The Behavioral Immune System

Mark Schaller

University of British Columbia

(2016)
In D. M. Buss (Ed.), *The Handbook of Evolutionary Psychology* (2nd Edition, Vol. 1, pp. 206-224). New York: Wiley.

Many animals engage in many behaviors that reduce their exposure to pathogens. Ants line their nests with resins that inhibit the growth of fungi and bacteria (Chapuisat, Oppliger, Magliano, & Christe, 2008). Mice avoid mating with other mice that are infected with parasitic protozoa (Kavaliers & Colwell, 1995). Animals of many kinds—from physiologically primitive nematode worms to neurologically sophisticated chimpanzees—strategically avoid physical contact with specific things (including their own conspecifics) that, on the basis of superficial sensory cues, appear to pose some sort of infection risk (Goodall, 1986; Kiesecker, Skelly, Beard, & Preisser, 1999; Schulenburg & Müller, 2004).

Humans, too, engage in a wide variety of behaviors that provide prophylactic protection against pathogen infection. Some—such as condom use and vaccination—are modern artifacts of recently evolved neocortical processes that allow people to engage in rational decision-making of many kinds. Many other behaviors—some obvious and some not—appear to be governed by a suite of more ancient and functionally specific stimulus-response mechanisms that comprise a sort of "behavioral immune system" (Schaller, 2011; Schaller & Park, 2011). This chapter provides an overview of research on the behavioral immune system by highlighting, and elaborating upon, 12 key points. Collectively, these 12 "things you need to know" summarize the scope of this research, and provide a foundation for thinking critically about it.

The Behavioral Immune System Is Adapted to a Functionally Unique Fitness Problem

Before discussing what we know in regard to the *how* and the *what* of the behavioral immune system (how it operates and what its implications are for psychological phenomena), it is important to address the question of *why*: Why is it even plausible that there evolved a specific set of psychological mechanisms devoted to behavioral prophylaxis against infection?

The conceptual argument for the evolution of the behavioral immune system begins with an assumption that infectious diseases imposed selection pressures on ancestral populations. The veracity of this assumption is not in doubt. Pathogens have been a presence in human and prehuman ecologies for many millions of years (Ewald, 1995; Wolfe, Dunavon, & Diamond, 2007). Their presence has not been benign. It has been estimated that infectious diseases have been responsible for more human deaths than all other causes of death combined (Inhorn & Brown, 1990). Selection pressures imposed by pathogens are sufficiently strong that genetic mutations conferring resistance to specific diseases can become widespread with unusual

rapidity (Barreiro & Quintana-Murci, 2010). Over the course of human evolutionary history, these selection pressures resulted in many adaptations that are fundamental to human nature—including, most obviously, sophisticated immunological defenses.

If the existence of the immune system testifies to the selection pressures imposed by infectious diseases, it also raises an important issue that must be addressed when considering whether a *behavioral* immune system might also have evolved. Nothing evolves for free. No matter how intense the selection pressures associated with infectious diseases were, it is unlikely that a separate set of defense mechanisms would evolve unless these mechanisms were associated with a separate set of adaptive benefits. Is it plausible that, in addition to immunological defenses, there also evolved an additional set of psychological mechanisms facilitating *behavioral* defenses against infection?

Yes, and the reasons pertain to several shortcomings associated with immunological mechanisms. First, immunological responses are costly. For example: An immune response to bacterial infection typically involves some increase in body heat (local inflammation, systemic fever), and this response consumes substantial metabolic resources. (By one estimate, a 13% increase in metabolic activity is required to increase human body temperature by just 1^{o} C; Dantzer, Kent, Bluth, & Kelley, 1991.) Second, immunological responses can be temporarily debilitating. Many symptoms of infection, such as fever and fatigue, are not directly caused by the invading pathogen itself; they are consequences of the immune system's means for combating that infection. Many of these symptoms inhibit individuals' ability to engage in various other forms of fitness-enhancing activity (mating, parental care for offspring, etc.). Third, immunological defenses are merely reactive, occurring only *after* pathogenic intruders have already entered the body and begun to do their damage. Because of the costs and limitations associated with immunological defenses, there would have been unique adaptive benefits associated with *proactive* defense: the behavioral prevention of infection in the first place.

But even this further consideration provides insufficient rationale for the evolution of psychological mechanisms devoted specifically to the problem of disease avoidance. Again, nothing evolves for free. It is unlikely that any functionally unique behavioral immune system would have evolved if, within ancestral ecologies, equally effective prophylaxis was facilitated by other (less functionally specific) psychological mechanisms. Disease-causing pathogens might sensibly be characterized as "small predators" (Kurzban & Leary, 2001, p. 197), and so one must consider the possibility that behavioral avoidance of pathogens has been governed by evolutionarily ancient psychological mechanisms that protect against predation more generally (Barrett, 2005). This fear-based predation-avoidance system governs responses to a wide range of bodily threats (including some that objectively aren't even predators at all, such as forest fires and floods). Is it plausible that there evolved a set of psychological mechanisms designed *specifically* to promote behavioral defense against infection?

Yes, and the reason is this: Even though disease-causing pathogens might logically be small predators, they are *functionally* different from other predatory threats (Tybur, Lieberman, Kurzban, & DiScioli, 2013). These functional differences arise from the fact that pathogens are not merely small; they are vanishingly small—too tiny to be perceived. Most other threats to human health and welfare (including objectively predatory threats such as snarling beasts and men with weapons, as well as things like fires and floods) are sizeable enough to be appraised as threats on the basis of sensory cues indicating size, location, movement, and sometimes even intent. By contrast, the organisms that cause infectious diseases (e.g., bacteria, viruses,

helminths) are so tiny as to be imperceptible to human sensory systems. At best, their presence may sometimes be diagnosed only indirectly (e.g., the smell of organic matter that has been consumed by bacteria, or the appearance of a person suffering from infection). The imperceptibility of pathogens not only has unique implications for detection; it also has unique implications for behaviors that might mitigate their threat. Different species of pathogens are transmitted in different ways and, until very recently in human history, those modes of transmission remained outside the realm of human comprehension. (In fact, until the emergence of modern medicine and public health practices, infectious diseases were not just deeply mysterious but also uniquely diabolical: Many rewarding behaviors that served to fulfill the most basic biological needs—such as eating, drinking, and sexual intercourse—also increased the risk of exposure to disease-causing pathogens.) Behavioral strategies that were effective in mitigating exposure to other threats may have been useless, or worse, as protection against infectious diseases. For example, grouping behavior may provide protection against predatory attacks, but it facilitates transmission of many disease-causing parasites. The upshot is that psychological mechanisms facilitating adaptive behavioral responses to other forms of threat were unlikely to have provided effective prophylaxis against pathogens. There would have been unique adaptive benefits associated with an additional set of mechanisms that did, namely the behavioral immune system.

