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# The Behavioral Immune System
## Its Evolution and Social Psychological Implications

MARK SCHALLER
LESLEY A. DUNCAN

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November 27, 1966. For one of the authors (Mark Schaller) that was a bad day. It was his fourth birthday and his parents threw him a party, with balloons and ice cream and birthday party games. This wasn't easy because the family was living on the Serengeti plains of Tanzania, where neither balloons nor ice cream (nor very many other young children for that matter) were readily available. Despite his parents' intrepid efforts, the day was a disaster. Mark's balloon popped. He dropped his ice cream into the dirt. He cried and cried and cried.

Meanwhile, elsewhere in Tanzania, on exactly the same day, a fellow named McGregor was also having a bad day—a really bad day. Indeed, the minor setbacks of Mark's birthday party are trivial in comparison to the truly tragic events that befell McGregor that day.

McGregor lived on the eastern edge of Lake Tanganyika, in an area that is now Gombe National Park. McGregor was a chimpanzee. Chimpanzees are, like people, a highly social species. Chimps typically stay in very close contact with their fellow group members. Their health and reproductive success depend on it. They may spend several hours each day grooming each other—removing fleas, mites, and other ectoparasites from each other's fur. For most of McGregor's life, his social experiences were not unusual. But, in 1966, a polio epidemic struck the Gombe chimpanzees, and McGregor fell victim. The consequences are described in detail by Goodall (1986). McGregor lost the use of his legs, forcing him to drag his body backwards with his arms, or to attempt a series of bizarre somersaults as a crude means of locomotion. He lost control of his bladder, and so his awkward movements were accompanied constantly by a buzzing swarm of flies. And, as if that wasn't painful enough, McGregor's physical privations precipitated near-complete social rejection—as indicated by observations recorded on November 27, 1966:

> Of the total number of 32 adult and adolescent chimpanzees who visited camp at the time, 17 approached the crippled male. … Only nine adults approached closely … and of these only four actually touched him (two aggressively). … Humphrey [possibly his biological nephew] was the only chimpanzee who sometimes slept within 20 meters of the stricken male. … Perhaps the most striking aspect was the fact that not once in the 24 hours was [he] involved in a session of social grooming. (Goodall, 1986, pp. 233–234)

Behavioral avoidance and social rejection of diseased individuals is observed not only in chimpanzees, but in many other species too. Mice avoid mating with mice that are infected with parasitic viruses, protozoa, and larval nematodes (e.g., Kavaliers, Colwell, Braun, & Choleris, 2003). Bullfrog tadpoles avoid swimming in proximity to tadpoles infected with debilitating intestinal parasites (Kiesecker, Skelly, Beard, & Preisser, 1999). Closer to home, human beings prefer to maintain distance from others who are described as diseased, especially if the alleged disease is perceived to be contagious (Crandall & Moriarty, 1995).

Why is this? To some extent, the explanation is straightforward. Goodall (1986, p. 234) suggested that "avoidance of conspecifics showing abnormal behavior may be highly adaptive since it reduces the risk of spreading contagious disease." More broadly, it has been suggested that many animals, including humans, have an evolved capacity to detect symptoms of parasitic infection in others, and to respond with behaviors—such as behavioral avoidance and social rejection—that reduce the likelihood of contracting that infection oneself (Eibl-Eibesfeldt, 1979; Kurzban & Leary, 2001; Schaller, Park, & Faulkner, 2003).

Although that explanation is straightforward and perhaps even obvious, it has many additional implications that are more complex and a lot less obvious. A deeper consideration of how such an evolved process might operate yields a large number of novel implications for human social cognition and interpersonal behavior more generally. Evolved mechanisms designed to inhibit contact with disease-carrying conspecifics are likely to promote specific kinds of aversive reactions toward many specific kinds of people who are, in fact, perfectly healthy.

The purpose of this chapter is to discuss some of these implications. But first, we need to consider carefully the selection pressures that presumably led to the evolution of these psychological mechanisms in the first place. We must spend a bit of time in the past.

## THE PAST

### *Evolution of the Behavioral Immune System*

Parasites are an enduring part of human history. Infectious bacteria and viruses have existed on the planet far longer than people and other primates have; and as long as animals have had guts, those guts have been infected with helminths and worms (Brothwell & Sandison, 1967; Ewald, 1993; van Blerkom, 2003).

