Recontextualizing the Behavioral Immune System Within Psychoneuroimmunology

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We believe that the concept of the behavioral immune system (BIS) picks out an important set of phenomena, namely, the relationships between the immune system and disease-avoidant psychology. However, our enthusiasm is tempered by the recognition that (a) there are unresolved ambiguities in the BIS concept, which has been used in a variety of (sometimes inconsistent) ways; (b) many types of phenomena it has been used to identify are already well characterized in other disciplines that have received inadequate attention by BIS researchers; and that (c) these disciplines offer a broader contextualization of the phenomena. We argue that the BIS concept should be recontextualized within and integrated with such research programs. More specifically, we believe that the BIS should be set within the framework of psychoneuroimmunology. We consider several ways in which the BIS might be differentiated from psychoneuroimmunology, arguing that none of these clearly sets the BIS apart. These include (a) the notion of the BIS a purely defensive, prophylactic response to disease threats; (b) the BIS as concerned primarily with disgust-related responses; (c) the BIS’s role in generating large-scale effects of pathogens on social structure; (d) the BIS as a primarily psychological, rather than a mechanistic, account of psycho-immune interactions; and (e) the BIS as a specifically evolutionary, rather than descriptive, approach to disease-avoidant psychology and behavior. We believe that centering BIS research within psychoneuroimmunology will better capture the value and novelty of BIS research, more accurately reflect the intentions of BIS researchers, and better advance their aims.

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We believe that the concept of the behavioral immune system (BIS) picks out an important set of phenomena, namely, the relationships between the immune system and disease-avoidant psychology. However, our enthusiasm is tempered by the recognition that (a) there are unresolved ambiguities in the BIS concept and it has been used in a variety of (sometimes inconsistent) ways, (b) many types of phenomena it has been used to identify are already well characterized in other disciplines that have received inadequate attention by BIS researchers, and that (c) these disciplines offer a broader theoretical contextualization of the phenomena. We argue that the BIS concept should be recontextualized within and integrated with such research programs. More specifically, we believe that the BIS should be set within the framework of psychoneuroimmunology (PNI).

While neuroimmunology encompasses both peripheral and central nervous systems, and psychoimmunology focuses on the interactions between specifically psychological variables and immune function, PNI highlights the contributions of the central nervous system, and the ways in which the brain instantiates psychological constructs, and mediates interactions be-
between psychological variables and the immune system. Its object of study is the psychoneuro-immune system (PNIS). Work in PNI was initially largely restricted to how mental states can influence physical health outcomes, addressing comparatively simple effects of peripheral immune responses on psychology, and largely concerned with “gross” nonspecific changes in both psychology and immune responses. However, especially in the last 15 years or so, the field has grown enormously, and there is an increasing recognition of the breadth and complexity of the interactions among the immune system and quite high-level psychological variables, including the fact that such effects on cognition can be highly specific, and not limited to general parameters. For example, the immune system has been linked to the cognitive, affective, and behavioral aspects of a range of psychiatric disorders, either as a cause, effect, or both, including, for example, depression (Janssen, Caniato, Verster, & Baune, 2010; Leonard & Myint, 2009; Müller, 2014; Schiepers et al., 2005; Tanaka et al., 2012), and schizophrenia (Debnath & Venkatasubramanian, 2013; Mondelli & Howes, 2014; Müller & Dursun, 2011; Müller, 2014). As we shall see, work by BIS theorists adds to the catalogue of such effects.

While it is true that once we conceptualize the BIS within PNI, the novelty of the contributions made by BIS researchers will become clearer, we nevertheless caution that we should avoid putting too much weight on the BIS concept, which should be seen more as a rallying point for multidisciplinary interaction, than as identifying a novel system or discipline, a feature that it shares with similar disciplines that have emerged alongside it, such as “ecoimmunology,” discussed later. We consider several ways in which the BIS might be differentiated from PNI, arguing that none of these clearly sets the BIS apart. These include (a) the notion of the BIS a purely defensive, prophylactic response to disease threats; (b) the BIS as concerned primarily with disgust-related responses; (c) the BIS’s role in generating large-scale effects of pathogens on social structure; (d) the BIS as a primarily psychological, rather than a mechanistic, account of psycho-immune interactions; and (e) the BIS as a specifically evolutionary, rather than descriptive, approach to disease-avoidant psychology and behavior.