Its Activation Is Associated With Disgust

If the behavioral immune system evolved as a means of facilitating functionally specific behavioral responses, it might be considered to be a psychologically unique motivational system (Aunger & Curtis, 2013; Bernard, 2012; Neuberg, Kenrick, & Schaller, 2011). Distinct motivational systems are typically associated with characteristic affective experiences—thirst, hunger, fear, jealousy, and so on. The affective experience associated with the behavioral immune system is disgust.

It has been argued that disgust evolved from a more primitive affective response that served the function of expelling harmful foodstuffs—which may be contaminated with poisons as well as pathogens—from entering an organism's oral cavity (Rozin, Haidt, & McCauley, 2008). In contemporary human populations, disgust is elicited not just by the taste of contaminated food, but also by the perception (via the full range of sensory and inferential organs) of many different kinds of stimuli that, throughout long stretches of human evolutionary history, were diagnostic of the presence of pathogens. Among the more obvious, such elicitors are body products that typically contain pathogens (e.g., feces), animal vectors through which pathogens may be transmitted (e.g., rats), and physical symptoms exhibited by individuals who are already infected (e.g., the sight of oozing sores, the sound of a sneeze).

Affective responses to these kinds of stimuli lie at the heart of empirical evidence attesting to the integral role of disgust in the behavioral immune system (Curtis, DeBarra, & Aunger, 2011; Oaten, Stevenson, & Case, 2009). Three kinds of evidence are especially compelling. First, disgust is elicited more strongly by these stimuli than by perceptually similar stimuli that are less likely to connote infection risk. For example, people are more disgusted by a yellowish liquid—which mimics the appearance of body products such as pus—than by an otherwise identical liquid that is blue (Curtis, Aunger, & Rabie, 2004). Second, pathogen-connoting stimuli elicit

high levels of disgust but do not elicit high levels of other negative emotions, whereas functionally distinct forms of threat—such as predatory threats—elicit high levels of other negative emotions, such as fear, but do not elicit much disgust (Bradley, Codispotti, Sabatinelli, & Lang, 2001). These results implicate a functionally specific linkage between disgust and the threat posed by pathogen infection. Third, the tendency for pathogen-connoting stimuli to elicit disgust is exaggerated under circumstances in which the functional benefits of pathogen-avoidance are especially great, such as when individuals are especially vulnerable to infection. For example, immunological defenses are suppressed during the early stages of pregnancy (rendering the pregnant woman, and the developing fetus, more vulnerable to the fitness costs associated with pathogen infection). Coincident with this natural vulnerability, women in the early stages of pregnancy also exhibit stronger disgust responses to pathogen-connoting stimuli (Fessler, Eng, & Navarette, 2005).

Disgust is elicited not just by objects that overtly connote the immediate presence of pathogens; it is also elicited by specific forms of social behavior—including unusual sexual acts and actions of other kinds that violate moral codes of conduct (Haidt, McCauley, & Rozin, 1994; Tybur et al., 2013). Although there are additional functional considerations specific to the domains of mating and morality (Tybur et al., 2013), both sexual and moral behaviors also have implications for pathogen transmission. Sexual contact puts people at risk of infection, and so sexual behavior has historically been governed by cultural norms that (imperfectly) distinguish between ostensibly safe and unsafe sex acts. Consequently, as a result of cultural learning processes, sexual behaviors perceived to be non-normative within an individual's local cultural context may also come to be intuitively associated with increased infection risk. In fact, norm violations of all kinds may have this intuitive connotation. In preindustrial societies, "most conventions pertaining to subsistence and social behavior operate as prescriptions to avoid illness; almost all rules have health implications" (Fabrega, 1997, p. 36). Consequently, many transgressions against normative codes of conduct in many other behavioral domains may also come to be intuitively associated with increased infection risk. Thus, along with disgust responses to more obvious pathogen-connoting stimuli, many disgust responses in the sexual and moral domains may be indirect manifestations of the behavioral immune system.

It Influences Responses to Things That Pose No Real Threat of Infection

The behavioral immune system governs responses not only to perceptual objects and actions that are truly diagnostic of increased infection risk, but also to many objects and actions that, objectively, pose no risk at all. The reasons for this overgeneralization lie partially in the common tendency for evolved cognitive mechanisms to operate on an "actual domain" of stimuli that is broader than the "proper domain" of fitness-relevant perceptual input (Sperber & Hirschfeld, 2004). But there is more to it than just that. Overgeneralization also follows from the adaptive principles articulated within *error management theory* (Haselton & Buss, 2000; Haselton, Nettle, & Andrews, 2005). Because perceptual cues connoting potential infection risk are only imperfectly diagnostic of actual infection risk, the appraisal of risk must inevitably produce errors. Two kinds of errors are possible: False-positive errors (inferring infection risk when there is none), and false-negative errors (failing to infer infection risk when, in fact, some risk exists). Both types of error are equally erroneous in a strictly logical sense; but they have different behavioral consequences, and these different behavioral consequences are likely to have

had different implications for health and reproductive fitness within ancestral populations. Whereas false-positive errors would have resulted in (objectively unnecessary) avoidance of benign objects, false-negative errors would have resulted in (sometimes fatal) contact with infectious objects. The evolutionary consequence is that the appraisal mechanisms that trigger the behavioral immune system are calibrated to avoid highly costly false-negative errors. This adaptive cognitive bias inevitably produces many false-positive errors instead.

So, from the subjective perspective of the perceiver, infection risk may be connoted not just by the subset of body products that might actually be diagnostic of pathogen infection (e.g., feces, pus), but also by objectively benign body products (such as urine). Infection risk may be connoted not just by the actual physical symptoms that people exhibit when infected, but also by morphological or behavioral anomalies more generally, even those that are objectively unrelated to infection status. Infection risk may be implied not just by norm violations in behavioral domains most relevant to pathogen transmission (e.g., food, hygiene, mating) but also by norm violations more generally. These false positive errors may offer no immediate functional benefits (and may even be costly); but they are manifestations of an underlying cognitive bias that, within ancestral ecologies, evolved as an adaptive means of avoiding even more substantially costly errors.

Its Effects Are Flexible and Context-Contingent

Even functionally beneficial mechanisms also have costs. The development, and later deployment, of any bodily system consumes resources (which then cannot be expended on the development or deployment of other bodily systems). One means of adaptively managing these cost–benefit trade-offs manifests as developmental plasticity. During the course of an organism's development, genes produce different phenotypic outcomes depending on informational inputs from the local ecology, so that mechanisms that are more functionally beneficial within that ecology become more fully developed, at the expense of less immediately relevant mechanisms. Immunological defenses are characterized by developmental plasticity (Curno, Behnke, McElligott, Reader, & Barnard, 2009). Analogously, development of the behavioral immune system is likely to be responsive to informational inputs indicating the chronic risk of infection in the local ecology, and this may contribute to chronic differences among individuals.

