DOI: 10.1111/spc3.12371

ARTICLE

WILEY

The behavioral immune system: Current concerns and future directions

Joshua M. Ackerman[1] | Sarah E. Hill[2] | Damian R. Murray[3]

[1]University of Michigan

[2]Texas Christian University

[3]Tulane University

Correspondence
Joshua Ackerman, Department of Psychology, University of Michigan, Ann Arbor, MI, USA.
Email: joshack@umich.edu

Funding information
Division of Behavioral and Cognitive Sciences, Grant/Award Number: BCS-1226731; National Science Foundation, Grant/Award Number: BCS-1226731

Abstract

The behavioral immune system is a motivational system that helps minimize infection risk by changing cognition, affect, and behavior in ways that promote pathogen avoidance. In the current paper, we review foundational concepts of the behavioral immune system and provide a brief summary of recent social psychological research on this topic. Next, we highlight current conceptual and empirical limitations of this work and delineate important questions that have the potential to drive major advances in the field. These questions include predicting the ontological development of the behavioral immune system, specifying the relationship between this system and the physiological immune system, and distinguishing conditions that elicit direct effects of situational pathogen threats versus effects that occur only in interaction with dispositional disease concerns. This discussion highlights significant challenges and underexplored topics to be addressed by the next generation of behavioral immune system research.

1 | INTRODUCTION

Perhaps no other factor in our evolutionary history has shaped human behavior more than the consequences of pathogen and parasitic infection. Even today, close to one quarter of all deaths around the world are due to infectious disease—a rate more than double that from violence or injury (WHO, 2015). In light of these substantial costs, people and other animals have evolved a broad range of responses intended to cope with pathogens, including cultural adaptations such as food handling practices (Billing & Sherman, 1998; Henrich, 2015) and biological adaptations such as sickness behavior (Dantzer & Kelley, 2007). The most specialized of these adaptations is the constellation of biological mechanisms comprising the physiological immune system. Although activation of the physiological immune system is remarkably efficient at managing pathogenic threats, it is metabolically costly and imperfect. Inflammatory responses following infection, for example, contribute to diseases of aging (Conner & Grisham, 1996; Khansari, Shakiba, & Mahmoudi, 2009), and immune activation temporarily inhibits the ability to solve other adaptive problems, such as attracting a mate or caring for offspring (Dantzer, 2001; Hart, 1988). In light of these drawbacks, another set of defensive mechanisms function to mitigate the threat of disease *before* infection occurs—a behavioral immune system (Murray & Schaller, 2016; Schaller, 2011; Schaller & Park, 2011).

In this article, we briefly review theory and research on the behavioral immune system and the implications of this system for classic domains of social psychology. We then raise a number of unresolved questions, empirical concerns, and directions for future work within this growing literature.

2 | THE BEHAVIORAL IMMUNE SYSTEM

The term "behavioral immune system" describes a complex suite of cognitive, affective, and behavioral mechanisms that ultimately help prevent pathogen transmission in the face of recurrent infectious disease threats. Proximately, this system functions by detecting threat-relevant cues in the environment and activating responses aimed at diminishing those threats. As with many evolved threat management systems, behavioral immune activity is marked by both contextual sensitivity and biases that aid adaptive responding.

Because not all situations afford identical levels of infection-related threat, nor all people identical levels of infection-related vulnerability, the behavioral immune system exhibits *functional flexibility*—sensitivity to the costs and benefits of pathogen avoidance (e.g., Schaller & Neuberg, 2012; Schaller & Park, 2011; Schaller, Park, & Kenrick, 2007). For example, people who perceive themselves more vulnerable to disease express more ethnocentrism and less sociability—two behavioral immune manifestations—in response to pathogen cues than do those who perceive themselves less vulnerable (Mortensen, Becker, Ackerman, Neuberg, & Kenrick, 2010; Navarrete & Fessler, 2006; Park, Schaller, & Crandall, 2007). This type of personal and contextual sensitivity, where factors implying vulnerability predict stronger responses and factors implying relative safety predict muted responses, characterizes many of the findings in this literature. The behavioral immune system is also marked by a tendency to *overgeneralize*—it is prone to activation even in cases where pathogen threats are absent. Because the specific symptoms caused by pathogens are ever-changing and highly variable, evolution of a system to perfectly classify individuals who do and do not harbor pathogens would be impossible. Evolved responses to such signal detection problems often rely on liberal identification criteria when targeting potential threats, what is known as the "smoke detector principle" (Haselton & Nettle, 2006; Nesse, 2005). Just as a smoke detector can sound an alarm in response to benign cues such as cooking fumes, a number of noninfectious physical and mental abnormalities can act as cues to the presence of pathogens in others, including disfigurements (Ackerman et al., 2009; Miller & Maner, 2011), disabilities (Park, Faulkner, & Schaller, 2003), obesity (Lund & Miller, 2014; Park et al., 2007), and elderly appearance (Duncan & Schaller, 2009; Miller & Maner, 2012). Although innocuous cues trigger many false alarms, the costs of these may be relatively small and outweighed by the costs of potentially missing true disease threats.

Outputs generated by the behavioral immune system can be characterized as either *proactive* or *reactive*—an important distinction receiving only recent attention in the literature. Reactive responses emerge in the presence of information connoting an immediate infection risk, such as smelling a foul odor or seeing someone with open sores. These responses generally take the form of avoidant or prophylactic behaviors (Curtis, de Barra, & Aunger, 2011; Tybur, Lieberman, Kurzban, & DeScioli, 2013), such as restricted sexual attitudes (Duncan, Schaller, & Park, 2009; Murray, Jones, & Schaller, 2013; Schaller & Murray, 2008), positivity toward condom use (Tybur, Bryan, Magnan, & Caldwell Hooper, 2011), and avoidance of people possessing cues heuristically associated with illness (Park et al., 2003, 2007). Proactive responses, in contrast, are aimed at managing the long-term threat of illness. Certain habitual actions such as hygiene behavior—which exist both universally and across species—qualify as proactive management of persistent bacterial and viral threats (e.g., Curtis & Biran, 2001). More distally proactive, pathogen threat amplifies the importance people place on a potential mate's physical attractiveness, symmetry, and secondary sex characteristics, all indicators of health (e.g., Gangestad & Buss, 1993; Hill, Prokosch, & DelPriore, 2015; Jones et al., 2012; Lee & Zietsch, 2011; Young, Sacco, & Hugenberg, 2011). This preference decreases the risk of pathogen exposure because healthy people are relatively less likely to be infected themselves, and also provides "next-generation" protection for offspring because these mates are relatively more likely to possess and transmit high levels of genetic immunocompetence (Stevenson, Case, & Oaten, 2011; Tybur & Gangestad, 2011). Such work suggests that reactive and proactive

behavioral immune responses help protect individuals, their offspring, and slow the rate of horizontal disease transmission within groups.

