behavioral, emotional, and biological responses, which have been molded by selective pressures to meet specific demands imposed by the environment (Weiner, 1992). This idea will strike a familiar chord in many readers. Almost from the time that it was introduced by William James in 1884 (James, 1884), the notion of specificity has captivated the attention of psychologists interested in adaptation. Over the last century, dozens of studies have attempted to establish it empirically, with varying degrees of success (see Levenson, 2003; for are view of the literature on the autonomic specificity of emotions). As a result, the viability of the specificity hypothesis remains uncertain today, and a source of continuing controversy within the emotions field.

Despite the empirical uncertainty surrounding it, the concept of specificity has remained conceptually attractive to researchers, especially those focusing on stress-related phenomena

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(Kemeny, 2003). The reason is that positing a situation-specific response, which is behaviorally, emotionally, and biologically integrated, helps theorists to answer some otherwise difficult questions (Weiner, 1992). For example, why do people show differing biobehavioral responses to the same threat? (Because they appraise the threat in different ways, which has downstream influences on emotion and physiology.) And why do some threats evoke different biobehavioral responses than others? (Because they pose unique adaptive demands, which trigger different appraisals, emotions, and physiology.) Thus, the notion of integrated specificity helps researchers navigate around some rough conceptual terrain, i.e., individual differences in responsivity and stressor-specific physiology, which might otherwise function as roadblocks to progress in this area.

It is with these issues in mind that many readers will find the paper by Denson, Spanovic, and Miller (2009) of interest. These authors culled data from 80 different experimental manipulations of stress, which had assessed cortisol secretion and immune outcomes in subjects engaged in public-speaking, marital-conflict, and emotion-induction tasks. Then, using a thoughtfully conceived "high-inference" coding system, they had judges rate how likely subjects in each study were to have experienced a variety of appraisals, emotions, and cognitions. To see how tightly these states were linked with biological responses to stressors, the authors entered them into multilevel models, alongside a handful of covariates that represented potential alternative explanations. The results led the authors to conclude that "Our results are consistent with the core concept behind the integrated specificity model. That is, within the context of a stressful event, organisms produce an integrated and coordinated response at multiple levels (i.e., cognitive, emotional, physiological; p. XX)."

My contention is that this is a charitable interpretation of the findings. The analyses did yield some evidence of specificity. Consistent with what was documented in a previous meta-analysis of this literature (Dickerson& Kemeny, 2004), cortisol levels rose in response to stressors that were judged to evoke threat, to elicit concern about negative social evaluation, and to trigger rumination. However, aside from these effects, there was little evidence of specificity. In fact, of the roughly 175 associations estimated for immune outcomes,[1] only 9 of them reached the .05 threshold for statistical significance, a number that would be expected by chance alone. And of the 29appraisals, emotions, cognitions, and composites assessed, none showed consistent associations across more than 2biological outcomes. (The exception was for anticipation, which was linked with counts of lymphocytes, T-cells, and T-helper cells, outcomes which are mathematically dependent upon one another). Emotions like fear and anger, which most theorists consider prime candidates for specificity (Davidson, 1994; Levenson, 2003), did not show any reliable biological correlates, and neither did worry, shame, guilt, or sadness.

With a pattern of findings like this, it becomes difficult to muster enthusiasm for integrated specificity, except perhaps in the case of cortisol. Yet the authors see things differently, concluding that "During acutely stressful occurrences, cortisol and immune reactivity to stress can be enhanced or attenuated depending on the emotions and appraisals elicited in specific situations (p. XX)." This is a sweeping conclusion given the results of their analysis, and one that assigns a lot of causal leverage to emotions and appraisals, despite the findings that few of those measured actually predicted outcomes reliably. It is especially difficult to reconcile the statement about immune reactivity with the results that are presented in the manuscript. As noted above, only 5% of the immune effects reached significance. Even if we assume that all

[1]This is probably a conservative estimate of the number of analyses conducted. The paper explored the influence of 29 subjectively coded moderators on 8 different aspects of immune responsivity to stress. Because there was no residual variance to explain for 2 outcomes, moderator analyses were carried out for only 6 immune endpoints. Thus, the total number of analyses was 29 * 6 = 174. However, some of the immune outcomes showed non-linear changes over the course of stressors. It is not clear if the authors examined whether the moderators predicted these nonlinear effects. If they did, the number of analyses would be even larger.

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of these effects are genuine, using them as a basis for making blanket statements about "immune function" is a stretch.

In fact, the meta-analysis surveyed a limited number of immune outcomes, and all but 1 of them were simple counts of different cell types, rather than indicators of functionality per se. Treating these data as a proxy for global immune function is inappropriate. The immune system is bit like a football team. To "win" the battle against invading pathogens, each of its cellular players needs to perform well in its distinct role and do so in synchrony with its teammates. No reasonable fan would make a prediction about how well his or her team will play based on the number of defensive backs or wide receivers that it has on the field at a given moment. And so it is with the immune system: To know whether its function has been enhanced or suppressed by stress, one would have to survey a much broader range of outcomes than is present in the current analysis.