This developmental process is just one relatively blunt instrument for solving the cost–benefit problem. Among primates and other big-brained animals, the cost–benefit problem is also substantially solved by neurocognitive and behavioral flexibility (van Schaik, 2013). The strength of any psychological response to a stimulus is variable from moment to moment, depending on additional information conveying the extent to which the functional benefits of the response might outweigh its costs within that particular circumstance. For example, cues connoting the presence of a predatory threat elicit greater fear and stronger activation of danger-connoting cognitions under conditions in which, on the basis of additional contextual information, perceivers feel more vulnerable to predation (Grillon, Pellowski, Merikangas, & Davis, 1997; Schaller, Park, & Mueller, 2003). The same principle applies to the behavioral immune system. Under conditions in which context-specific information makes a person's vulnerability to infection psychologically salient, people are more likely to appraise perceptually ambiguous stimuli as connoting an infection risk (Miller & Maner, 2012), and they are likely to

exhibit more pronounced affective, cognitive, and behavioral responses to those infection-connoting stimuli.

This *functional flexibility* principle (Schaller & Park, 2011) has informed much research on the cognitive and behavioral implications of the behavioral immune system. The utility of this principle lies not so much in the (intuitively obvious) observation that disease-avoidant psychological responses are likely to be variable across circumstances. Rather, the value lies in its specific implications for scientific inference and discovery. By employing research methods that experimentally manipulate the salience of individuals' vulnerability to infectious diseases, and that then measure some specific cognitive or behavioral outcome, one can test the extent to which there is a causal influence of perceived vulnerability on that outcome. If it is, and if that effect is *specific* to infection-vulnerability (compared to control conditions that make individuals feel vulnerable to equally dangerous but conceptually distinct forms of threat), it implies that the behavioral immune system has some psychologically *unique* influence on that outcome.

Dozens of psychological experiments have been conducted that apply these basic logical principles to a variety of psychological phenomena. The following four sections identify four broad domains of phenomena for which there is experimental evidence attesting to the unique implications of the behavioral immune system.

It Has Unique Implications for Interpersonal Attraction

Many infectious diseases manifest in symptoms that affect a person's facial appearance—making it less symmetrical, less prototypical. Given the adaptive tendency for risk-averse and overgeneralized appraisal, even subtle nonsymptomatic deviations from facial symmetry or prototypicality may be intuitively interpreted by perceivers as indicating some infection risk. This is likely to manifest in subjective judgments of attractiveness, which may help explain why faces that are less symmetrical and less prototypical are also judged to be less attractive (Rhodes, 2006). If so—if the subjective assessment of facial attractiveness reflects the underlying means of identifying, and avoiding, sources of infection—then the relative unattractiveness of asymmetrical and nonprototypical faces may be exaggerated when perceivers temporarily feel more vulnerable to infection.

Exactly this effect was reported by Young, Sacco, and Hugenberg (2011). They experimentally manipulated whether the risk of pathogen infection was temporarily salient or (in a control condition) whether a different kind of threat was salient. They then assessed subjective preferences for faces varying in bilateral symmetry, as well as preferences for other (nonsocial) objects that also varied in symmetry. Results revealed that the typical preference for symmetrical faces was exaggerated when the threat of infection was salient. Preference for symmetrical objects of other kinds was not exaggerated. Thus, not only was the effect specific to circumstances that made perceivers feel vulnerable to infection, it was also specific to the perception of faces.

The results of Young et al. (2011) were obtained on judgments of both same-sex and opposite-sex faces. Other research using similar methods indicates that the effect may be especially pronounced in perceptions of *opposite-sex* faces (Little, DeBruine, & Jones, 2011). This result implies that the exaggerated preference for symmetrical faces (and perhaps for more subjectively attractive faces in general) is especially likely to occur in mating contexts. There are many

possible reasons (Tybur & Gangestad, 2011). Some follow straightforwardly from the functional logic of behavioral prophylaxis against infection: Because sexual behavior puts individuals in especially intimate (and sometimes especially enduring) physical contact with other individuals, the mating domain is one in which individuals may be especially vigilant for cues indicating possible infection, and may be especially discriminating in their responses when any such cues are perceived. Other reasons go beyond the simple avoidance of sexual contact, and pertain to the genetic fitness of offspring that might result from sexual contact. For example, bilateral symmetry and other subjectively attractive features may not only be diagnostic of an individuals' current health, they may also be diagnostic of the extent to which that individuals' genes provide a basis for effective immunological defenses against infection—genes that are likely to be passed on to offspring who, in turn, are likely to have better immunological defenses and to be more reproductively fit themselves.

Even if the behavioral immune system does have implications for attraction that manifest especially strongly in mating contexts, its implications for attraction are not limited just to mating contexts. Several experiments show that, compared to conditions in which people feel vulnerable to other forms of threat, when people temporarily feel vulnerable to infection, they express stronger preferences for physically attractive political candidates and more highly prioritize physical attractiveness when selecting group leaders (White, Kenrick, & Neuberg, 2013). The latter effect was specific to the selection of *leaders*, and did not emerge on preferences for group members more generally.

Overall, it appears that activation of the behavioral immune system has unique consequences for the subjective appeal of attractive people, and that these consequences may occur especially strongly within behavioral domains (such as mate choice and leader selection) that have especially important implications for individuals' own immediate or long-term outcomes.

It Has Unique Implications for Stigma and Prejudice

Much evidence implicates the behavioral immune system in the stigmatization of, and prejudice against, different categories of people (Kurzban & Leary, 2001; Oaten, Stevenson, & Case, 2011; Schaller & Neuberg, 2012). The most obvious implications are for prejudices toward people who actually are suffering from infectious diseases. But perhaps the most striking implications—which follow from the principles of error-management theory (discussed earlier)—are found in prejudicial responses to people who, objectively, pose no infection risk at all.

Among these targets of prejudice are people whose appearance is characterized by superficial morphological anomalies, such as facial birthmarks (Ryan, Oaten, Stevenson, & Case, 2012). Results from one study revealed that the semantic concept "disease" was implicitly activated into working memory by the perception of a facially disfigured man even when perceivers *knew* that the disfigurement was merely a superficial birthmark and that the man was healthy (Schaller & Duncan, 2007). In fact, "disease" was implicitly associated more strongly with the superficially disfigured man than it was with a man who was known to suffer from an infectious disease but who appeared superficially normal.

These implicit prejudicial responses are elicited by the perceptions of people who appear morphologically anomalous in other ways too. Consistent with the logic of functional flexibility, these prejudices emerge most strongly under conditions in which perceivers feel more vulnerable

to infection. Compared to control conditions in which other threats are salient, when the threat posed by infectious disease is temporarily salient, people exhibit stronger implicit prejudices against people who are physically disabled, elderly, or obese (Duncan & Schaller, 2009; Park, Faulkner, & Schaller, 2003; Park, Schaller, & Crandall, 2007). Prejudice against obese people is particularly revealing. Obesity was unlikely to have been prevalent in the ancestral populations in which the behavioral immune system evolved; even in contemporary ecologies, obesity is unlikely to be objectively diagnostic of pathogen infection (if anything, infection is more likely to cause weight loss than weight gain). The findings, therefore, highlight the logical implications of error management theory (Haselton et al., 2005): The behavioral immune system can be tricked by novel and objectively irrelevant stimuli, and it produces prejudice accordingly.