The BIS As Disease Prophylaxis

As suggested by the term itself, the BIS encompasses the specifically behavioral components of disease avoidance. There is a need for a concept to refer specifically to the behavioral aspects of disease avoidance (i.e., an ethology of disease avoidance), especially in comparative studies under naturalistic conditions where the ability to measure and manipulate disease-related variables is limited (Nunn & Altizer, 2006). Some work that is frequently cited by BIS researchers falls into such a category (Hart, 1990, 2011). However, as Schaller notes when introducing the term, the BIS would better be called the “psychological immune system,” but because that term was already in use, referring to a distinct set of processes (Gilbert, Pinel, Wilson, Blumberg, & Wheatley, 1998), Schaller chose “behavioral” (Schaller, 2006, p. 97). Hence, the term “behavioral” is not restricted to ethology, but should be taken as referring to psychology more generally, as when psychology is called “behavioral science.” For example, Schaller and colleagues (Schaller, 2006; Schaller & Duncan, 2007; Schaller & Park, 2011) define the BIS as “a suite of psychological mechanisms that (a) detect cues connoting the presence of infectious pathogens in the immediate environment, (b) trigger disease-relevant emotional and cognitive responses, and thus (c) facilitate behavioral avoidance of pathogen infection” (Schaller & Park, 2011, p. 99).

The term “behavioral” has another connotation in the BIS literature, namely as referring to psychological and behavioral defenses that serve to avoid disease in the first place, short of activating the physiological mechanisms of the classical immune system (CIS). This is perhaps the primary way in which the BIS is distinguished from related immunological concepts. The BIS is nearly always described as providing a first-line prophylactic defense against disease, and this is presented as one of its primary advantages, the idea being that whereas the CIS is merely reactive, engaged only once pathogens have been contacted, the BIS serves to avoid the costs associated with CIS reactions to active infections. For example, Neuberg, Kenrick, and Schaller (2011) define the BIS as “a system designed not to fight pathogens postinfection
but rather to avoid infection in the first place” (p. 1045).

However, this approach to differentiating the BIS is problematic. First, a large body of work tells us that the relationships between our psychology and immune function also promote a variety of psychological in response not only to potential infection, but also to active infection, potentially serving a number of functions. For example, “sickness behavior” in response to infection (and its psychological correlates) have been hypothesized to function to facilitate recovery, conserve resources, avoid further infection, prevent transfer to other group members, and avoid conflict in a weakened state (Adelman & Martin, 2009; Dantzer & Kelley, 2007; Maes et al., 2012). In line with this, some BIS researchers have incorporated responses to active infection into their definition of the BIS. For example, Fincher and Thornhill (2012) define the BIS as including not only antiparasite psychology, but also “psychology and behavior that manages infectious diseases when they occur” (p. 62). Sickness behavior has been a central topic in PNI.

Another problem with interpreting the BIS as an exclusively prophylactic system is the extent to which we can draw a clear distinction between the BIS and the CIS, and the degree to which this reflects a difference between preventative versus reactive responses. There is a large body of work in PNI documenting the deep intertwining of psychological variables and the CIS, much of which has been neglected by BIS theorists. Furthermore, even the work of some BIS theorists calls into question the tenability of such a distinction. For example, Schaller claims that the BIS is “an integrated set of psychological mechanisms that facilitate prophylactic behavioral defense against pathogens . . . Previsouly unexplored, however, is the intriguing possibility that these processes might also have an influence on the real immune system” (Schaller et al., 2010, p. 649). Schaller et al. (2010) demonstrate that simple visual exposure to signs of illness primes the CIS to deal with potential infection. While it is true that the particular effects found by Schaller et al. were previously unexplored, the notion that such psycho-immune linkages are likely to occur has long been suggested by PNI.

Others have found similar effects. Miller and Maner (2011) found that activation of the CIS (recent illness) increased activation of the BIS in the form of attention to and avoidance of perceived disease threats (disfigured individuals). Stevenson et al. (2012) found that activation of the BIS via induction of disgust increased body temperature, and also up-regulated oral immune markers. More broadly, Rubio-Godoy et al. (2007) point to a number of interconnections between the CIS and psychological disease avoidance via the shared use of serotonin systems. Finally, Macmurray et al. (2014) present evidence that variants in cytokine regulatory genes are likely to be pleiotropically linked with behavioral and psychological responses via the action of cytokines on the central nervous system. They studied the gamma-interferon (IFNG) +874 A-allele, which is known to down-regulate serotonergic activity, thereby allowing it to affect behavioral and psychological variables. The IFNG +874 A-allele is associated with greater susceptibility to a variety of infectious diseases, but also confers protection against pregnancy loss, thereby potentially trading off greater reproductive success against increased disease risk. The authors hypothesized that such increased disease risk may be offset by psychological and behavioral tendencies that protect against infection, determined by genetic factors pleiotropically linked to the IFNG +874 A-allele. They found that the IFNG +874 A-allele was associated with increased harm avoidance, and decreased exploratory excitability and extraversion. While noting that their results present some support for BIS theories, Macmurray et al. specifically criticize the notion that the CIS is merely reactive, suggesting instead that the CIS also mediates proactive responses, both traditional physiological responses, and psychological defenses.