So what to conclude from the results of the meta-analysis? Some would argue that if they are taken at face value, we need to abandon the integrated specificity hypothesis, or at least narrow its scope to cortisol. But before a such a verdict is rendered, it would be useful to step back and consider whether the hypothesis got the scientific equivalent of a fair trial. In my opinion the answer to this question is a definitive "no," because the review didn't look for specificity in places where it is most likely to be present, and as a result may have overlooked the most critical evidence.

Take the autonomic nervous system (ANS) as an example. The ANS has been the focus of most of the theory and research on specificity to date, and the peripheral structures it regulates are the only loci where consistent physiologic differences between various emotions and appraisals have been observed (e.g., Ekman et al., 1983; Levenson et al., 1992; Tomaka et al., 1993; Tomaka et al., 1997). Along with the hypothalamic-pituitary-adrenocortical (HPA) axis, the ANS is the major conduit linking the brain, where stressful experiences are registered and processed, with peripheral structures like the heart, lungs, and muscles, which facilitate coping behaviors like fighting and fleeing. The ANS was critical in solving many of our ancestors' most pressing challenges, e.g., predation, especially those that demanded rapid changes in behavior and physiology to be made. (Its actions can unfold within seconds of the appearance of a threat, whereas it takes at least 15 minutes for cortisol to be secreted and have effects on a target tissue.) Thus, if selective pressures in the ancestral world gave rise to an integrated stress response, the ANS almost certainly would have been involved, functioning as a conduit through which the brain could rapidly direct activity of various organs and tissues in the periphery. Although there have been dozens and dozens of studies on ANS responses to stress over the past several decades, they were omitted from the Denson et al. (2009), leaving what is probably the most definitive source of evidence for specificity out of consideration.

The review did look for evidence of specificity in the immune system, and there are solid theoretical reasons to believe it could be present there. When our ancestors were fighting with or fleeing from predators, they faced a high probability of being wounded and acquiring infections. Some preparatory activation of white blood cells would have been advantageous in these circumstances, because it would decrease the interval between these events and mobilization of the immune response (Dhabhar & McEwen, 2001; Segerstrom & Miller, 2004). The most likely place for an such an integrated response to manifest is a functional indicator of responsivity—for example, how much cytokine a cell makes when it encounters a pathogenic stimulus or how much it proliferates when stimulated to do so. Many studies in the literature have assessed these types of outcomes and shown that stress primes some cells, like monocytes, to respond more aggressively to pathogenic material the next time that they are exposed it (Steptoe et al., 2007). If selective pressures were prone to couple fear or anger with an immune function, these sorts of processes would seem to be likely candidates.

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Yet Denson et al. (2009) include just one indicator of functional responsivity, natural killer cell cytotoxicity. Instead, they focus heavily on counts of cell types, assuming that these values provide information relevant to functional activity. For example, in interpreting the data on correlates of lower T-cell counts, they state that "judgments of anticipation were also associated with downregulation of cell-mediated immunity (p. XX)" and go on to explain that this would "produce an adaptive release of energy when it is needed, while simultaneously suppressing energy expenditure on cell-mediated immunity (p. XX)." This conjecture might be accurate, but it is premised upon multiple assumptions: that more cells can be equated with better function in a roughly linear fashion; that T-lymphocytes alone can be used to gauge cell-mediated immunity, without a consideration of the other cellular players it involves; that the number of T-cells in peripheral circulation is indicative of the function of T-cells in the lymphoid organs; and that having fewer cells in circulation relative to lymphoid organs is metabolically efficient. Few immunologists would be comfortable making any of these assumptions. Indeed, to test hypotheses about functions of the immune system, the focus should be on readouts that are in fact functional in nature.

Interpretation of the rumination data is also more complicated than is acknowledged. Almost by definition, rumination is a cognitive process that happens after a stressor has ended, not during it. It follows, then, that any cortisol or immune response elicited by rumination would be observed after the stressor has ended, not during it. But in many studies that informed the meta-analysis, the biological endpoints were measured on just one occasion, immediately following the stressor. This timeline makes it difficult to interpret any of the positive findings. It is unlikely that subjects would have begun ruminating so quickly and, even if they had, that the biological residue of that process would be evident just a few minutes later. Had these analyses focused on outcomes measured during stressor recovery, they would be much more interpretable.

So where does this leave us? I'm afraid that the answer is back where we started, with a lot of affection for the concept of integrated specificity, but little in the way of definitive evidence to refute or accept it.

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