3 | INFLUENCE OF THE BEHAVIORAL IMMUNE SYSTEM ON SOCIAL PSYCHOLOGY

Research on the behavioral immune system has revealed two key social consequences of system activation: (1) increased aversion and avoidance of unfamiliar and outgroup targets and (2) strengthened cohesion with familiar and ingroup targets. These outcomes are relevant for many processes traditionally examined in social psychology, some of which we briefly review next (also see Table 1).

3.1 | Interpersonal perception

A variety of early stage cognitive processes are recruited in the identification of potential pathogenic threats. Because of the risks of interpersonal contagion, visual attention is oriented toward people bearing cues to infection. For instance, perceivers find it more difficult to visually disengage from faces possessing physical abnormalities, even ones that only heuristically connote disease (Ackerman et al., 2009). People higher in disease concern also report greater distinctions between ingroup and outgroup members and are more likely to classify ambiguous, unfamiliar targets as threatening (e.g., Makhanova et al., 2015; Miller & Maner, 2012; Reid et al., 2012).

3.2 | Judgment and decision-making

In addition to early stage processes, infectious disease threats affect inferences, judgments, and decisions about both people and objects. Most broadly, these influences motivate choices that help perceivers avoid exposure to unfamiliar stimuli that may offer contamination dangers (e.g., Inbar & Pizarro, 2014; Peng, Chang, & Zhou, 2013). For example, pathogen concerns increase the perceived risk of uncertain decisions (Prokosch et al., 2017), leading to reduced desire

TABLE 1 Behavioral immune system research in social psychology

Social psychological domain	Behavioral immune system findings and predictions	Relevant literature
Interpersonal perception	Overgeneralization of cues associated with disease; greater attention to these cues	Ackerman et al. (2009); Makhanova, Miller, and Maner (2015); Reid et al. (2012)
Judgment and decision-making	Avoidance of controllable risk; devaluation of objects and environments associated with unfamiliar people; desire for health and attractiveness improvements	Ackerman, Tybur, and Mortensen (in press); Huang, Ackerman, and Sedlovskaya (2017); Mortensen et al. (2010); Prokosch, Gassen, Ackerman, and Hill (2017)
Close relationships	Greater preference for healthy, symmetrical, attractive mates; desire for novel sexual partners (in women)	Gangestad and Buss (1993); Hill et al. (2015); Murray et al. (2013); Tybur and Gangestad (2011); Young et al. (2011)
Stereotyping and prejudice	Aversion and stigmatization expressed toward outgroup members, especially those associated with dirtiness and disease	Faulkner, Schaller, Park, and Duncan (2004); Huang, Sedlovskaya, Ackerman, and Bargh (2011); Navarrete and Fessler (2006); Park et al. (2007)
Group processes and cultural norms	Higher conformity to cultural norms; increased collectivism; more social conservatism; stringent punishment for violating moral norms	Fincher, Thornhill, Murray, and Schaller (2008); Horberg, Oveis, Keltner, and Cohen (2009); Murray, Kerry, and Gervais (2017); Murray and Schaller (2012); Murray, Schaller, and Suedfeld (2013); Murray, Trudeau, and Schaller (2011); Thornhill, Fincher, and Aran, 2009; Wu and Chang (2012)

for social affiliation (Sacco, Young, & Hugenberg, 2014) and devaluation of consumer products that have previously been in contact with strangers (Huang et al., 2017). As a potential solution to infection threat, these concerns also motivate desire for health- and appearance-improving products and behaviors (Ackerman et al., in press).

3.3 | Close relationships

Concern about infectious disease plays an important role in our romantic pursuits as well. Chronic concerns and markers of situational threat such as high ecological pathogen prevalence increase the value people place on physical attractiveness (e.g., Ackerman et al., in press; Gangestad & Buss, 1993; Lee & Zietsch, 2011; White, Kenrick, & Neuberg, 2013) and lessen preference for mates with sex-divergent or abnormal visual features (e.g., Lee, Brooks, Potter, & Zietsch, 2015; Little, DeBruine, & Jones, 2011). Cues of pathogen prevalence (both real and perceived) are also associated with more cautious and restrictive sexual attitudes and behaviors among those hoping to avoid infection (Murray et al., 2013; Schaller & Murray, 2008), but more sexual opportunism among those who perceive their own physiological immune functioning as insufficient for survival in pathogen dense ecologies (Hill et al., 2015).

3.4 | Stereotyping and prejudice

Perhaps the most intriguing and well-studied outcome of behavioral immune activity involves stereotypes and prejudicial attitudes expressed toward outgroup members. People facing illness risks are more likely to stigmatize individuals possessing cues heuristically associated with disease (Crandall & Moriarty, 1995; Faulkner et al., 2004; Huang et al., 2011; Miller & Maner, 2012; Mortensen et al., 2010; Navarrete & Fessler, 2006; Park et al., 2007; Peterson, 2017; van Leeuwen, Park, Koenig, & Graham, 2012). For instance, the early stages of pregnancy, which are marked by suppressed immune functioning and thus infection vulnerability, are associated with increased exhibition of ethnocentrism (Navarrete, Fessler, & Eng, 2007). These types of attitudes may facilitate avoidance of people who possess novel pathogens or practices unsuited to managing local pathogen threats.

3.5 | Group processes and cultural norms

Finally, the behavioral immune system plays a role in shaping intragroup attitudes and behaviors. For example, research shows that disease threats are associated with greater conformity to social norms (Murray & Schaller, 2012; Wu & Chang, 2012) and higher levels of disgust—an emotion that motivates pathogen avoidance (Huang, Ackerman, & Newman, 2017; Oaten, Stevenson, & Case, 2009; Rozin, Haidt, & McCauley, 1993; Tybur et al., 2013)—and predict greater sensitivity to moral violations (e.g., Horberg et al., 2009; Jones & Fitness, 2008; Murray, Kerry, & Gervais, 2017; Tybur, Lieberman, & Griskevicius, 2009; Wheatley & Haidt, 2005). At the cross-cultural level of analysis, pathogen prevalence also predicts greater emphasis on moral and social norms (Murray et al., 2011; Van Leeuwen et al., 2012), including aversion to physical contact during culturally normative rituals (Murray, Fessler, Kerry, White, & Marin, 2017). Pathogen threat may even be at the root of many fundamental dimensions of culture, from individualism/collectivism (Fincher et al., 2008) to social and political orientation (Murray, Schaller, & Suedfeld, 2013; Terrizzi, Shook, & McDaniel, 2013; White et al., 2013) to religious belief (Fincher & Thornhill, 2008, 2012).

4 | CURRENT EMPIRICAL CONCERNS

The last 15 years of behavioral immune system research have provided a wealth of new knowledge regarding the long reach of human's pathogen management psychology. As the field matures, however, it faces several significant challenges and unanswered questions (Gangestad & Grebe, 2014; Tybur, Frankenhuis, & Pollet, 2014). Further, the field is currently lacking theoretical models that account for the sometimes contradictory effects documented in

the literature (Faulkner et al., 2004; Gelfand et al., 2011; Murray & Schaller, 2012; Tybur et al., 2016). Issues such as these need to be addressed empirically to advance our understanding of the structure and functioning of the behavioral immune system.