Hence, the BIS and CIS are deeply intertwined in both directions: the BIS can activate the CIS, and the CIS itself is involved in modifying psychological responses as an integrated part of such responses, rather than simply being a separate line of defense that is only activated once infection occurs. All of this suggests that, while there may be instances of psychological defenses that neither activate, nor are activated by the CIS, the BIS and CIS may not be merely two distinct but highly interactive systems. Instead, given the depth of immune-psychology interactions and the degree to which these systems are intertwined, it is unlikely that prophyl-
lactic cognition or behavior can be cleanly separated from activation of the immune system proper. This is precisely the picture presented by PNI. Indeed, results such as these, including Schaller and colleagues’ own work, are, in effect, examples of PNI.

The BIS and Disgust

Disgust has perhaps been the primary disease-avoidant psychological response studied by BIS theorists, and is hypothesized to underlie many of the individual- and social-level responses associated with the BIS (Clay et al., 2012; Duncan & Schaller, 2009; Inbar et al., 2009, 2012; Prokop, Usak, & Fančovičová, 2010; Terrizzi et al., 2013; Tybur et al., 2010). Disgust, in turn, has received more attention in the BIS literature than in other related literatures, and BIS theorists have considerably advanced our understanding of disgust in ways that enrich the aims of related literatures. Many of these authors have suggested that the BIS bears a special relationship to disgust, so perhaps we should construe the BIS as emphasizing disgust-specific elements of disease avoidance. There is general agreement that one of the primary evolved functions of disgust is to provide protection against pathogens, so a focus on disgust in BIS research makes eminent sense.

However, we caution against postulating too exclusive a relationship between disgust and the BIS. First, not all disease-avoidant psychological responses are mediated by disgust, but also appear to involve fear, or other related emotions. Indeed, it appears that in other animals disgust is restricted to the avoidance of orally incorporated pathogens, and that it is only in humans that disgust mediates reactions to pathogens that are conveyed via nonoral routes (e.g., insects and bodily fluids), whereas avoidance of such pathogens in other animals is mediated via different, fear-like reactions (Kavaliers et al., 2003, 2005; Clark & Fessler, 2014). Second, while disgust may have a special connection to disease and immune variables in virtue of its primary functions in pathogen avoidance, it is worth emphasizing (to a greater extent than the BIS literature has) that PNI has identified immune correlates of many emotions. For example, shame involves immune responses that have been hypothesized to derive from more basic ancestral responses in response to loss and injury in violent encounters (Dickerson et al., 2004). Immune correlates have also been found for fear, anxiety, and stress (Denson et al., 2009; Glaser & Kiecolt-Glaser, 2005; Segerstrom & Miller, 2004), and anger and aggression (Chrousos, 2009; Moons, Eisenberger, & Taylor, 2010). Hence, the preparatory role of psycho-immune responses in emotions is not restricted to disgust. Finally, in humans at least, disgust has arguably acquired functions that have nothing to do with disease avoidance, such as the avoidance of suboptimal mates (sexual disgust), and the rejection of norm violators (moral disgust; Fessler & Navarrete, 2004; Borg, Lieberman, & Kiehl, 2008; Rozin et al., 2010; Tybur et al., 2009, 2013). This further raises the possibility that immune system reactions originally designed purely to fight infection, (e.g., immune-induced nausea), may have been co-opted for functions unrelated to disease avoidance, for example, the use of immune-induced nausea to activate the cognitive mechanisms underlying disgust in the service of mate choice or norm enforcement (Clark & Fessler, 2014). All of this should provide caution against postulating too close an identification the BIS with disgust-related responses.