The behavioral immune system appears also to have implications for xenophobia. There are many conceptually distinct psychological causes of xenophobia, some of which are linked to other threats implicitly associated with members of coalitional outgroups (including threats to economic resources and the threat of interpersonal violence; e.g., Esses, Dovidio, Jackson, & Armstrong, 2002; Schaller et al., 2003). In addition to those other threats, there are several reasons why outgroup members—especially those who are perceived subjectively to be "foreign"—might also be intuitively associated with infection risk. Some foreign peoples have physical appearances that may be subjectively appraised as anomalous, and so may trigger the behavioral immune system for the same reason that obese people do. A second possibility lies in the potential for exotic peoples to be sources of exotic pathogens (which may be especially virulent when introduced into local populations). A third reason is perhaps less obvious but also of potentially greater evolutionary importance: Outsiders may be ignorant of, or less personally invested in complying with, local cultural norms; and so are more likely to violate those norms. Because many local norms historically served as barriers to pathogen transmission, the presence of outsiders within one's local community may have increased the infection risk to everyone within that community. The implication is that when people are subjectively perceived to be foreigners, they are likely to elicit the psychologically unique form of prejudice associated with the behavioral immune system. This is especially likely when perceivers are—or simply perceive themselves to be—especially vulnerable to infection. Consistent with this hypothesis, women in their first trimester of pregnancy (whose immunological defenses are temporarily suppressed) exhibit exaggerated ethnocentrism and xenophobia (Navarette, Fessler, & Eng, 2007). Also, compared to control conditions in which other threats are salient, when the threat of infection is temporarily salient, people exhibit more exaggerated prejudice against immigrants from subjectively foreign places (Faulkner, Schaller, Park, & Duncan, 2004).

These finding not only have unique conceptual implications, they also have practical implications for the design of intervention strategies that might reduce prejudices (Schaller & Neuberg, 2012). One set of experiments reveals that, among people who chronically perceive themselves to be vulnerable to pathogen infection, prejudice can be reduced by interventions that specifically target these perceptions of vulnerability and bolster feelings of immunity or invulnerability instead (Huang, Sedlovskaya, Ackerman, & Bargh, 2011).

It Has Unique Implications for Conformity and Reactions to Nonconformity

If cultural rituals and traditions and norms historically helped to inhibit pathogen transmission (Fabrega, 1997), it follows that activation of the behavioral immune system may lead people to be especially observant of norms, and to respond especially harshly to norm violations. These effects may manifest in a variety of specific psychological phenomena, including conformity, political conservatism, and moral judgment.

Effects on conformity are documented by recent experiments showing consistent findings across multiple measures, and across multiple cultural contexts: Compared to control conditions in which other forms of threat are salient, when people feel temporarily vulnerable to the threat posed by infectious diseases, they express greater liking for people with conformist traits, endorse more conformist attitudes, and are more likely to behaviorally conform with majority opinion (Murray & Schaller, 2012; Wu & Chang, 2012). Thus, while other self-protective motives may also lead to increased conformity (Griskevicius, Goldstein, Mortensen, Cialdini, & Kenrick, 2006), the behavioral immune system appears to have implications for conformity that are uniquely powerful.

The same rationale implies further implications for conservative political attitudes (which are characterized by attitudinal defense of long-standing cultural traditions, and by intolerant responses to individuals who deviate from those traditions). Many studies, using both correlational and experimental methods and employing both indirect and direct indicators of conservatism, indicate that when the behavioral immune system is activated more strongly, people are more conservative (Terrizzi, Shook, & McDaniel, 2013). In one illustrative experiment, Helzer and Pizarro (2011) asked people to complete a measure of political attitudes in the hallway of a public building. In one condition they did so while standing next to an anti-bacterial hand-sanitizer dispenser—a perceptual cue that makes the threat of infection temporarily salient. In that condition (compared to a control condition), people endorsed more politically conservative attitudes.

This conceptual framework may help explain why disgust has a carry-over effect on moral judgments (e.g., Erskine, Kacinik, & Prinz, 2011; Horberg, Oveis, Keltner, & Cohen, 2009; Schnall, Haidt, Clore, & Jordan, 2008; Wheatley & Haidt, 2005). Given that the appraisal of infection risk often triggers disgust, the emotional experience of disgust may serve as a signal indicating vulnerability of pathogen infection. Consequently, people are more likely to morally condemn actions that violate cultural codes of conduct and other social norms.

It May Have Implications for Behavioral Dispositions More Broadly

Several studies suggest that the behavioral immune system may influence general behavioral tendencies of the sort typically measured as personality traits. For example, in the domain of sexual behavior, people differ in their dispositional tendency toward restricted versus unrestricted (e.g., promiscuous) mating strategies (Jackson & Kirkpatrick, 2007; Simpson & Gangestad, 1991). Results from one study revealed that women (but not men) who perceived themselves to be more chronically vulnerable to infection reported more restricted mating dispositions, and these effects emerged primarily when the threat of infectious diseases was temporarily salient (Murray, Jones, & Schaller, 2013). Why was this effect specific to women? It

may reflect the historically adaptive tendency for women, more than men, to be risk-averse in the mating domain (Haselton & Buss, 2000). Consequently, women may be especially sensitive to the costs associated with unrestricted mating strategies—including the increased risk of contracting infectious diseases.

For both men and women, social interactions of any kind may facilitate pathogen transmission. Consequently, people who are more socially gregarious may be more susceptible to infection (Nettle, 2005). Activation of the behavioral immune system might, therefore, be expected to inhibit socially gregarious dispositional tendencies. There is some support for this hypothesis: When the threat of infection was made temporarily salient, people expressed reduced dispositional tendencies toward extraversion and agreeableness; and these effects emerged most strongly among people who chronically felt most vulnerable to infection (Mortensen, Becker, Ackerman, Neuberg, & Kenrick, 2010).

Even beyond the domain of social interaction, any kind of approach-oriented, risk-tolerant, or exploratory behavioral style may increase individuals' risk of coming into contact with pathogens within their natural environment—and so may be inhibited when the behavioral immune system is activated. Some support for this hypothesis is found in additional results reported by Mortensen et al. (2010). When the threat of infection was temporarily salient, people reported lower levels on the trait "openness to experience," and this effect, too, occurred primarily among people who chronically felt most vulnerable to infection.

Compared to the other lines of work reviewed earlier, these findings on dispositional traits probably need to be interpreted more cautiously. The experimental design employed by Mortensen et al. (2010) did not include a control condition in which other kinds of threat were made salient, and so it is difficult to confidently conclude that the effects were unique to the perceived threat of infection. These results, along with those of Murray et al. (2013), are also complicated by interactions between experimental manipulations and chronic individual differences. Still, bearing these caveats in mind, the results do provide preliminary evidence that the behavioral immune system may lead to risk-averse behavioral dispositions in general.