In the following sections, we highlight three issues that are paramount to advancing research in this area: (1) the interface between physiological and behavioral immune activity, in particular which physiological factors are associated with or mediate psychological outcomes, (2) the lifespan development of the behavioral immune system, and (3) when we should expect to observe main effects versus interactions between situational threat cues and chronic disease concerns.

4.1 | Linking the physiological and behavioral immune systems

To provide a functional defense against infection, both behavioral and physiological immune systems must work in concert. How these systems interact to effectively prevent or manage pathogenic threats is a relatively new question for the field. One barrier to quick resolution of this question involves the complexity of the physiological immune system. Although called a "system," this is actually an extremely complex set of systems, each of which is governed by its own set of rules, hormones, cells, and signaling proteins. These different systems are united by shared function (to detect, contain, and kill invading agents), yet each is somewhat distinct, making study of the behavioral–physiological relationship difficult to assess empirically. For example, an immunologist asked how to best measure immunocompetence (to assess its relationship with the behavioral immune responses), might typically reply: "Which part do you want to know about?" A somewhat similar case could be made about the complexity of the behavioral immune system. This "system" also involves multiple psychological processes whose individual relevance depends on the specific inputs and outputs being examined. For instance, different emotions (e.g., disgust, worry, and anger) and cognitions (e.g., vulnerability beliefs and group stereotypes) may underlie changes in interpersonal versus intrapersonal perception.

Although difficult to conduct, research examining the dynamics between the physiological and behavioral immune systems is needed to move the field forward. Of particular importance is modeling the degree to which these systems are complementary (working in tandem, providing overlapping functions) or compensatory (activation of one predicting decreased activation of the other). We describe each of these viewpoints below.

A complementary relationship implies that, relative to people whose early development was marked by little investment in immune functioning, people who have invested more in immune building should show stronger physiological and behavioral activity in response to infection threat. This positively correlated organization of the two systems reflects a common underlying immune quality, which could emerge as a function of interpersonal differences in gene frequencies, stability and resource richness of the developmental environment, or proximate somatic integrity (as influenced by injury, sleep loss, etc.). According to this perspective, for example, we should find that individuals residing in high illness risk ecologies invest heavily in both behavioral and physiological forms of pathogen defense. Although evidence for a complementary organization is limited, several studies have found that pathogen threat cues (e.g., sneezing people) not only activate behavioral avoidance strategies but also may trigger a physiological immune response, perhaps in preparation to fight off infection (Makhanova et al., 2017; Schaller, Miller, Gervais, Yager, & Chen, 2010; Stevenson, Hodgson, Oaten, Barouei, & Case, 2011). Additionally, sex differences in immune functioning exist, with women showing both relatively greater BIS and PIS responsivity (e.g., Bouman, Heineman, & Faas, 2005; Druschel & Sherman, 1999; Duncan et al., 2009), suggestive of redundant systems.

A compensatory organization, on the other hand, implies that decreased activation of one system is balanced by increased activation of the other. For example, Miller and Maner (2011) found that recently ill participants (who therefore have temporarily reduced physiological immune activity; Hendaus, Jomha, & Alhammadi, 2015) displayed heightened attention to and avoidance of disfigured individuals, a common behavioral manifestation of behavioral immune activation. Others find similar effects in women during the first trimester of pregnancy when the physiological immune system is suppressed (Navarrete et al., 2007). More recent evidence indicates that an active behavioral

immune system may allow downregulation of basal inflammation, suggesting that behavioral activation may help prevent chronic inflammation and promote long-term health and longevity (Hill et al., 2017; see also Kandrik et al., 2017). Together, these different lines of research suggest that the behavioral and physiological immune systems may work together to serve as a first line of defense against pathogens, with behavioral activity being the preferred mode of illness avoidance in environments where the behavioral management of pathogens is likely to be effective, in cases where the physiological immunity is suppressed (Miller & Maner, 2011; Navarrete et al., 2007) and in contexts where longevity is favored (Hill et al., 2017). The physiological immune system, on the other hand, is likely to be the favored form of defense in environments where behavioral management of pathogens is unlikely to be effective (e.g., working as a nurse in a hospital), where behavioral activity would be costly to fitness (e.g., being the parent of a sick child; in the face of a sexual opportunity), or among those prioritizing outcomes other than longevity (see the following life history section). Further research is needed to examine the dynamic relationship between these systems, as well as the benefits and drawbacks associated with each form of pathogen avoidance.

Another opportunity for advancement involves the need to bridge behavioral immune research with research from psychoneuroimmunology on psychological and behavioral mechanisms (sickness behavior) that function to help fight infection. Sickness behavior is characterized by anhedonia, social withdrawal, and decreases in exploratory, foraging, and sexual activity (Dantzer & Kelley, 2007). Although originally believed to be a maladaptive byproduct of infection, this change in motivational states is now understood to reflect a well-orchestrated constellation of behaviors that function to conserve energy, mitigate further bodily damage from infection, and avoid injury or predation while in a weakened state (Dantzer, 2001; Dantzer & Kelley, 2007; Kluger & Rothenburg, 1979; Medzhitov, Schneider, & Soares, 2012). Combining behavioral immune research with insights from psychoneuroimmunology sparks a number of novel questions whose answers will be essential to maturation of the field (see Clark & Fessler, 2014). Do certain forms of behavioral immune activation reflect a pre-emptive sickness behavior? How does chronic inflammation impact behavioral immune outcomes such as person perception, sensation seeking, and risk tolerance? What specific physiological mechanisms are involved in particular behavioral outcomes?

Relatedly, a number of assumptions about self-perceived illness vulnerability need to be validated using actual immunological functioning measures. The perceived vulnerability to disease scale (PVD; Duncan et al., 2009) is an index of infection resistance perceptions and chronic pathogen concerns. Although the PVD scale is widely used in behavioral immune research (e.g., Faulkner et al., 2004; Navarrete & Fessler, 2006; Prokop, Usak, & Fančovičová, 2010; Welling, Conway, DeBruine, & Jones, 2007), little is known about the degree to which scales like this one reflect actual immunological vulnerabilities. Do people who perceive themselves more vulnerable to illnesses actually have less immunocompetence than people who perceive themselves less vulnerable? Developing a valid self-report measure of immunocompetence (if such a measure can be created) will be an important next step in enhancing our understanding of the dynamic relationship between behavioral and physiological immune systems.

4.2 | Life history investment in the behavioral immune system

In an ideal world, organisms could devote a boundless supply of energy to physiological and behavioral immune defenses as well as to solutions for the myriad other adaptive problems they face (predator avoidance, child care, etc.). Furthermore, activation of these systems would be free of collateral costs, such as DNA damage from inflammation or social isolation from avoiding others who potentially harbor infectious agents. We do not inhabit such an ideal world, however. Each of us has a limited pool of bodily resources that can be dedicated to managing pathogen threats, and any pathogen management strategy that one might adopt will involve tradeoffs. Individual differences in the development of pathogen management strategies are therefore expected to vary based on features of the person and the situation that influence the costs and benefits of investing in pathogen defense versus alternative activities.