Not all parasites are harmful, but many are. The European plague outbreak in the Middle Ages killed millions of people (Lippi & Conti, 2002). Bacterial diseases wiped out up to 90% of the native populations in the Americas (Guerra, 1993). These are relatively recent events, but the dangers posed by parasites are hardly recent phenomena. The very fact that humans and other animals have evolved extraordinarily sophisticated immune systems attests to the antiquity of parasitic infections, to the enormous selection pressures that parasitic infections have exerted on animal populations, and to the fitness-conferring benefits associated with any adaptation that contributes to an antiparasite defense system.

The immune system is just one kind of antiparasite defense system, and, although effective in many ways, it has its downsides too. The mobilization of immunological defenses often consumes substantial metabolic resources, robbing individuals of energy that might be devoted to other fitness-enhancing tasks (Brown, 2003; Klein & Nelson, 1999). Specific features of immunological defense, such as fever, may be even further debilitating. Organisms are well-

served by the existence of an immune system, but they are best served when that immune system is engaged as infrequently as possible.

In addition, the immune system is limited by the boundaries of physical anatomy. The immune system is designed to combat infectious agents only after they are detected upon contact with the individual's body. It cannot prevent contact in the first place.

Therefore, it is entirely plausible that selection pressures posed by parasites led to the evolution of an additional antiparasite defense system as well, one designed to inhibit contact with infectious agents in the first place. This system is comprised of a set of mechanisms that allow individuals to detect the potential presence of parasites in the objects and individuals around them, and to engage in behaviors that prevent contact with those objects and individuals. This has been called the *behavioral immune system* (Schaller, 2006).

The operation of some sort of evolved behavioral immune system is implicated by abundant evidence—across many species—pertaining to foraging and feeding behavior. Sheep selectively avoid grazing on grasses contaminated with their own fecal waste (Cooper, Gordon, & Pike, 2000). People too show a disgust reaction and behavioral rejection toward foods that are potentially contaminated by parasites (Rozin, Millman, & Nemeroff, 1986). Of course, it's not just foods and other inanimate objects that host potentially-dangerous parasites. Other individuals do as well. So it's no surprise that the behavioral immune system also compels avoidance, and even outright rejection, of conspecifics (like poor McGregor) that demonstrate symptoms of physical illness.

### *How It Works: Cue Detection and Response*

Any effective defense system requires the coordinated operation of at least two kinds of underlying mechanism: mechanisms designed to detect cues signaling threat, and other mechanisms that respond to those cues by mobilizing some sort of defensive response. This is the case for the "real" immune system. Specific mechanisms distinguish the difference between organic entities that belong in the body and those—like viruses—that don't. When pathogenic intruders are detected, other mechanisms within the system are triggered that attempt to repel those pathogens through a variety of physiological means. In an analogous fashion, the behavioral immune system also is comprised of mechanisms designed for detection and response.

The detection mechanisms employ the organism's ordinary sensory organs as a means of recognizing parasite-connoting cues at a distance. Frogs use specific kinds of chemical signals for this purpose (Kiesecker et al., 1999). Many mammals use olfactory cues of some sort (Kavaliers et al., 2003), and surely people do too. In addition, given our highly-developed visual systems—which allow us to detect many different kinds of fitness-connoting signals from a distance—our parasite-detection mechanisms are sensitized to detect visual cues signaling possible parasitic infection. This makes sense, of course, given that the symptoms of many parasitic infections are manifest in individual's superficial appearance or behavior (e.g., skin lesions, rashes, coughing spasms).

The behavioral immune system also includes mechanisms designed to respond in functionally-useful (i.e., fitness-enhancing) ways once a parasite-connoting cue has been detected. Behavioral avoidance is the functionally-relevant "goal" for which these mechanisms are designed. But behavior doesn't just happen; it is the product of underlying psychological activity. In humans, this activity involves both affective and cognitive mechanisms.

Emotions are instrumental in motivating immediate behavioral reactions (see Buck, chapter 6, this volume; Ellsworth, chapter 5, this volume; Forgas, chapter 7, this volume; Lieberman, chapter 11, this volume). Both fear and disgust motivate behavioral avoidance. Disgust in particular seems likely to be an important part of the behavioral immune system. The capacity for disgust may have arisen originally to protect individuals from the ingestion of toxins and other food-based contaminants (Rozin & Fallon, 1987), but the mechanisms involved in the disgust experience appear to have evolved to serve a parasite-defense function as well. Disgust is triggered by the visual perception of skin lesions, runny noses, and other obvious symptoms of parasitic infection (Curtis, Aunger, & Rabie, 2004; Curtis & Biran, 2001).