It May Help to Explain Cross-Cultural Differences

The functionally flexible psychological mechanisms that define the behavioral immune system have implications that manifest most immediately in individuals' feelings, cognitions, and actions. Hence, the most inferentially relevant research focuses on individual-level psychological outcomes. But these individual-level psychological phenomena may have further implications that manifest at the level of entire human populations. If the behavioral immune system is activated especially frequently or especially strongly among individuals who comprise a particular population, then those individuals are likely, on average, to exhibit somewhat different psychological tendencies compared to individuals who comprise a different population. What might lead to population-level variability in the extent to which the behavioral immune system is activated? Ecological variability in the actual prevalence of disease-causing pathogens. This implies that worldwide cross-cultural differences may be partially attributable to ecological differences in pathogen prevalence.

Building on pioneering research on the population-level correlates of pathogen prevalence (Gangestand & Buss, 1993; Low, 1990), there is now a substantial body of evidence

documenting relations between pathogen prevalence and the psychological profiles of different populations worldwide. Most of this work focuses on cross-national comparisons, and many of the results conceptually mimic findings from the psychological experiments reviewed earlier. In countries characterized by higher levels of pathogen prevalence, the people inhabiting those countries place a higher value on physical attractiveness, and exhibit more xenophobic attitudes toward ethnic outgroups (Gangestad, Haselton, & Buss, 2006; Schaller & Murray, 2010). They also conform more strongly to majority opinion, exert stronger conformity pressures on others, express more highly authoritarian attitudes, more strongly endorse moral values pertaining to group loyalty and purity, and more strongly endorse collectivistic cultural values (Fincher, Thornhill, Murray, & Schaller, 2008; Murray, Schaller, & Suedfeld, 2013; Murray, Trudeau, & Schaller, 2011; Van Leeuwen, Park, Koenig, & Graham, 2012). On measures of personality traits, they have lower scores on both extraversion and openness to experience, and women within those countries report more restricted mating strategies (Schaller & Murray, 2008).

Ecological variation in pathogen prevalence also predicts additional societal outcomes that may be emergent consequences of individual-level attitudes and actions. In places where pathogens are more highly prevalent, there is more frequent ethnic conflict, governments are more authoritarian in their policies, and there are lower levels of scientific and technological innovation (Letendre, Fincher, & Thornhill, 2010; Murray, 2014; Thornhill, Fincher, & Aran, 2009). These and other conceptually related findings (e.g., Fincher & Thornhill, 2012) suggest that the functionally flexible implications of the behavioral immune system may help explain many worldwide cross-cultural differences.

The findings are provocative; but because of inferential limitations that accompany the underlying methods, one must be cautious in drawing conclusions (Pollet, Tybur, Frankenhuis, & Rickard, 2014; Schaller & Murray, 2011). These methods are necessarily correlational, and pathogen prevalence naturally correlates with other variables—including other threats to human welfare as well as societal structures that mitigate those threats—that may have conceptually independent implications for cultural norms (e.g., Hruschka & Henrich, 2013; Van de Vliert, 2013). The most inferentially compelling results are those in which pathogen prevalence remains a unique predictor of cultural differences even when statistically controlling for such variables. Some of the cross-national findings do meet this stricter standard of evidence. For instance, even when controlling for plausible demographic and economic confounds and for other threats to human life, ecological variation in pathogen prevalence still predicts cross-cultural variation in extraversion, openness, conformity, and collectivism (Fincher et al., 2008; Murray et al., 2011; Schaller & Murray, 2008).

Another thorny inferential issue arises within any analysis that treats contemporary geopolitical entities as units of analyses. Countries are not conceptually equivalent to cultures; the societal structures and popular norms observed in one country are rarely independent of those in other countries (Nettle, 2009). Relations between pathogen prevalence and cultural differences are more compelling when observed not only in analyses of contemporary geopolitical entities but also in analyses of small-scale societies that more closely approximate distinct cultural entities. Several results provide this sort of replication, and further support hypotheses linking pathogen prevalence to cultural differences in collectivistic attitudes and authoritarian governance (Cashdan & Steele, 2013; Murray, Schaller, & Suedfeld, 2013).

Finally, even if pathogen prevalence does play some unique role in creating cultural differences, there are multiple conceptually distinct explanatory processes through which this effect might

occur. The functionally flexible individual-level psychological mechanisms that govern individuals' cognitions and actions offer one plausible explanation. But other conceptually distinct processes may contribute as well. These include developmental processes that manifest as phenotypic plasticity, as well as additional population-level processes (including genetic evolution) that transcend a psychological level of analysis entirely. (For a more detailed discussion of these different processes, see Schaller & Murray, 2011).

It Has Both Obvious and Nonobvious Health Implications

Because of its implications for the reduction of infection risk, the behavioral immune system almost certainly had positive implications for human health through much of human evolutionary history. It is likely that some of these same health benefits continue in contemporary human contexts too. These infection-buffering benefits may be more fully realized by interventions that target the psychological mechanisms that characterize the system. Education-based interventions that focus on rational decision-making processes often prove to be only modestly successful means of changing prophylactic behavior; in contrast, interventions that activate the behavioral immune system may be more effective in promoting safe-sex practices, hand-washing, and other infection-reducing behaviors (Porzig-Drummond, Stevenson, Case, & Oaten, 2009; Tybur, Bryan, Magnan, & Caldwell Hooper, 2011).

The individual-level manifestations of the behavioral immune system may have population-wide epidemiological implications. The speed and scope of epidemic outbreaks within a population are influenced by the behavioral tendencies of individuals within that population—tendencies regarding sexual promiscuity, social gregariousness, conformity to hygiene-relevant norms, and so on. One interesting implication is that, because different contemporary cultural populations differ in these behavioral dispositions, the dynamics of disease epidemics may also differ predictably within different cultures.

Ironically, some behavioral consequences of the system may, indirectly, have *negative* health implications too (perhaps especially in modern societies characterized by long life expectancies). If activation of the behavioral immune system inhibits extraversion, as some evidence suggests, then chronic activation may inhibit the development and maintenance of social relationships. This may put people at greater risk of loneliness and insufficient social support, which are associated with poorer long-term health outcomes (Cacioppo, Hawkley, & Berntson, 2003; Cohen, 2004). Under circumstances in which infectious diseases pose a substantial threat of health outcomes, these long-term costs—if they occur—are likely to be outweighed by the health benefits conferred by reduced infection risk. But, in modern societies in which the health threat posed by infectious diseases is minimal, or is effectively managed by technological innovations, any such long-term costs may represent a more troubling consequence.

There may also be implications for some psychopathologies. One type of obsessive compulsive disorder, as well as other specific phobias, may result in part from abnormal hyperactivity in appraisal or response mechanisms associated with the behavioral immune system (Cisler, Olatunji, & Lohr, 2009; Marks & Nesse, 1994). If so, research on these mechanisms may help illuminate the etiology of these psychopathologies, and perhaps have practical implications for effective treatment.