The idea that organisms make tradeoffs when allocating energetic resources to the various activities inherent in survival and reproduction is at the heart of *life history theory*, a predictive framework from the biological sciences. Broadly speaking, life history theory predicts that organisms will allocate bodily resources in ways that best promote

fitness within their local ecology (this is a developmental process, not necessarily an agentic one; Del Giudice, Gangestad, & Kaplan, 2015). Although this theory has been most frequently applied to address questions of developmental timing, sexual debut, and reproductive timing, life history theory can also be used to derive predictions about contexts that favor investment in a more or less active behavioral immune system.

One interesting implication of this framework is that investments in pathogen defense should be governed by tradeoffs that implicitly weigh the fitness costs and benefits of behavioral investment (an *avoidance* strategy) against the fitness costs and benefits of investment in the physiological immune system (a *management* strategy). For instance, individuals from relatively controllable, predictable ecologies should invest more energy in the development of a robust behavioral immune system than those from unpredictable, highly pathogenic ecologies. This is because the costs associated with pathogen avoidance are lower (i.e., people can effectively control exposure to germs) and the benefits are greater (i.e., decreased somatic damage from inflammation). Consistent with this idea, a growing body of research finds evidence for increased investment in inflammatory activity among those from unpredictable early life environments (Miller et al., 2009; Miller, Chen, & Parker, 2011). This suggests that uncontrollable environments may promote the development of a pathogen management (rather than avoidance) strategy. However, research is needed to examine whether this investment is accompanied by reduced allocation to the behavioral immune system. In addition to offering new insights into situational factors that contribute to development of the behavioral immune system, such research may provide clues into the relationship between the physiological and behavioral immune systems (see preceding section).

A second hypothesis falling from this conceptual framework is that investment in the behavioral immune system should vary depending on an individual's life history strategy—an ecologically contingent developmental pattern emerging in response to specific features of the early childhood environment (Belsky, Steinberg, & Draper, 1991; Kuzawa, McDade, Adair, & Lee, 2010). Early life environments characterized by high levels of harshness and unpredictability (e.g., low SES and inconsistent or low-quality paternal investment) sensitize people to "faster" life history strategies. These strategies are characterized by expedited physiological and sexual development, earlier sexual debut, and high mating relative to parenting efforts (Belsky, Houts, & Fearon, 2010; Chisholm, 1993; Ellis, 2004; Murray, Gildersleeve, Fales, & Haselton, 2017). Early life environments that are more benign and predictable, on the other hand, tend to encourage "slower" life history strategies, which are characterized by delayed maturation, later sexual debut, and higher parenting compared to mating effort (Ellis, Figueredo, Brumbach, & Schlomer, 2009; Kaplan & Gangestad, 2005).

Combining these insights with research on the behavioral immune system yields the prediction that individuals who favor slower life history strategies may favor increased investment in behavioral relative to physiological forms of immunity. Investment in the behavioral immune system can promote long-term health and longevity by limiting the frequency of physiological immune activity and its corresponding inflammatory response (both of which tax the body and contribute to diseases of aging). Individuals who follow faster life history strategies, on the other hand, may exhibit decreased investment in the development of an active behavioral immune system because longevity is of reduced importance and pathogen avoidance behaviors can conflict with goals relevant to a fast strategy (e.g., unrestricted sexual activity and risk-taking behaviors; e.g., Borg & de Jong, 2012). Consistent with this idea, women—who have relatively slower life history strategies due to their relatively greater investment in child-bearing and rearing—tend to have a more finely tuned behavioral immune response than men (Druschel & Sherman, 1999; Duncan et al., 2009). These predictions notwithstanding, much remains to be learned about how life history-relevant features of the person and situation influence development of the behavioral immune system.

4.3 | Situational threats, individual differences, and person by situation interactions

The behavioral immune literature documents a diverse array of triggers for pathogen-avoidant cognitions and behaviors. The most obvious of these triggers are situational cues, such as coughing or sneezing people, media reports about disease outbreaks, or disgusting sights and smells. Reactivity to situational cues is of clear adaptive significance given

they signal the potential presence of infectious agents in the immediate environment. A second type of trigger is more personality-based: wide individual differences exist in chronic concerns about disease transmission and germs (e.g., differences in disease anxiety, perceived infection vulnerabilities, or disgust sensitivity). Although the roots of these individual differences are currently not well understood, interpersonal variation likely reflects both intrapersonal and ecological tradeoffs. Intrapersonally, people vary in their ability to cope with infection as well as in their tendency to engage in behaviors that exacerbate or mitigate the chance of pathogen exposure (McDade, 2005; Round & Mazmanian, 2009). Ecologically, people exist in physical and social environments that dictate the frequency of pathogen contact (e.g., climatic variation and cultural norms; Guernier, Hochberg, & Guégan, 2004) and the means by which this contact can be managed (e.g., cooking and food taboos; Henrich, 2015). Individual differences in disease concern are therefore likely to emerge from these various influences over ontogeny (e.g., Gangestad, Haselton, & Buss, 2006; Wang, Michalak, & Ackerman, in press). For instance, growing up in a pathogen dense environment may be accompanied by chronic concerns about germs, to the extent that these concerns are effective at mitigating infection.

Much of the research on behavioral immune system outputs finds person by situation interactions, where individuals' responses to situational cues of pathogen threat differ based on their chronic levels of perceived vulnerability to infection risk. For example, people high in chronic pathogen concerns might more readily detect or respond to situational cues of threat, whereas people lower in such concerns may be less sensitive or reactive to situational cues. The literature now documents an array of such interactive effects (see Murray & Schaller, 2016). In contrast, other outcomes manifest as main effects of situational cues or chronic concerns. As of this writing, no model or explanatory framework currently accounts for *why* (or when) behavioral immune responses should be exhibited as main effects or person-by-situation interactions. This is a question of growing importance, yet it is a question without a satisfactory answer (Tybur et al., 2014). Next, we suggest some speculative possibilities.

One possibility pertains to input differences. That is, certain types of situational disease cues may be generally more evocative than others. Stronger disease threat manipulations may evoke behavioral immune responses in the majority of individuals regardless of dispositional factors. Weaker manipulations, on the other hand, may only evoke disease-avoidant responses in people chronically averse to pathogen threats. For example, experimental manipulations using pictures and vignettes of disease-relevant stimuli find that responses are dependent on individuals' reactivity to pathogen cues. For example, Miller and Maner (2012) had participants read vignettes about highly pathogenic environments and found situational by dispositional interactions on interpersonal and intergroup perceptions. Under control conditions, those higher in perceived vulnerability to disease set more lenient thresholds for categorizing targets as obese, whereas disease primes made these thresholds more lenient for all participants (Miller & Maner, 2012). Such interactive results are common (see also Ackerman et al., in press; Duncan & Schaller, 2009; Mortensen et al., 2010). A different pattern emerges in research studies employing other manipulations, however. For example, Murray and Schaller (2012) and Murray, Kerry, and Gervais (2017) had participants discuss at length the time in their life when they felt most vulnerable to disease-causing bacteria and germs and, in doing so, partly controlled for individual differences in reactivity to specific disease cues. In both of these studies, main effects of experimental condition and dispositional worry about disease emerged (on sensitivity to normative and moral violations, respectively), but no interaction occurred between the two. Thus, theoretical refinement is needed to systematically account for which types of experimental procedures are likely to result in which types of effects.