Disgust may motivate an immediate and impulsive avoidant response, but that's it. The emotional experience alone cannot compel wariness about future interactions, nor can disgust alone precipitate more planful actions (such as coordinated efforts at quarantine and social exclusion) that help to eliminate the long-term threat posed by possibly parasitized individuals. To facilitate these kinds of fitness-relevant behaviors, various cognitive processes must be engaged as well. In humans the detection of any parasite-connoting cue may have immediate implications on higher-order cognitive processes involved in inference and memory, which may then influence the specific nature of attitudes and other enduring social knowledge structures. These, in turn, are likely to have consequent effects on social decision making and behavior.

If the behavioral immune system influenced reactions only to truly diseased individuals, it would still constitute a worthwhile topic of scientific inquiry, but would perhaps be of limited relevance to the broader range of social psychological phenomena. In fact, however, the behavioral immune system appears to operate in such a way that it often precipitates aversive reactions to individuals who are perfectly healthy. Consequently, it has direct implications for many phenomena that lie squarely in the center of the social psychological literature—including interpersonal attraction, intergroup prejudice, and social stigmas of various kinds. To understand why, it is useful to apply the logic of signal detection.

### *The Signal Detection Problem and its Solution: Oversensitivity and Overgeneralization*

The behavioral immune system is designed not to respond to the presence of parasites, per se, but rather to the *perceived* presence of parasites as indicated by superficial sensory signals. Many of these cues, presumably, are probabilistically predictive of the presence of parasites. But even the most diagnostic of symptoms is highly imperfect. (Some healthy people cough, and some sick people don't.) The result is a classic signal-detection problem, with the potential to make both false-positive errors (a healthy person is erroneously perceived to be sick) and false-negative errors (a sick person is erroneously perceived to be healthy). Any general tendency toward avoiding false positives leads to an increase in the rate of false negatives, and vice versa. Evolutionary logic indicates that this dilemma will be resolved in favor of minimizing the error that poses the greatest costs to an individual's fitness, even if that results in an increased rate of making the other kind of error (Haselton & Nettle, 2006; Nesse, 2005; for a broader discussion of signaling and signal-detection systems see Gangestad & Thornhill, chapter 3, this volume). In this case, as with most evolved systems designed for self-protection, the fitness costs associated with false negatives are considerably greater than those associated with false positives. The adaptive resolution is clear: The behavioral immune system errs on the side of false positives (Kurzban & Leary, 2001). Thus, we are hypervigilant for signs of sickness, and any such signal (whether it's a tubercular cough or merely some innocuous guttural tic, whether it's a rash of

infectious pox or merely some superficial allergic inflammation) is liable to trigger aversive emotional, cognitive, and behavioral reactions.

It is unlikely that there was a finite and stable set of symptoms associated with parasitic infections in ancestral environments. Different kinds of parasite would have produced different infectious symptoms. (The rash diseases—such as measles, mumps, and scarlet fever—are all evolutionarily ancient, as is tuberculosis, and all are associated with somewhat different specific symptoms.) Different individuals are likely to have responded differently to the same kind of parasitic infection (the rhinovirus may manifest in a cough, or in a runny nose, or both, or neither). And parasitic species themselves—especially bacteria and viruses—evolve at an exceptionally rapid pace, an evolution that is reflected in the highly variable nature of infectious symptoms over time (Ewald, 1993). A behavioral immune system that was calibrated too tightly to specific perceptual cues would have resulted, over time, in many costly false-negative errors. More adaptive would be a system that responded to a broader, more crudely-defined range of cues. This suggests that the behavioral immune system errs not merely on the side of oversensitivity, but also on the side of overgeneralization: Any gross deviation from the species-typical norms in morphology and motor behavior may be implicitly interpreted as symptomatic of a parasitic infection, and so may trigger the behavioral immune response (Kurzban & Leary, 2001; Zebrowitz & Montepare, 2006).