The Effects of Pathogens on Social Structure: The BIS and Ecoimmunology (EI)

Another area that has received considerable attention from BIS researchers is the effects of parasites on large-scale social structure and behaviors, and such theorists have provided novel arguments concerning such effects. For example, Fincher and Thornhill (2012) have argued that parasite prevalence affects a wide range of social variables, such as collectivism versus individualism, and the psychological mechanisms that help to mediate them, for example, personality traits such as extraversion and introversion. While Fincher and Thornhill cite the BIS concept, here the BIS is seen as one mediator of the broader effects of pathogens on social structure and intergroup relations proper, which they call the Parasite-Stress Theory of Sociality (PSTS), and the concept itself does not play a prominent role in their articulation of these dynamics. However, as they note, the PSTS and the BIS concept overlap with the aims of another discipline, EI, which emerged at about the
same time (Martin et al., 2011; Viney & Riley, 2014). EI represents the synthesis of results from many subdisciplines, and advocates a cross-level, cross-disciplinary methodology. More specifically, EI theorists emphasize (a) the effects of environmental factors under natural conditions, (b) shifting from primarily genetic approaches to whole-organism approaches, (c) developmental and other epigenetic factors, (d) variability in immune responses at the level of individual differences, as well as (d) variability because of ecological factors such as latitude, temperature, and season. EI especially emphasizes trade-offs between immune responses and other selective pressures, such as growth, reproduction, and social status, and how these affect variation in immunological strategies at both individual and social levels, stressing that that immune responses must be both up- and down-regulated in response to such pressures. As Trotter et al. (2011) put it, “Organisms evolve optimal immunity...not maximal immunity” (p. 41). EI is contrasted in these respects with traditional immunology, which has tended to see the lack of immune responses under various conditions as a failure, rather than as an adaptive trade-off.

The BIS concept, too, would benefit from a greater consideration of such trade-offs. As noted above, the BIS is typically construed as mediating low-cost prophylactic defenses against infection. However, the notion of “cost” involved here is usually restricted to the costs of CIS responses considered in themselves (e.g., metabolic costs), and is not typically situated within the nexus of selective pressures that determine the overall evolutionary costs of psychological disease avoidance. However, when situated within a broader evolutionary cost-benefit perspective, psychological defenses can be equally or more costly than CIS responses. This is abundantly illustrated in the dynamics of disgust responses that have been the focus of much BIS research, as disgust responses are modulated in response to other factors, for example, hunger, sexual arousal, or different stages of the reproductive cycle. Such trade-offs provide yet another reason to question the characterization of the BIS as an exclusively prophylactic response.

Of particular importance for research into the BIS and PSTS is the concept of the social immune system, as articulated by Cremer and colleagues in the context of EI (Cremer et al., 2007; Cremer & Sixt, 2009). Cremer at al. consider the notion of a social immune system in the context of insect colonies that form superorganisms (e.g., ants and termites), examining both prophylactic and reactive responses to disease, at the level of both the individual and the colony. Cremer at al. focus primarily on drawing analogies between such colonies and individual immune systems in vertebrates (e.g., comparisons of specialized antiparasite workers in a colony and T-cells in the immune system of individual vertebrates) thereby demonstrating common principles underlying various branches of immunology. Cremer et al. do not devote much attention to analogies between the social behavior of insect colonies and the social behaviors of other species. However, such insights are also applicable to social-level responses of many species, humans in particular, a point articulated by Cotter and Kilner (2010). Nevertheless, while the BIS has been employed in accounts of social-level phenomena, we believe that its proper subject is the psychological mechanisms operating at the level of individual psychology, while theories such as the PSTS focus on social variables proper. BIS mechanisms mediate, and are shaped by, social variables, but nevertheless constitute a distinct level of analysis.

Back to PNI

If we are correct that the proper place of the BIS concept lies in individual-level level psychology, then we believe that employing the framework of PNI will often be preferable to utilizing the concept of the BIS. As Schaller notes, the BIS would better be termed the “psychological immune system” (Schaller, 2006, p. 97). If so, then the corresponding discipline would best be called psycho-immunology. Such a discipline already exists, and, when we incorporate the role of the central nervous system in mediating psycho-immune interactions, is best labeled PNI. While research done using the BIS concept certainly goes beyond existing research in PNI in its discussion of, for example, social factors and their underlying psychology, it is unclear that the BIS offers an advance over existing concepts, or picks out some well-defined, theoretically meaningful subset of such disciplines, via an emphasis on behavioral pro-
phyllaxis, disgust, and so forth. In any case, the BIS literature has thus far failed to maximize the integration of the BIS literature with well-established disciplines and theories of the same phenomena, including detailed accounts of the neurophysiological mechanisms underlying interactions between psychology and immune function developed within PNI.