A second possibility is that the presence of main versus interactive effects may be due to output differences —differences in the outcome variables of interest. Ephemeral variation in perceived disease threat has been linked to variables ranging from person and face perception to prejudice to sexual attitudes, and these diverse domains vary widely in their psychological strength, attitudinal malleability, and relevant psychological mechanisms. It is possible that main effects of situational disease cues are more likely to emerge for relatively malleable outcomes. Examples of such variables include conformist attitudes to abstract art, liking for people who defy social norms, self-evaluation of appearance, or social categorization of novel faces (Ackerman et al., in press; Makhanova et al., 2015; Murray & Schaller, 2012; Wu & Chang, 2012). Interactive effects instead may emerge for variables that are more impervious

to perceptual or attitudinal change, such that situational disease cues are only influential in concert with high dispositional worry about disease. Consistent with this logic, interactive effects emerge for outcomes such as desire for future sexual variety, attitudes toward promiscuous sexual behavior, and self-reported social surgency (Hill et al., 2015; Mortensen et al., 2010; Murray et al., 2013). Again, however, not all results conform to this pattern.

A third possibility pertains to sample differences. Participants from different ecologies or cultures may have differentially calibrated sensitivities to disease cues based upon pathogen prevalence within their local or developmental environments (e.g., de Barra, DeBruine, Jones, Mahmud, & Curtis, 2013; Huang et al., 2017). At least one study suggests, however, that the zero-order relationship between actual local levels of disease threat and sensitivity to disease cues is either nonuniform or nonexistent: in a sample of over 11,000 participants in 30 countries, Tybur et al. (2016) did not find evidence for a relationship between levels of disease within the local ecology and average levels of disgust sensitivity. However, this nonrelationship does not imply that ecological factors are irrelevant to other outcomes or to overall calibration of the behavioral immune system.

A final possibility is that the smattering of both interactive and main effects in the behavioral immune system literature is simply due to statistical issues. As is increasingly evident in the broader social psychological literature, meta-analyses reveal that effect sizes resulting from experimental primes are often small (e.g., Weingarten et al., 2016). This area of research, much like other areas, would therefore benefit from meta-analyses of the various outcomes reviewed earlier, as well as the behavioral immune literature as a whole, in order to help researchers make correct decisions about study implementation. Terrizzi et al. (2013) took this approach in meta-analyzing the association between chronic disease concern and social conservatism and found an overall effect size in the small to medium range. As the field matures, increased use of such techniques along with modern methodological refinements (e.g., Funder et al., 2014) should more clearly inform questions of when and to what extent behavioral immune activity will be found.

5 | CONCLUSION

Recent research on the behavioral immune system has revealed its connections to diverse social psychological phenomena. The empirical concerns identified here represent, we believe, opportunities for major advancements that will help to integrate research across areas of psychology and across disciplines as well. The behavioral immune literature is a still-growing area of investigation—one which may only now be entering its prime. We look forward to the new contributions this work can provide for our understanding of physiology, personality, and social psychology.

ACKNOWLEDGMENT

This work was supported in part by National Science Foundation grant BCS-1226731 to J. M. Ackerman.

ORCID

Joshua M. Ackerman http://orcid.org/0000-0001-6322-2194

REFERENCES

Ackerman, J. M., Becker, D. V., Mortensen, C. R., Sasaki, T., Neuberg, S. L., & Kenrick, D. T. (2009). A pox on the mind: Disjunction of attention and memory in the processing of physical disfigurement. *Journal of Experimental Social Psychology*, *45*(3), 478–485.

Ackerman, J. M., Tybur, J. M., & Mortensen, C. R. (in press). Infectious disease and imperfections of self-image. *Psychological Science*. https://doi.org/10.1177/0956797617733829

Belsky, J., Houts, R. M., & Fearon, R. P. (2010). Infant attachment security and the timing of puberty: Testing an evolutionary hypothesis. *Psychological Science*, *21*(9), 1195–1201.

Belsky, J., Steinberg, L., & Draper, P. (1991). Childhood experience, interpersonal development and reproductive strategy: An evolutionary theory of socialization. *Child Development*, *62*, 647–670.

Billing, J., & Sherman, P. W. (1998). Antimicrobial functions of spices: Why some like it hot. *The Quarterly Review of Biology*, *73*(1), 3–49.

Borg, C., & de Jong, P. J. (2012). Feelings of disgust and disgust-induced avoidance weaken following induced sexual arousal in women. *PLoS One*, *7*(9), e44111.

Bouman, A., Heineman, M. J., & Faas, M. M. (2005). Sex hormones and the immune response in humans. *Human Reproduction Update*, *11*(4), 411–423.

Chisholm, J. S. (1993). Death, hope, and sex: Life-history theory and the development of reproductive strategies. *Current Anthropology*, *34*, 1–24.

Clark, J. A., & Fessler, D. M. (2014). Recontextualizing the behavioral immune system within psychoneuroimmunology. *Evolutionary Behavioral Sciences*, *8*(4), 235–243.

Conner, E. M., & Grisham, M. B. (1996). Inflammation, free radicals, and antioxidants. *Nutrition*, *12*(4), 274–277.

Crandall, C. S., & Moriarty, D. (1995). Physical illness stigma and social rejection. *British Journal of Social Psychology*, *34*(1), 67–83.

Curtis, V., de Barra, M., & Aunger, R. (2011). Disgust as an adaptive system for disease avoidance behaviour. *Philosophical Transactions of the Royal Society of London B: Biological Sciences*, *366*(1563), 389–401.

Curtis, V., & Biran, A. (2001). Dirt, disgust, and disease: Is hygiene in our genes? *Perspectives in biology and medicine*, *44*(1), 17–31.

Dantzer, R. (2001). Cytokine-induced sickness behavior: Where do we stand? *Brain, Behavior, and Immunity*, *15*(1), 7–24.

Dantzer, R., & Kelley, K. W. (2007). Twenty years of research on cytokine-induced sickness behavior. *Brain, Behavior, and Immunity*, *21*(2), 153–160.

de Barra, M., DeBruine, L. M., Jones, B. C., Mahmud, Z. H., & Curtis, V. A. (2013). Illness in childhood predicts face preferences in adulthood. *Evolution and Human Behavior*, *34*(6), 384–389.

Del Giudice, M., Gangestad, S. W., & Kaplan, H. S. (2015). Life history theory and evolutionary psychology. In D. M. Buss (Ed.), *The handbook of evolutionary psychology* (pp. 88–114). Hoboken, NJ: John Wiley and Sons, Inc.

Druschel, B. A., & Sherman, M. F. (1999). Disgust sensitivity as a function of the Big Five and gender. *Personality and Individual Differences*, *26*(4), 739–748.