Thus, the behavioral immune system operates in a manner analogous to the real immune system. Just as the antipathogen defense system provided by the real immune system is hypersensitive to intrusion, and may be mobilized in response to organic matter that is entirely benign (or even beneficial, as in the case of organ transplants), the behavioral immune system too responds in a hypersensitive and overgeneral way to the perceived presence of parasites in the sensory environment. This has far-reaching implications for social perception and behavior: Simply because people may display some superficial form of non-normality, we may respond to them—even if they are perfectly healthy—as though they are carriers of some contagious disease.

### *The Cost–Benefit Problem and Its Solution: Functional Flexibility*

Antiparasite defense systems confer adaptive benefits, but they also incur costs whenever they are triggered. We have already mentioned the physiological costs associated with the mobilization of the real immune system. There are analogous costs associated with the operation of the behavioral immune system. The emotional, cognitive, and behavioral responses triggered by the behavioral immune system all consume metabolic resources. And because of the finite resources available to an individual at any moment, the activation of the behavioral immune system limits the extent to which other adaptive behaviors might be engaged (e.g., disgust and behavioral avoidance are typically incommensurate with mating motives; for additional discussions of evolutionary cost–benefit analyses and their implications, see Simpson & LaPaglia, chapter 10, this volume; Todd, chapter 9, this volume). Therefore, like many adaptive psychological systems, the operation of the behavioral immune system is likely to have evolved so as to be functionally flexible and responsive to regulatory cues (Schaller, Park, & Kenrick, in press; see also Kenrick et al., chapter 4, this volume). Aversive responses to potentially-parasitized others are most likely to be triggered when additional cues in the immediate environment indicate that the functional benefits of these responses are especially likely to outweigh the functional costs.

Some regulatory cues lie in chronic individual differences in attitudes, traits, and temperament. People differ in the extent to which they are vulnerable (or, perhaps more importantly, *perceive* themselves to be vulnerable) to the transmission of contagious diseases. Other regulatory cues lie in temporary features of the immediate situation. Information present in any specific context may make germs and their potential transmission especially salient for a short period of time. Still other cues lie in chronic features of the local ecology. In some geographical contexts, parasitic diseases have posed an especially strong threat to individual fitness, with persistent consequences on local rituals and norms pertaining to hygiene, food preparation, and so on. Regardless of the locus of these regulatory cues—whether chronic or temporary, and whether rooted in the external environment or a perceiver's own idiosyncratic knowledge structures—the information they provide is likely to moderate the activation of the behavioral immune system. If one is unaware of (or feels invulnerable to) the threat of disease, the activation of the system is likely to be muted. On the other hand, if the threat of disease is highly salient (or if one feels highly vulnerable), the reactivity of the system is likely to be more pronounced.

## THE PRESENT

These speculations about the evolution of the behavioral immune system make sense within the adaptive framework of evolutionary psychology. Ideally, this kind of conjecture should not simply be sensible; it should also be useful—even to scholars who care nothing about the evolutionary past. In fact, the principles of adaptive overgeneralization and functional flexibility imply a broad range of effects on contemporary social cognition and behavior. Some of these implications have been empirically tested in recent years. The results highlight the operation of the behavioral immune system in a wide range of contemporary social psychological phenomena.

### *Aversive Responses to Superficial Disfigurements and Disabilities*

There is now a substantial body of work documenting aversive responses to people displaying non-normative morphological cues of various kinds, including superficial facial anomalies and physically disabling conditions. These aversive responses may result from a variety of conceptually distinct psychological processes, many of which have nothing to do with parasite avoidance at all (for reviews, see Heatherton, Kleck, Hebl, & Hull, 2000). Is there reason to suppose that, in addition to these other processes, the specific mechanisms implicated in the behavioral immune system also play a substantial role? Yes. Evidence in favor of that assertion emerges from studies that do at least one of two things. They take measures that assess the specific kinds of semantic information that are cognitively associated with morphologically anomalous individuals. Or they test the extent to which aversive responses are facilitated under circumstances in which perceivers feel more vulnerable to the potential spread of contagious disease. Or they do both. These studies not only implicate the role of the behavioral immune system in reactions toward a variety of objectively noncontagious peoples, they also document novel phenomena whereby these reactions vary under predictable circumstances.