One additional implication is especially provocative: The perceptual mechanisms through which people appraise infection risk (and which facilitate behavioral prophylaxis against infection) may also affect actual immunological responses. Results from recent experiments show that exposure to disgust-eliciting stimuli in turn stimulates oral immune function (Stevenson, Hodgson, Oaten, Barouei, & Case, 2011; Stevenson et al., 2012). Of course, immunological responses can be affected by stressful psychological experiences of many different kinds; so it is important to test whether any such effects are *unique* to the perceptual appraisal of infection risk. Results from one experiment did so (Schaller, Miller, Gervais, Yeager, & Chen, 2010). Participants were exposed to visual images connoting the risk of either pathogen infection or (in a control condition) interpersonal violence, and measures were taken of their white blood cells' production of a pro-inflammatory cytokines in response to a bacterial stimulus. Results revealed that, even in comparison to the control condition, the perception of infection risk was associated with a more aggressive immune response.

There Is Still a Lot That We Do *Not* Know

There is a lot that we do not yet know about the behavioral immune system. It may be worthwhile, for instance, to explore more fully the extent to which the behavioral immune system contributes to the development, and content, of individuals' intuitive theories about contagion (Rozin, Millman, & Nemeroff, 1986). Another potentially interesting connection is to the literature on self-medication. Many animal species—including humans and other primates—strategically ingest nonnutritional botanical substances that aid in antipathogen defense (Huffman, 2003). Although most of this research focuses on the benefits of self-medication for controlling infections that have already occurred, some forms of self-medication may serve a prophylactic function too. Other potential implications may also arise from close examination of other animal species. For instance, chimpanzees have been observed to act aggressively toward diseased conspecifics (Goodall, 1986). This may seem counterintuitive and maladaptive, given that acts of aggression typically involve close interpersonal contact. And yet, if aggression isolates (or kills) the victim, then its short-term risks may be outweighed by long-term fitness benefits—realized not only by the aggressor but by others too.

It will also be important to examine more closely the interrelations between affective, cognitive, and behavioral responses that characterize the behavioral immune system. The study of specific cognitive and behavioral responses has often proceeded independently of research on disgust, and vice versa. It remains unclear what the exact role of disgust is in producing the various cognitive and behavioral manifestations of the system, or if some of these additional manifestations occur even in the absence of a disgust response. It seems likely that some level of disgust accompanies certain kinds of cognitive and/or behavioral responses (e.g., xenophobic responses to foreigners), but disgust may be a mere concomitant of these responses rather than a necessary causal antecedent. In contrast, other prophylactic behaviors—such as conformity and moral condemnation of nonconformity—may be unaccompanied by the immediate arousal disgust; but this does not mean that disgust has no causal implications for these behaviors. In fact, because of its effects on memory, attitude formation, and interpersonal communication, the experience of disgust at any one moment in time may have important causal consequences for the effective long-term deployment of these behavioral strategies (Schaller, 2014).

There is also much to be learned about the *appraisal* of infection risk. Whereas lots of research has examined responses to stimuli that have been intuitively appraised as connoting an infection risk, less research has examined the appraisal process itself. Many inferential inputs that trigger the behavioral immune system may be outputs of an appraisal system that evolved separately and that operates on a wider domain of sensory signals (Woody & Szechtman, 2011). But there may also be unique appraisal mechanisms that evolved to serve the specific function of identifying pathogen-connoting cues. If so, such mechanisms may operate within specific sensory modalities, such as olfaction (Kavaliers, Choleris, & Pfaff, 2005; Olsson et al., 2014).

Finally, it will be useful to know the biological substrates of the behavioral immune system. Research on the physiological correlates of emotional experiences reveals that disgust—aroused in response to pathogen-connoting stimuli—is associated with unique patterns of autonomic nervous system activity and neurological activity (Kreibig, 2010; Vytal & Hamann, 2010). But not much is known about the functional connections between anatomical structures, neurochemical processes, and the various cognitive and behavioral phenomena that are manifestations of the behavioral immune system. Even less is known about *genetic* substrates. Research has begun to document genetic correlates of disgust sensitivity and other potentially relevant variables (e.g., Kang, Kim, Namkoong, & An, 2010; Kavaliers et al., 2005; MacMurray, Comings, & Napolioni, 2014), but we still know next to nothing about the genetic bases of the system. It is not necessary to have this knowledge in order to make discoveries about psychological phenomena. But in order to most firmly locate these phenomena within the context of human evolution, it will be useful to know more about the specific bits of genetic information that (within ancestral populations) evolved in response to the unique selection pressures imposed by infectious diseases and that (within individual organisms) construct the unique elements of human physiology that facilitate the behavioral avoidance of infection.

It Is an Evolutionary Psychology Success Story

Research on the behavioral immune system provides a prototypic example of the scientific benefits that can accrue from the application of an evolutionary approach to psychological questions. Most psychological scientists are compelled, by either internal inclinations or external incentives, to study topics that matter in the here and now. And for most psychological scientists, the "here and now" includes obvious cognitive and technological innovations that provide modern solutions to pathogen transmission. This makes it easy to disregard the historically potent problem posed by infectious diseases, to overlook the behavioral means through which the problem might plausibly have been solved in ancient ecologies, and to be blinded to the enduring implications for psychological phenomena. Specialized tools are sometimes necessary to transcend this kind of scientific myopia. The logical principles of evolutionary psychology provide such tools.

Despite the long history of inquiry into human motivation, it is only more recently that behavioral scientists, explicitly informed by the logical principles of evolutionary psychology, have identified a psychologically unique motivational system facilitating behavioral avoidance of pathogen infection (Aunger & Curtis, 2013; Bernard, 2012; Neuberg, et al., 2011). Similarly, psychologists have been studying xenophobia and conformity and interpersonal attraction for decades and decades and decades; but it is only in the past few years—aided by the toolkit of

evolutionary psychology—that the motivational psychology of disease avoidance has been implicated as an important influence on these and other psychological phenomena.

Evolutionary psychology not only provides a logical basis for deducing that a behavioral immune system is likely to exist, but also additional logical tools that help to articulate how it works and what the specific consequences might be. Especially useful are evolutionary cost/benefit analyses (in which costs and benefits of psychological responses are defined by their repercussions for reproductive fitness). These cost/benefit analyses provide the logical basis for deductive principles regarding the stimuli to which the behavioral immune system responds, and the contexts within which those responses are especially likely or unlikely to occur. These logical principles have yielded dozens of hypotheses, which have been tested by empirical data, and have produced many novel discoveries. It would be wrong to assert that these conceptual insights and empirical findings *could not* have occurred in the absence of the analytic tools associated with evolutionary psychology. But it is not wrong to observe that, for the most part, they *did not*. If the scientific value of any meta-theoretical perspective is measured by its demonstrated utility in generating new hypotheses and empirical discoveries, then research on the behavioral immune system testifies convincingly to the success of evolutionary psychology.

Acknowledgments

Preparation of this manuscript was supported by a grant from the Social Sciences and Humanities Research Council of Canada.