Duncan, L. A., & Schaller, M. (2009). Prejudicial attitudes toward older adults may be exaggerated when people feel vulnerable to infectious disease: Evidence and implications. *Analyses of Social Issues and Public Policy*, *9*(1), 97–115.

Duncan, L. A., Schaller, M., & Park, J. H. (2009). Perceived vulnerability to disease: Development and validation of a 15-item self-report instrument. *Personality and Individual Differences*, *47*(6), 541–546.

Ellis, B. J. (2004). Timing of pubertal maturation in girls: An integrated life history approach. *Psychological Bulletin*, *130*(6), 920–958.

Ellis, B. J., Figueredo, A. J., Brumbach, B. H., & Schlomer, G. L. (2009). Fundamental dimensions of environmental risk: The impact of harsh vs. unpredictable environments on the evolution and development of life history strategies. *Human Nature*, *20*, 204–268.

Faulkner, J., Schaller, M., Park, J. H., & Duncan, L. A. (2004). Evolved disease-avoidance mechanisms and contemporary xenophobic attitudes. *Group Processes & Intergroup Relations*, *7*(4), 333–353.

Fincher, C. L., & Thornhill, R. (2008). Assortative sociality, limited dispersal, infectious disease and the genesis of the global pattern of religion diversity. *Proceedings of the Royal Society of London B: Biological Sciences*, *275*(1651), 2587–2594.

Fincher, C. L., & Thornhill, R. (2012). Parasite-stress promotes in-group assortative sociality: The cases of strong family ties and heightened religiosity *Behavioral and Brain Sciences*, *35*(2), 61–79.

Fincher, C. L., Thornhill, R., Murray, D. R., & Schaller, M. (2008). Pathogen prevalence predicts human cross-cultural variability in individualism/collectivism. *Proceedings of the Royal Society B*, *275*(1640), 1279–1285.

Funder, D. C., Levine, J. M., Mackie, D. M., Morf, C. C., Sansone, C., Vazire, S., & West, S. G. (2014). Improving the dependability of research in personality and social psychology: Recommendations for research and educational practice. *Personality and Social Psychology Review*, *18*(1), 3–12.

Gangestad, S. W., & Buss, D. M. (1993). Pathogen prevalence and human mate preferences. *Ethology and Sociobiology*, *14*(2), 89–96.

Gangestad, S. W., & Grebe, N. M. (2014). Pathogen avoidance within an integrated immune system: Multiple components with distinct costs and benefits *Evolutionary Behavioral Sciences*, *8*(4), 226–234.

Gangestad, S. W., Haselton, M. G., & Buss, D. M. (2006). Evolutionary foundations of cultural variation: Evoked culture and mate preferences. *Psychological Inquiry*, *17*(2), 75–95.

Gelfand, M. J., Raver, J. L., Nishii, L., Leslie, L. M., Lun, J., Lim, B. C., … Aycan, Z. (2011). Differences between tight and loose cultures: A 33-nation study. *Science*, *332*(6033), 1100–1104.

Guernier, V., Hochberg, M. E., & Guégan, J. (2004). Ecology drives the worldwide distribution of human diseases. *PLoS Biology*, *2*(6), 740–746.

Hart, B. L. (1988). Biological basis of the behavior of sick animals. *Neuroscience & Biobehavioral Reviews*, *12*(2), 123–137.

Haselton, M. G., & Nettle, D. (2006). The paranoid optimist: An integrative evolutionary model of cognitive biases. *Personality and Social Psychology Review*, *10*(1), 47–66.

Hendaus, M. A., Jomha, F. A., & Alhammadi, A. H. (2015). Virus-induced secondary bacterial infection: A concise review. *Therapeutics and Clinical Risk Management*, *11*, 1265–1271.

Henrich, J. (2015). *The secret of our success: How culture is driving human evolution, domesticating our species, and making us smarter*. Princeton, NY: Princeton University Press.

Hill, S.E., Gassen, J., Makhanova, A., White, J., Proffitt Leyva, R.P., Peterman, J., Prokosch, M. L., Eimerbrink, M., Nicolas, S.C., Reynolds, T.A., Maner, J.K., McNulty, J.K., Eckel, L.A., Nikonova, L., Brinkworth, J.F., Phillips, M., Mitchell, J., & Boehm, G. W. (2017). The behavioral immune system protects the body from chronic basal inflammation. Manuscript submitted for publication.

Hill, S. E., Prokosch, M. L., & DelPriore, D. J. (2015). The impact of perceived disease threat on women's desire for novel dating and sexual partners: Is variety the best medicine? *Journal of Personality and Social Psychology*, *109*(2), 244–261.

Horberg, E. J., Oveis, C., Keltner, D., & Cohen, A. B. (2009). Disgust and the moralization of purity. *Journal of Personality and Social Psychology*, *97*(6), 963–976.

Huang, J. Y., Ackerman, J. M., & Newman, G. E. (2017). Catching (up with) magical contagion: A review of contagion effects in consumer contexts. *Journal of the Association for Consumer Research*, *2*(4), 430–443.

Huang, J. Y., Ackerman, J. M., & Sedlovskaya, A. (2017). (De) contaminating product preferences: A multi-method investigation into pathogen threat's influence on used product preferences. *Journal of Experimental Social Psychology*, *70*, 143–152.

Huang, J. Y., Sedlovskaya, A., Ackerman, J. M., & Bargh, J. A. (2011). Immunizing against prejudice effects of disease protection on attitudes toward out-groups. *Psychological Science*, *22*(12), 1550–1556.

Inbar, Y., & Pizarro, D. A. (2014). Pollution and purity in moral and political judgment. *Advances in experimental moral psychology: Affect, character, and commitments*, 111–129.

Jones, A., & Fitness, J. (2008). Moral hypervigilance: The influence of disgust sensitivity in the moral domain. *Emotion*, *8*(5), 613–627.

Jones, B. C., Feinberg, D. R., Watkins, C. D., Fincher, C. L., Little, A. C., & DeBruine, L. M. (2012). Pathogen disgust predicts women's preferences for masculinity in men's voices, faces, and bodies. *Behavioral Ecology*, *24*(2), 373–379.

Kandrik, M., Hahn, A. C., Fisher, C. I., Wincenciak, J., DeBruine, L. M., & Jones, B. C. (2017). Are physiological and behavioral immune responses negatively correlated? Evidence from hormone-linked differences in men's face preferences. *Hormones and Behavior*, *87*, 57–61.

Kaplan, H. S., & Gangestad, S. W. (2005). Life history theory and evolutionary psychology. In D. M. Buss (Ed.), *The handbook of evolutionary psychology* (pp. 68–95). Hoboken, NJ: John Wiley and Sons, Inc.

Khansari, N., Shakiba, Y., & Mahmoudi, M. (2009). Chronic inflammation and oxidative stress as a major cause of age-related diseases and cancer. *Recent Patents on Inflammation & Allergy Drug Discovery*, *3*(1), 73–80.