Park, Faulkner, and Schaller (2003) reported a pair of studies that implicate the behavioral immune system in aversive responses to individuals who are physically disabled. There is a large literature documenting the fact that people are uncomfortable around others who

are disabled, and often attempt to behaviorally avoid close contact with these others (e.g., Snyder, Kleck, Strenta, & Mentzer, 1979). If this prejudice results in part from the heuristic operation of the behavioral immune system, it follows that behavioral avoidance might be especially strong among individuals who are chronically concerned about the spread of contagious diseases. Consistent with this hypothesis, Park et al. (2003) found that individuals who score highly on measure of "perceived vulnerability to disease" (PVD) were less likely to report having friends or acquaintances with disabilities. In addition, Park et al. employed reaction time methods to assess the extent to which disabled individuals (compared to morphologically normal individuals) were implicitly linked to semantic information connoting disease. Results revealed that, not only were disabled individuals more likely than nondisabled individuals to be associated with disease, this effect was stronger among perceivers who scored more highly on either the PVD measure or on a measure assessing sensitivity to disgust.

In this implicit association study, the disabled target individuals were described in such a way that, by any objective standard, they posed no realistic disease threat whatsoever. The results are therefore consistent with the conjecture that the behavioral immune system responds automatically to visual cues of morphological anomaly, even when rational appraisal indicates the absence of any realistic threat. Duncan (2005) conducted a strong test of the alleged automaticity of this response. Participants were provided with brief biographical sketches of two men, and each biographical sketch was accompanied by a facial photograph. One man had a very noticeable "port wine stain" birthmark on his face, but this birthmark was explicitly described as superficial and the man himself was described as strong and healthy. The other man looked just fine, but was described as suffering from a strain of drug-resistant tuberculosis. Participants then responded to a computer-based reaction time task, designed to assess which of the two men was more strongly associated with the semantic concept "disease." Results showed that, across all participants, there was a general tendency to associate disease with the facially-disfigured man (who was known to be healthy) more strongly than the man who was actually known to suffer from a contagious disease (but who looked normal). In short, even when processes of rational appraisal explicitly indicate otherwise, facial disfigurements may implicitly connote the threat of contagious disease.

### *Antifat Attitudes*

Previous research has suggested that negative attitudes toward fat people are rooted, in part, in personal ideologies and cultural value systems that prescribe hard work, self-denial, and willpower (Crandall, 1994; Crandall & Martinez, 1996). Consistent with this perspective, fat people are commonly stereotyped as lazy, and are more strongly stigmatized when their obesity is attributed to personally-controllable causes (e.g., Teachman, Gapinski, Brownell, Rawlins, & Jeyaram, 2003). But fat people are also commonly stereotyped as dirty or smelly, and images of fat people tend to arouse disgust (Harvey, Troop, Treasure, & Murphy, 2002)—observations hinting at the possibility that antifat attitudes may also be rooted in the operation of the behavioral immune system.

This possibility is entirely plausible, given our speculations about how the behavioral immune system operates. If the system is sensitive to any gross deviation from morphological norms, then it's likely to react aversively to individuals with bodies that are either skeletally thin or hugely obese. There has been very little research examining aversive reactions to super-skinny

people, but some recent studies explicitly examined whether the heuristic operation of the behavioral immune system might contribute to antifat attitudes.

Park, Schaller, and Crandall (2006) examined whether antifat attitudes were predicted by individual differences in perceived vulnerability to disease—focusing specifically on a subscale that assesses wariness of germs and their transmission. Results indicated that these individual differences did indeed predict antifat attitudes: People who were chronically more concerned about germs also expressed a stronger dislike of fat people. This effect was especially strong when antifat attitudes were measured immediately after the visual perception of specific obese individuals—a result consistent with the idea that the behavioral immune system is hypersensitive to visual cues. It's worth noting also that the effect on antifat attitudes was statistically independent of the predictive effect of separate measures assessing attributions about willpower. This suggests that ideological processes and parasite-defense processes both contribute to antifat attitudes, but in different ways.

This last conclusion is further substantiated by another study reported by Park et al. (2006). This experiment assessed cognitions implicitly associated with obese individuals, and examined the impact of a manipulation designed to make specific concerns temporarily salient. Results revealed that the implicit association linking fat people (compared to nonfat people) with disease was amplified following a manipulation that made infectious pathogens especially salient. The amplifying effect of the pathogen-salience manipulation emerged only on implicit associations linking fat people to disease; it did not increase associations linking fat people with unpleasant concepts in general. In contrast, a manipulation that made ideological concerns salient led to an increased implicit association between fat people and unpleasantness, but had minimal impact on the fat–disease association.