References

Aunger, R., & Curtis, V. (2013). The anatomy of motivation: An evolutionary ecological approach. *Biological Theory, 8,* 49–63.

Barreiro, L. B., & Quintana-Murci, L. (2010). From evolutionary genetics to human immunology: How selection shapes host defence genes. *Nature Reviews Genetics, 11,* 17–30.

Barrett, H. C. (2005). Adaptations to predators and prey. In D. M. Buss (Ed.), *The handbook of evolutionary psychology* (pp. 200–223). Hoboken NJ: Wiley.

Bernard, L. C. (2012). Evolved individual differences in human motivation. In R. M. Ryan (Ed.), *The Oxford handbook of human motivation* (pp. 381–407). Oxford, England: Oxford University Press.

Bradley, M. M., Codispotti, M., Sabatinelli, D., & Lang, P. J. (2001). Emotion and motivation II: Sex differences in picture processing. *Emotion, 1,* 300–319.

Cacioppo, J. T., Hawkley, L. C., & Berntson, G. G. (2003). The anatomy of loneliness. *Current Directions in Psychological Science, 12,* 71–74.

Cashdan, E, & Steele, M. (2013). Pathogen prevalence, group bias, and collectivism in the Standard Cross-Cultural Sample. *Human Nature, 24,* 59–75.

Chapuisat, M., Oppliger, A., Magliano, P. & Christe, P. (2007). Wood ants use resin to protect themselves against pathogens. *Proceedings of the Royal Society B, 274,* 2013–2017.

Cisler, J. M., Olatunji, B. O., & Lohr, J. M. (2009). Disgust, fear, and the anxiety disorders: A critical review. *Clinical Psychology Review, 29,* 34–46.

Cohen, S. (2004). Social relationships and health. *American Psychologist, 59,* 676–684.

Curno, O., Behnke, J. M., McElligott, A. G., Reader, T., & Barnard, C. J. (2009). Mothers produce less aggressive sons with altered immunity when there is a threat of disease during pregnancy. *Proceedings of the Royal Society B, 276,* 1047–1054.

Curtis, V., Aunger, R., & Rabie, T. (2004). Evidence that disgust evolved to protect from risk of disease. *Proceedings of the Royal Society B, 271,* S131–S133.

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

Dantzer, R. Kent, S., Bluthe, R. M., & Kelley, K. W. (1991). Cytokines and sickness behaviour. *European Neuropsychopharmacology, 1,* 377–379.

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,* 97–115.

Erskine, K. J., Kacinik, N. A., & Prinz, J. J. (2011). A bad taste in the mouth: Gustatory disgust influences moral judgment. *Psychological Science, 22,* 295–299.

Esses, V. M., Dovidio, J. F., Jackson, L. M., & Armstrong, T. L. (2002). The immigration dilemma: The role of perceived group competition, ethnic prejudice, and national identity. *Journal of Social Issues, 57,* 389–412.

Ewald, P. W. (1993). *Evolution of infectious disease*. New York, NY: Oxford University Press.

Fabrega, H. (1997). Earliest phases in the evolution of sickness and healing. *Medical Anthropology Quarterly, 11,* 26–55.

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

Fessler, D. M. T., Eng, S. J. & Navarrete, C. D. (2005). Elevated disgust sensitivity in the first trimester of pregnancy: Evidence supporting the compensatory prophylaxis hypothesis. *Evolution and Human Behavior, 26,* 344–351.

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,* 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,* 1279–1285.

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

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

Goodall, J. (1986). Social rejection, exclusion, and shunning among the Gombe chimpanzees. *Ethology and Sociobiology, 7,* 227–239.

Grillon, C., Pellowski, M., Merikangas, K. R., & Davis, M. (1997). Darkness facilitates acoustic startle reflex in humans. *Biological Psychiatry, 42,* 453–460.

Griskevicius, V., Goldstein, N. J., Mortensen, C. R., Cialdini, R. B., & Kenrick, D. T. (2006). Going along versus going alone: When fundamental motives facilitate strategic nonconformity. *Journal of Personality and Social Psychology, 91,* 281–294.

Haidt, J., McCauley, C., & Rozin, P. (1994). Individual differences in sensitivity to disgust: A scale sampling seven domains of disgust elicitors. *Personality and Individual Differences, 5,* 701–713.

Haselton, M. G., & Buss, D. M. (2000). Error management theory: A new perspective on biases in cross-sex mind reading. *Journal of Personality and Social Psychology, 78,* 81–91.

Haselton, M. G., Nettle, D., & Andrews, P. W. (2005). The evolution of cognitive bias. In D. M. Buss (Ed.), *The handbook of evolutionary psychology* (pp. 724–746). Hoboken, NJ: Wiley.

Helzer, E. G., & Pizarro, D. A. (2011). Dirty liberals! Reminders of physical cleanliness influence moral and political attitudes. *Psychological Science, 22,* 517–522.

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

Hruschka, D. J., & Henrich, J. (2013). Institutions, parasites and the persistence of in-group preferences. *PLoS ONE 8*(5), e63642.

Huang, J. Y., Sedlovskaya, A., Ackerman, J. M., & Bargh, J. A. (2011). Immunizing against prejudice: Effects of disease protection on outgroup attitudes. *Psychological Science, 22,* 1550–1556.

Huffman, M. A. (2003). Animal self-medication and ethno-medicine: Exploration and exploitation of the medicinal properties of plants. *Proceedings of the Nutrition Society, 62,* 371–381.

Inhorn, M. C., & Brown, P. J. (1990). The anthropology of infectious disease. *Annual Review of Anthropology, 19,* 89–117.

Jackson, J. J., & Kirkpatrick, L. A. (2007). The structure and measurement of human mating strategies: Toward a multidimensional model of sociosexuality. *Evolution and Human Behavior, 28,* 382–391.

Kang, J. I., Kim, S. J., Namkoong, K., & An, S. K. (2010). Assocation of DRD4 and COMT polymorphisms with disgust sensitivity in healthy volunteers. *Neurophysiology, 61,* 105–112.

Kavaliers, M. & Colwell, D. D. (1995). Discrimination by female mice between the odours of parasitized and non-parasitized males. *Proceedings of the Royal Society B*, *261,* 31–35.

Kavaliers, M., Choleris, E., & Pfaff, D.W. (2005). Recognition and avoidance of the odors of parasitized conspecifics and predators: differential genomic correlates. *Neuroscience and Biobehavioral Reviews 29,* 1347–1359.

Kiesecker, J. M., Skelly, D. K., Beard, K. H., & Preisser, E. (1999). Behavioral reduction of infection risk. *Proceedings of the National Academy of Sciences, USA, 96,* 9165–9168.

Kreibig, S. D. (2010). Autonomic nervous system activity in emotion: A review. *Biological Psychology, 34,* 394–421.

Kurzban, R., & Leary, M. R. (2001). Evolutionary origins of stigmatization: The functions of social exclusion. *Psychological Bulletin, 127,* 187–208.