Kluger, M. J., & Rothenburg, B. A. (1979). Fever and reduced iron: Their interaction as a host defense response to bacterial infection. *Science*, *203*(4378), 374–376.

Kuzawa, C. W., McDade, T. W., Adair, L. S., & Lee, N. (2010). Rapid weight gain after birth predicts life history and reproductive strategy in Filipino males. *Proceedings of the National Academy of Sciences*, *107*(39), 16800–16805.

Lee, A. J., Brooks, R. C., Potter, K. J., & Zietsch, B. P. (2015). Pathogen disgust sensitivity and resource scarcity are associated with mate preference for different waist-to-hip ratios, shoulder-to-hip ratios, and body mass index. *Evolution and Human Behavior*, *36*(6), 480–488.

Lee, A. J., & Zietsch, B. P. (2011). Experimental evidence that women's mate preferences are directly influenced by cues of pathogen prevalence and resource scarcity. *Biology Letters*, *7*, 892–895.

Little, A. C., DeBruine, L. M., & Jones, B. C. (2011). Exposure to visual cues of pathogen contagion changes preferences for masculinity and symmetry in opposite-sex faces. *Proceedings of the Royal Society of London B: Biological Sciences*, *278*, 2032–2039.

Lund, E. M., & Miller, S. L. (2014). Is obesity un-American? Disease concerns bias implicit perceptions of national identity. *Evolution and Human Behavior*, *35*(4), 336–340.

Makhanova, A., Miller, S. L., & Maner, J. K. (2015). Germs and the out-group: Chronic and situational disease concerns affect intergroup categorization. *Evolutionary Behavioral Sciences*, *9*(1), 8–19.

Makhanova, A., Plant, A. E., Eckel, L. A., Nikonova, L., Ackerman, J. M., & Maner, J. K. (2017). The psychology of disease avoidance: Physiological and situational antecedents of implicit social bias. Manuscript submitted for publication.

McDade, T. W. (2005). Life history, maintenance, and the early origins of immune function. *American Journal of Human Biology*, *17*(1), 81–94.

Medzhitov, R., Schneider, D. S., & Soares, M. P. (2012). Disease tolerance as a defense strategy. *Science*, *335*(6071), 936–941.

Miller, G. E., Chen, E., Fok, A. K., Walker, H., Lim, A., Nicholls, E. F., … Kobor, M. S. (2009). Low early-life social class leaves a biological residue manifested by decreased glucocorticoid and increased proinflammatory signaling. *Proceedings of the National Academy of Sciences*, *106*, 14716–14721.

Miller, G. E., Chen, E., & Parker, K. J. (2011). Psychological stress in childhood and susceptibility to the chronic diseases of aging: Moving toward a model of behavioral and biological mechanisms. *Psychological Bulletin*, *137*, 959–997.

Miller, S. L., & Maner, J. K. (2011). Sick body, vigilant mind the biological immune system activates the behavioral immune system. *Psychological Science*, *22*(12), 1467–1471.

Miller, S. L., & Maner, J. K. (2012). Overperceiving disease cues: The basic cognition of the behavioral immune system. *Journal of Personality and Social Psychology*, *102*(6), 1198–1213.

Mortensen, C. R., Becker, D. V., Ackerman, J. M., Neuberg, S. L., & Kenrick, D. T. (2010). Infection breeds reticence the effects of disease salience on self-perceptions of personality and behavioral avoidance tendencies. *Psychological Science*, *21*(3), 440–447.

Murray, D. R., Fessler, D. M., Kerry, N., White, C., & Marin, M. (2017). The kiss of death: Three tests of the relationship between disease threat and ritualized physical contact within traditional cultures. *Evolution and Human Behavior*, *38*(1), 63–70.

Murray, D. R., Gildersleeve, K. A., Fales, M. R., & Haselton, M. G. (2017). MHC homozygosity is associated with fast sexual strategies in women. *Adaptive Human Behavior and Physiology*, *3*(2), 101–117.

Murray, D. R., Jones, D. N., & Schaller, M. (2013). Perceived threat of infectious disease and its implications for sexual attitudes. *Personality and Individual Differences*, *54*(1), 103–108.

Murray, D. R., Kerry, N., & Gervais, W. M. (2017). On disease and deontology: Multiple tests of the influence of disease threat on moral vigilance. *Social Psychological and Personality Science*.

Murray, D. R., & Schaller, M. (2012). Threat (s) and conformity deconstructed: Perceived threat of infectious disease and its implications for conformist attitudes and behavior. *European Journal of Social Psychology*, *42*(2), 180–188.

Murray, D. R., & Schaller, M. (2016). The behavioral immune system: Implications for social cognition, social interaction, and social influence. *Advances in Experimental Social Psychology*, *53*, 75–129.

Murray, D. R., Schaller, M., & Suedfeld, P. (2013). Pathogens and politics: Further evidence that parasite prevalence predicts authoritarianism *PloS One*, *8*(5), e62275.

Murray, D. R., Trudeau, R., & Schaller, M. (2011). On the origins of cultural differences in conformity: Four tests of the pathogen prevalence hypothesis. *Personality and Social Psychology Bulletin*, *37*(3), 318–329.

Navarrete, C. D., & Fessler, D. M. (2006). Disease avoidance and ethnocentrism: The effects of disease vulnerability and disgust sensitivity on intergroup attitudes. *Evolution and Human Behavior*, *27*(4), 270–282.

Navarrete, C. D., Fessler, D. M., & Eng, S. J. (2007). Elevated ethnocentrism in the first trimester of pregnancy. *Evolution and Human Behavior*, *28*(1), 60–65.

Nesse, R. M. (2005). Natural selection and the regulation of defenses: A signal detection analysis of the smoke detector principle. *Evolution and Human Behavior*, *26*(1), 88–105.

Oaten, M., Stevenson, R. J., & Case, T. I. (2009). Disgust as a disease-avoidance mechanism. *Psychological Bulletin*, *135*(2), 303–321.

Park, J. H., Faulkner, J., & Schaller, M. (2003). Evolved disease-avoidance processes and contemporary anti-social behavior: Prejudicial attitudes and avoidance of people with physical disabilities. *Journal of Nonverbal Behavior*, *27*(2), 65–87.

Park, J. H., Schaller, M., & Crandall, C. S. (2007). Pathogen-avoidance mechanisms and the stigmatization of obese people. *Evolution and Human Behavior*, *28*(6), 410–414.

Peng, M., Chang, L., & Zhou, R. (2013). Physiological and behavioral responses to strangers compared to friends as a source of disgust. *Evolution and Human Behavior*, *34*(2), 94–98.

Prokop, P., Usak, M., & Fančovičová, J. (2010). Risk of parasite transmission influences perceived vulnerability to disease and perceived danger of disease-relevant animals. *Behavioural Processes*, *85*(1), 52–57.

Prokosch, M. L., Gassen, J., Ackerman, J. M., & Hill, S. E. (2017). Caution in the time of cholera: Pathogen threats decrease risk tolerance. Manuscript submitted for publication.