These results not only have implications for understanding contemporary prejudices toward obese individuals, they also have unique implications for understanding the operation of the behavioral immune system itself. It might be logical to perceive dramatically underweight individuals as potential parasite carriers (given that many parasitic infections do result in substantial weight loss), but there is little logical basis to associate obesity with contagious parasites. Nor is there much reason to assume that truly obese individuals were evident in the ancestral environments during which the behavioral immune system presumably evolved. The results of Park et al. (2006) therefore highlight the heuristic (nonrational) operation of the behavioral immune system, and they highlight its adaptive overgeneralization. The behavioral immune system responds not merely to specific cues that were evident in ancestral environments; it appears to have evolved so as to respond to any kind of apparent morphological deviation from population norms.

### *Responses to Physically Attractive and Unattractive Others*

The behavioral immune system may be sensitive not only to gross deviations from morphological norms, but may also be attuned to some relatively subtle deviations—at least in the realm of facial physiognomy.

Human visual systems are highly attuned to facial features. We have specialized neurological equipment dedicated to the visual perception of faces (Kanwisher, 2000). Our subjective impressions of another's attractiveness are influenced by specific aspects of facial physiognomy (such as bilateral symmetry, and the extent to which the size of specific facial features match population prototypes) that we appear to process implicitly and without conscious

awareness (e.g., Langlois & Roggman, 1990; see also Halberstadt, chapter 15, this volume). It has been argued that these sorts of subtle morphological variables are predictive of an individual's health status and future health outcomes (Fink & Penton-Voak, 2002; Thornhill & Gangestad, 1999). Consistent with this argument, evidence reveals that not only are substantially anomalous faces judged to be less healthy, but so too are faces that are simply perceived to be subjectively less attractive (Zebrowitz, Fellous, Mignault, & Andreoletti, 2003; Zebrowitz & Rhodes, 2004).

Gangestad and Buss (1993; see also Gangestad, Haselton, & Buss, 2006) report a particularly interesting finding bearing on the link between physical attractiveness and the presence of parasites. Employing a cross-cultural methodology to test a hypothesis about functional flexibility, they found physical attractiveness was an especially prized attribute in a mate within societies that historically had a high prevalence of infectious parasites,

This evidence has been interpreted as indicating that a subjective assessment of another's facial attractiveness serves as an indicator of that individual's genetic fitness. However, the same evidence is consistent with a process whereby individuals use facial attractiveness (or rather, *un*attractiveness) as an heuristic indicating the actual presence of potentially-contagious parasites. Is there any special empirical reason to suppose that unattractiveness really does trigger the behavioral immune system? Possibly. If attractiveness was simply a clue to genetic fitness, one might expect the impact of physical (un)attractiveness to be rather constrained in scope—exerting effects primarily in the domain of mating relations, but of limited impact in other domains of social life. In fact, however, physical attractiveness is valued—and physical unattractiveness compels aversive responses—across a broad range of social inferences and interactions (Biddle & Hamermesh, 1998; Eagly, Ashmore, Makhijani, & Longo, 1991; Matter & Matter, 1989). In addition, if attractiveness was merely a cue to genetic fitness, then one might expect the effects of pathogen prevalence, described above, to be especially strong among female perceivers (because women are especially attentive to indicators of genetic fitness). In fact, however, the results of Gangestad et al. (2006) show the opposite effect: The moderating impact of pathogen prevalence was stronger among men than among women. These results don't argue against the hypothesis that attractiveness serves as an heuristic cue for genetic fitness, but they do suggest that something else might be going on as well. That something else may be the operation of the behavioral immune system.

### *Xenophobia and Ethnocentrism*

In human populations, the behavioral immune system may be responsive not only to morphological cues, but also to a broader set of cues indicating that another individual is foreign to the local population. There are at least two plausible reasons why. First, contact with individuals from previously-unencountered populations is associated with an increased risk of contracting contagious diseases to which one has no acquired immunity. Second, foreign peoples are likely to be unaware of, and more likely to violate, local customs (such as those pertaining to food preparation and personal hygiene) that serve as barriers to the transmission of disease. Thus, in contemporary social ecologies, the mechanisms that define the behavioral immune system may generalize beyond the tendency to respond to cues signaling morphological anomaly; they may respond to cues signaling cultural foreignness as well. Regardless of their local social environment, individuals may be especially adept at learning to detect a wide range of inferential cues that discriminate between familiar and foreign peoples. And when those cues are detected,

they may promote the familiar emotional, cognitive, and behavioral responses associated with the behavioral immune system.