Letendre, K., Fincher, C. L., & Thornhill, R. (2010). Does infectious disease cause global variation in the frequency of intrastate armed conflict and civil war? *Biological Reviews, 85,* 669–683.

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, 278,* 2032–2039.

Low, B. S. (1990). Marriage systems and pathogen stress in human societies. *American Zoologist, 30,* 325–339.

MacMurray, J., Comings, D. E., & Napolioni, V. (2014). The gene-immune-behavioral pathway: Gamma-interferon (IFN-γ) simultaneously coordinates susceptibility to infectious disease and harm-avoidance behaviors. *Brain, Behavior, and Immunity, 35,* 169–175.

Marks, I. M., & Nesse, R. M. (1994). Fear and fitness: An evolutionary analysis of anxiety disorders. *Ethology and Sociobiology, 15,* 247–261.

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*, 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,* 440–447.

Murray, D. R. (2014). Direct and indirect implications of pathogen prevalence for scientific and technological innovation. *Journal of Cross-Cultural Psychology, 45,* 971–985.

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

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,* 180–188.

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,* 318–329.

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

Nettle, D. (2005). An evolutionary approach to the extraversion continuum. *Evolution and Human Behavior, 26,* 363–373.

Nettle, D. (2009). Ecological influences on human behavioral diversity: A review of recent findings. *Trends in Ecology and Evolution, 24,* 618–624.

Neuberg, S. L., Kenrick, D. T., & Schaller, M. (2011). Human threat management systems: Self-protection and disease avoidance. *Neuroscience and Biobehavioral Reviews, 35,* 1042–1051.

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

Oaten, M., Stevenson, R. J., & Case, T. I. (2011). Disease avoidance as a functional basis for stigmatization. *Philosophical Transactions of the Royal Society B, 366,* 3433–3452.

Olsson, M. J., Lundström, J. N., Kimball, B. A., Gordon, A. R., Karshikoff, B., Hosseini, N., . . . Lekander, M. (2014). The scent of disease: Human body odor contains an early chemosensory cue of sickness. *Psychological Science, 25,* 817–823.

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,* 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,* 410–414.

Pollet, T. V., Tybur, J. M., Frankenhuis, W. E., & Rickard, I. J. (2014). What can cross-cultural correlations teach us about human nature? *Human Nature, 25*(3), 410–429.

Porzig-Drummond, R., Stevenson, R., Case, T., & Oaten, M. (2009). Can the emotion of disgust be harnessed to promote hand hygiene? Experimental and field-based tests. *Social Science and Medicine, 68,* 1006–1012.

Rhodes, G. (2006). The evolutionary psychology of facial beauty. *Annual Review of Psychology*, *57,* 199–226.

Rozin, P., Haidt, J., & McCauley, C. R. (2008). Disgust. In M. Lewis, J. M. Haviland-Jones, & L. F. Barrett (Eds), *Handbook of emotions* (3rd ed., pp. 757–776). New York, NY: Guilford Press.

Rozin, P., Millman, L., & Nemeroff, C. (1986). Operation of the laws of sympathetic magic in disgust and other domains. *Journal of Personality and Social Psychology, 50,* 703–712.

Ryan, S., Oaten, M., Stevenson, R. J., & Case, T. I. (2012). Facial disfigurement is treated like an infectious disease. *Evolution and Human Behavior, 33,* 639–646.

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

Schaller, M. (2014). When and how disgust is and is not implicated in the behavioral immune system. *Evolutionary Behavioral Sciences, 8*, 251–256.

Schaller, M., & Duncan, L. A. (2007). The behavioral immune system: Its evolution and social psychological implications. In J. P. Forgas, M. G. Haselton, & W. von Hippel (Eds.), *Evolution and the social mind: Evolutionary psychology and social cognition* (pp. 293–307). New York, NY: Psychology Press.

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,* 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,* 212–221.

Schaller, M., & Murray, D. R. (2010). Infectious diseases and the evolution of cross-cultural differences. In M. Schaller, A. Norenzayan, S. J. Heine, T. Yamagishi, & T. Kameda (Eds.), *Evolution, culture, and the human mind* (pp. 243–256). New York, NY: Psychology Press.

Schaller, M. & Murray, D. R. (2011). Infectious disease and the creation of culture. In M. Gelfand, C.-y. Chiu, & Y.-y. Hong (Eds.), *Advances in culture and psychology* (Vol. 1, pp. 99-151). New York, NY: Oxford University Press.

Schaller, M., & Neuberg, S. L. (2012). Danger, disease, and the nature of prejudice(s). *Advances in Experimental Social Psychology, 46,* 1–55.

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

Schaller, M., Park, J. H., & Mueller, A. (2003). Fear of the dark: Interactive effects of beliefs about danger and ambient darkness on ethnic stereotypes. *Personality and Social Psychology Bulletin, 29,* 637–649.

Schnall, S., Haidt, J., Clore, G. L. & Jordan, A. (2008). Disgust as embodied moral judgment. *Personality and Social Psychology Bulletin, 34,* 1096–1109.

Schulenburg, H., & Müller, S. (2004). Natural variation in the response of *Caenorhabditis elegans* towards *Bacillus thuringiensis*. *Parasitology, 128,* 433–443.

Simpson, J. A., & Gangestad, S. W. (1991). Individual differences in sociosexuality: Evidence for convergent and discriminant validity. *Journal of Personality and Social Psychology, 67,* 870–883.

Sperber, D., & Hirschfeld, L. A. (2004). The cognitive foundations of cultural stability and diversity. *Trends in Cognitive Science, 8,* 40–46.

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

Stevenson, R. J., Hodgson, D., Oaten, M. J., Moussavi, M., Langberg, R., Case, T. I., & Barouei, J. (2012). Disgust elevates core body temperature and up-regulates certain oral immune markers. *Brain, Behavior, and Immunity, 26,* 1160–1168.

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,* 99–108.

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

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

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

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

Van de Vliert, E. (2013). Climato-economic habitats support patterns of human needs, stresses, and freedoms. *Behavioral and Brain Sciences, 36,* 465–480.

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,* 429–437.

van Schaik,C. P. (2013). The costs and benefits of flexibility as an expression of behavioural plasticity: A primate perspective. *Philosophical Transactions of the Royal Society B, 368*, 20120339.

Vytal, K., & Hamann, S. (2010). Neuroimaging support for discrete neural correlates of basic emotions: A voxel-based meta-analysis. *Journal of Cognitive Neuroscience, 22,* 2864–2885.

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

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

Wolfe, N. D., Dunavan, C. P., & Diamond, J. (2007). Origins of major human infectious diseases. *Nature, 447,* 279–283.

Woody, E., Szechtman, H. (2011). Adaptation to potential threat: the evolution, neurobiology, and psychopathology of the security motivation system. *Neuroscience and Biobehavioral Reviews, 35,* 1019–1033.

Wu, B., & Chang, L. (2012). The social impact of pathogen threat: How disease salience influences conformity. *Personality and Individual Differences, 53,* 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,* 558–563.