Reid, S. A., Zhang, J., Anderson, G. L., Gasiorek, J., Bonilla, D., & Peinado, S. (2012). Parasite primes make foreign-accented English sound more distant to people who are disgusted by pathogens (but not by sex or morality). *Evolution and Human Behavior*, *33*(5), 471–478.

Round, J. L., & Mazmanian, S. K. (2009). The gut microbiome shapes intestinal immune responses during health and disease. *Nature Reviews. Immunology*, *9*(5), 313.

Rozin, P., Haidt, J., & McCauley, C. R. (1993). Disgust. In M. Lewis & J. M. Haviland (Eds.), *Handbook of emotions* (pp. 575–594). New York: Guilford Press.

Sacco, D. F., Young, S. G., & Hugenberg, K. (2014). Balancing competing motives: Adaptive trade-offs are necessary to satisfy disease avoidance and interpersonal affiliation goals. *Personality and Social Psychology Bulletin*, *40*(12), 1611–1623.

Schaller, M. (2011). The behavioural immune system and the psychology of human sociality. *Philosophical Transactions of the Royal Society, B: Biological Sciences*, *366*(1583), 3418–3426.

Schaller, M., Miller, G. E., Gervais, W. M., Yager, S., & Chen, E. (2010). Mere visual perception of other people's disease symptoms facilitates a more aggressive immune response. *Psychological Science*, *21*(5), 649–652.

Schaller, M., & Murray, D. R. (2008). Pathogens, personality, and culture: Disease prevalence predicts worldwide variability in sociosexuality, extraversion, and openness to experience. *Journal of Personality and Social Psychology*, *95*(1), 212–221.

Schaller, M., & Neuberg, S. L. (2012). Danger, disease, and the nature of prejudice(s). *Advances in Experimental Social Psychology*, *46*, 1–54.

Schaller, M., & Park, J. H. (2011). The behavioral immune system (and why it matters). *Current Directions in Psychological Science*, *20*(2), 99–103.

Schaller, M., Park, J. H., & Kenrick, D. T. (2007). Human evolution and social cognition. In R. I. M. Dunbar, & L. Barrett (Eds.), *Oxford handbook of evolutionary psychology* (pp. 491–504). Oxford UK: Oxford University Press.

Stevenson, R. J., Case, T. I., & Oaten, M. J. (2011). Proactive strategies to avoid infectious disease. *Philosophical Transactions of the Royal Society, B: Biological Sciences*, *366*(1583), 3361–3363.

Stevenson, R. J., Hodgson, D., Oaten, M. J., Barouei, J., & Case, T. I. (2011). The effect of disgust on oral immune function. *Psychophysiology*, *48*(7), 900–907.

Terrizzi, J. A., Shook, N. J., & McDaniel, M. A. (2013). The behavioral immune system and social conservatism: A meta-analysis. *Evolution and Human Behavior*, *34*(2), 99–108.

Thornhill, R., Fincher, C. L., & Aran, D. (2009). Parasites, democratization, and the liberalization of values across contemporary countries *Biological Reviews*, *84*(1), 113–131.

Tybur, J. M., Bryan, A. D., Magnan, R. E., & Caldwell Hooper, A. E. (2011). Smells like safe sex: Olfactory pathogen primes increase intentions to use condoms. *Psychological Science*, *22*(4), 478–480.

Tybur, J. M., Frankenhuis, W. E., & Pollet, T. V. (2014). Behavioral immune system methods: Surveying the present to shape the future. *Evolutionary Behavioral Sciences*, *8*(4), 274–283.

Tybur, J. M., & Gangestad, S. W. (2011). Mate preferences and infectious disease: Theoretical considerations and evidence in humans. *Philosophical Transactions of the Royal Society B*, *366*, 3375–3388.

Tybur, J. M., Inbar, Y., Aarøe, L., Barclay, P., Barlow, F. K., de Barra, M., … Žeželj, I. (2016). Parasite stress and pathogen avoidance relate to distinct dimensions of political ideology across 30 nations. *Proceedings of the National Academy of Sciences*, *113*, 12408–12413.

Tybur, J. M., Lieberman, D., & Griskevicius, V. (2009). Microbes, mating, and morality: Individual differences in three functional domains of disgust. *Journal of Personality and Social Psychology*, *97*(1), 103–122.

Tybur, J. M., Lieberman, D., Kurzban, R., & DeScioli, P. (2013). Disgust: Evolved function and structure. *Psychological Review*, *120*, 65–84.

Van Leeuwen, F., Park, J. H., Koenig, B. L., & Graham, J. (2012). Regional variation in pathogen prevalence predicts endorsement of group-focused moral concerns. *Evolution and Human Behavior*, *33*(5), 429–437.

Wang, I. M., Michalak, N. M., & Ackerman, J. M. (in press). Threat of infectious disease. In *The SAGE handbook of personality and individual differences*, Los Angeles: Sage.

Weingarten, E., Chen, Q., McAdams, M., Yi, J., Hepler, J., & Albarracin, D. (2016). On priming action: Conclusions from a meta-analysis of the behavioral effects of incidentally-presented words. *Current Opinion in Psychology*, *12*, 53–57.

Welling, L. L. M., Conway, C. A., DeBruine, L. M., & Jones, B. C. (2007). Perceived vulnerability to disease is positively related to the strength of preferences for apparent health in faces. *Journal of Evolutionary Psychology*, *5*(1), 131–139.

Wheatley, T., & Haidt, J. (2005). Hypnotic disgust makes moral judgments more severe. *Psychological Science*, *16*(10), 780–784.

White, A. E., Kenrick, D. T., & Neuberg, S. L. (2013). Beauty at the ballot box disease threats predict preferences for physically attractive leaders. *Psychological Science*, *24*(12), 2429–2436.

World Health Organization. (2015). World health in 2015: From MDGs, millennium development goals to SDGs, sustainable development goals. Geneva, Switzerland.

Wu, B. P., & Chang, L. (2012). The social impact of pathogen threat: How disease salience influences conformity. *Personality and Individual Differences*, *53*(1), 50–54.

Young, S. G., Sacco, D. F., & Hugenberg, K. (2011). Vulnerability to disease is associated with a domain-specific preference for symmetrical faces relative to symmetrical non-face stimuli. *European Journal of Social Psychology*, *41*(5), 558–563.

Joshua Ackerman is an Associate Professor of Psychology at the University of Michigan. His research integrates principles of evolutionary psychology and nonconscious influence to study topics including threat processing and management, motivation, social cognition, sensation, and judgment and decision making.

Sarah Hill is an Associate Professor of Psychology at Texas Christian University. She applies the principles of evolutionary psychology to the study of human motivation, social behavior, and health.

Damian R. Murray is an assistant professor at Tulane University. He employs the principles of evolutionary biology and psychology in the study of human cognition and social behavior. Much of his work focuses upon the different ways in which infectious disease shapes human behavior and sociality.

How to cite this article: Ackerman JM, Hill SE, Murray DR. The behavioral immune system: Current concerns and future directions. *Soc Personal Psychol Compass*. 2018;12:e12371. https://doi.org/10.1111/spc3.12371