Consistent with this reasoning, Schiefenhövel (1997) observed that people often display disgust reactions when speaking about ethnic outgroups, and Rozin, Haidt, McCauley, and Imada (1997, p. 73) suggested that "disgust in humans serves as an ethnic or outgroup marker." To more rigorously test this conjecture, Faulkner, Schaller, Park, and Duncan (2004) conducted a series of studies that exploited the logic of functional flexibility.

In one set of studies, Faulkner et al. (2004) tested whether chronic concerns of vulnerability to parasitic infections—as measured by the "perceived vulnerability to disease" (PVD) scale—predicted attitudes towards immigrants from various geographical regions. Results revealed that higher levels of PVD predicted stronger anti-immigrant attitudes—but only toward immigrants from subjectively foreign locations. There was no such effect on attitudes toward culturally familiar immigrant populations.

The contribution of the behavioral immune system to xenophobic attitudes was also implicated in a pair of experiments reported by Faulkner et al. (2004). In both experiments, participants were first exposed to a brief slide show that either made salient the potential dangers posed by germs and germ transmission, or (in a control condition) made salient other dangers that were irrelevant to disease (e.g., electrocution). Results from both experiments revealed more strongly xenophobic attitudes after germs (rather than disease-irrelevant threats) were made salient. For instance, in one of these experiments, participants in Vancouver were told about a government program designed to recruit new immigrants to Canada, and were asked to indicate how much money should be spent to recruit immigrants from a variety of different countries that had been prerated as either culturally familiar (e.g., Taiwan, Poland) or unfamiliar (e.g., Mongolia, Brazil). Participants who had been exposed to the control slide show allocated roughly equal amounts of money to recruit immigrants from both familiar and unfamiliar places, but those for whom germ transmission had been made salient were much more likely to allocate money to recruit immigrants from familiar rather than unfamiliar places.

These findings are complemented by more recent work by Navarrete and Fessler (2006). In one study they observed that not only does perceived vulnerability to disease predict more negative attitudes toward foreign peoples (xenophobia), it also predicts more positive attitudes toward one's own cultural ingroup (ethnocentrism). In another study, they found that another disease-relevant individual difference variable—sensitivity to disgust—also predicts both xenophobia and ethnocentrism.

These results do not diminish the importance of the many other psychological processes that contribute to xenophobia and ethnocentrism. There is no doubt that these phenomena are multiply-determined; they are influenced also by processes pertaining to fear, mistrust, conflict, social identity, and mere categorization. But the fact of those well-known processes should not blind us to the apparent role of a less obvious process that also contributes to xenophobia and ethnocentrism: The hypersensitive and overgeneralized operation of a psychological system designed to protect our bodies from contact with parasites.

## THE FUTURE

The results reviewed above suggest that the behavioral immune system has implications for a broad range of psychological responses to people who, in fact, may be completely healthy. In future research, it will be worthwhile to examine additional implications, perhaps particularly in

the realm of actual interpersonal behavior. The subtle operation of the behavioral immune system may contribute, for instance, to many specific acts of aggression and social ostracism (e.g., see Spoor & Williams, chapter 17, this volume).

It will also be worthwhile to consider implications that exist not merely at the individual level of analysis, but at the societal level of analysis. The behavioral immune system may play an important role in shaping the collective belief systems that define a culture (Schaller, 2006). One route is through interpersonal communication. Cultural norms are sculpted, often unintentionally, through communication processes (Schaller, 2001). People may be especially likely to communicate about things that seem relevant to disease and disease transmission—as indicated perhaps by the finding that disgust-arousing stories are especially likely to be communicated, and to become culturally popular (Heath, Bell, & Sternberg, 2001). Moreover, disease-relevant arguments and rhetorical devices may be especially persuasive in sculpting popular opinion and public policy. (The abundance of parasite-relevant imagery in Nazi propaganda offers one sobering historical example.) The intriguing upshot, still largely unexplored, is that the evolution of the behavioral immune system may not only exert a pervasive influence on human social cognition; it may also, as a consequence, influence human culture.

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