PNI is both more established and more integrated with other disciplines. The term was introduced in 1975 (Ader & Cohen, 1975). There are journals, textbooks, and departments of PNI, and a Google Scholar search for the term “psychoneuroimmunology” yields ~25,000 results. In contrast, use of the BIS concept is new (“behavioral immune system” yields ~250 results), and has largely been restricted to a particular school of researchers. Specifically, it has by evolutionary psychologists (broadly construed) who are interested in a cluster of issues concerning the influence of pathogens on psychology and social structure, especially by social psychologists interested in intergroup emotions, disgust in particular (Clay et al., 2012; Fincher & Thornhill, 2012; Schaller, 2011; Terrizzi et al., 2013; Thornhill et al., 2010; Tybur et al., 2010; Van Vugt & Park, 2009). This literature is not tightly connected to the broader PNI program, in either direction: only a handful of PNI researchers have referenced the BIS concept, and, more surprisingly, there are very few mentions of PNI in the BIS literature, where, even if one wishes to avoid an identification of BIS theory with PNI, PNI is nonetheless clearly relevant.

It might be thought that we should avoid postulating too close a relationship between the BIS and PNI on the grounds that PNI should be restricted to the “mechanistic” explanations of immune-psychology interactions. On this view, the mechanisms identified by PNI might underpin the BIS, but, strictly speaking, belong to a different level of analysis. Some psychologists see neural and physiological theories as irrelevant, or at least unnecessary, to psychological research. However, it seems to us that BIS theory is also committed to such cross-level mechanisms. Furthermore, attempts to screen off the “mechanistic” level are, in our opinion both methodologically and substantively flawed, and fail to take advantage of the breadth of research in neuroscience, immunology, and so forth that bears directly on such hypotheses. The multilevel nature of PNI offers the possibility for greater integration and illumination, and paying attention to the neural, physiological, and so forth levels allows us to talk about the evolution of different components at multiple levels, and yields insight into the evolution of the system in ways that approaching it solely from the (social) psychological level cannot. Such a multilevel, multidisciplinary approach is precisely what makes newly emerging concepts such as EI and the BIS useful.

Relatedly, it might also be thought that we should see the BIS concept as concerned specifically with the evolved nature of the behavioral, psychological, and social systems that interact with and extend the immune system proper, whereas PNI is not concerned with the evolutionary history of the PNIS, but rather simply aims to characterize the PNIS. We think this would be a mistake for several reasons. First, in principle, there is nothing that should restrict the BIS to its evolved components. The deployment of cultural strategies to deal with disease would also seem to be part of the BIS, a possibility hinted at in some PSTS papers (e.g., Fincher & Thornhill, 2012). Second, there is a rich tradition of evolutionary theorizing within PNI, with many papers devoted to the evolution of various aspects of the PNIS (Adamo, 2006; Kinney & Tanaka, 2009; Maier, Watkins, & Fleshner, 1994; Maier & Watkins, 1998; Segerstrom, 2010; Walls, 2005). If we want to refer to such specifically evolutionary considerations, then the term “evolutionary psychoneuroimmunology” would be preferable, and more consistent with the ways in which evolutionary approaches to existing disciplines have been demarcated (e.g., evolutionary psychology).

Conclusion

We hypothesize that the excitement surrounding, and perceived novelty of, the BIS concept may be attributable more to the growing realization on the part of those in the BIS literature of the importance of the kinds of processes that have been identified within PNI, rather than to the identification of novel concepts or phenomena. It thereby serves as a rallying point for evolutionary psychologists, and as an avenue for the introduction of ideas from other disciplines. It is similar in this respect to other new lines of inquiry such as EI, which
represents less a newly demarcated field or set of phenomena than the introduction of broader findings from, for example, evolutionary developmental biology and systems biology into traditional ecology and immunology, and an increasing recognition of the intersection of traditionally distinct disciplines. We believe that the penetration of such awareness will be facilitated by grounding BIS research in PNI. We believe that the overlap between PNI and the BIS concept is clear. There is a prima facie case to be made that they are very often describing the same system(s), and that BIS theorists are effectively engaging in PNI research. Indeed, work by some BIS theorists (e.g., Schaller et al.’s (2010) work on immune priming in disgust) is exemplary of the kind of cross-level research that will be facilitated by contextualizing the BIS within PNI. Ceteris paribus, it is preferable to choose a more established term to describe a set of phenomena. Established concepts are preferable for the pedestrian reason that they facilitate cross-referencing, and allow research over time to coalesce around particular concepts. Such theoretical entrenchment is also usually associated with a greater degree of integration with related concepts and disciplines. This is, after all, the goal of BIS theory. In any case, whether the BIS is subsumed into PNI or not, its advocates should devote more effort to articulating the relevance of the disciplines to one another. This, we think, will better capture the value and novelty of BIS research, more accurately reflect the intentions of BIS researchers, and better advance their aims.

References


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