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Review

The behavioural immune system and the psychology of human sociality

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Because immunological defence against pathogens is costly and merely reactive, human anti-pathogen defence is also characterized by proactive behavioural mechanisms that inhibit contact with pathogens in the first place. This *behavioural immune system* comprises psychological processes that infer infection risk from perceptual cues, and that respond to these perceptual cues through the activation of aversive emotions, cognitions and behavioural impulses. These processes are engaged flexibly, producing context–contingent variation in the nature and magnitude of aversive responses. These processes have important implications for human social cognition and social behaviour—including implications for social gregariousness, person perception, intergroup prejudice, mate preferences, sexual behaviour and conformity. Empirical evidence bearing on these many implications is reviewed and discussed. This review also identifies important directions for future research on the human behavioural immune system—including the need for enquiry into underlying mechanisms, additional behavioural consequences and implications for human health and well-being.

Keywords: health; infection; mating; norms; prejudice; sociality

1. INTRODUCTION

Humans and other animals have a long history of living in proximity to parasitic organisms—bacteria, viruses, helminths—that cause infectious diseases. This proximity imposed substantial selection pressures on ancestral populations, resulting in many different adaptations that, in a variety of ways, mitigate the potential fitness costs posed by these pathogens. Most obviously, there evolved the sophisticated suite of physiological mechanisms that define immunological defence systems, which are designed to detect the presence of pathogens within the body and, when detected, to mobilize physiological responses that encapsulate, kill or otherwise eliminate these pathogenic intruders. Immunological defence against infection has obvious fitness benefits, but can be substantially costly too [1]. An immune response is metabolically costly (consuming caloric resources that might otherwise be devoted to other important physiological systems) and can be temporarily debilitating (because of fever, fatigue and other physiological consequences of an aggressive immunological response). And, of course, immunological defence is merely *reactive*—triggered only after the pathogenic infection has occurred within the body.

Given these limits and costs associated with immunological defence against pathogens, additional fitness benefits would have accrued from an additional set of *proactive* mechanisms that—by guiding organisms' behaviour—inhibit contact with pathogens in the first place. These mechanisms offer a sort of behavioural prophylaxis against infection [2]. Indeed, it is not merely metaphorical to suggest that these mechanisms comprise a *behavioural immune system* that is separate from, and complementary to, the 'real' immune system [3–5].

Behavioural defence against pathogens has been observed across a wide variety of animal species [6,7]. Some forms of behavioural defence—such as cytokine-induced sickness behaviour [8,9] and self-medication [10,11]—are reactive, rather than proactive. But there is also abundant evidence of proactive behavioural defence as well: wood ants collect pieces of coniferous resin as a prophylactic defence against bacteria and pathogenic fungi [12]; bullfrog tadpoles selectively avoid swimming near infected tadpoles [13]; female mice respond aversively to the odours of male mice infected with nematode parasites [14]; chimpanzees avoid social contact with (and may even respond aggressively towards) other chimpanzees infected with polio [15]. In short, just as the 'real' immune system is characterized by mechanisms that facilitate adaptive immunological responses to pathogens that enter the body, the behavioural immune system is characterized by mechanisms that facilitate adaptive psychological responses to perceptual cues connoting the presence of pathogens in the immediate perceptual environment—including the presence of pathogens in conspecifics. The specific nature of the perceptual detection and behavioural response mechanisms may vary across species, but the existence of these detection and response mechanisms is common across species.

In recent years, the behavioural immune system has received considerable attention in the study of human

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behaviour, with an emphasis on the specific psychological mechanisms (pertaining to attention, perception, cognition and emotion) that guide human behaviour. Much of this work has focused on one specific emotion—disgust—that is associated with disease-avoidance behaviour, on the specific kinds of perceptual things that elicit disgust, and on the specific circumstances under which a disgust response is either exaggerated or reduced [2,16,17]. This work has been reviewed extensively elsewhere [18,19]. My focus here is on a set of complementary programmes of research that focus less on emotion and more on social cognition and social interaction—lines of enquiry that explore how the behavioural immune system guides people's perceptions of, thoughts about and behaviour towards other individuals within their immediate social ecologies. The upshot is an emerging literature documenting many subtle but important linkages between anti-pathogen defence and the contours of human social life.

2. THE BEHAVIOURAL IMMUNE SYSTEM: DETECTION, RESPONSE AND FUNCTIONAL FLEXIBILITY

Like the 'real' immune system, the behavioural immune system includes both detection and response mechanisms. Detection mechanisms employ sensory systems (e.g. olfaction, vision) to detect things that appear to pose some infection risk. These things include inanimate objects (e.g. a pile of faeces, a piece of putrid meat). These things also include conspecifics (i.e. other people) who may be inferred to pose an infection risk either because (i) they appear already to be infected or because (ii) they tend to behave in ways that increase the likelihood that infections will be spread to others (e.g. by failing to observe customary hygiene practices). When a superficial cue connoting infection risk is detected, it triggers a cascade of adaptive psychological responses. These responses include not only the emotional experience of disgust but also the activation of aversive cognitions into working memory, and the arousal of a motivational system that guides decision-making strategies and motor movements in ways that minimize the infection risk (e.g. behavioural avoidance of and social condemnation of people who appear to pose an infection risk).

Of course, just as immunological response has costs (as well as benefits), the detection and response mechanisms that characterize the behavioural immune system can also be costly (as well as adaptively beneficial). These costs arise because disease-avoidant behavioural responses can consume considerable metabolic resources, and because these responses may inhibit the satisfaction of other fitness-relevant goals (e.g. avoidance of interpersonal contact can interfere with valuable opportunities for social exchange, mating, etc.). Therefore, just as with other adaptive psychological systems, the mechanisms that comprise the behavioural immune system are 'functionally flexible': They are sensitive to contextual information bearing on cost–benefit ratio, with predictable context–contingent variation in the nature and magnitude of response [5,20,21].

The benefits offered by the behavioural immune system (reduction of infection risk) are a direct function of individuals' actual vulnerability to infection: these benefits are minimal under conditions in which perceivers are invulnerable to infection, and are relatively greater under conditions in which vulnerability to infection is also relatively greater. Consequently, the detection and response mechanisms that characterize the behavioural immune system are sensitive to any kind of information that suggests increased vulnerability to the transmission of infectious diseases. This information may arise from sources either internal to the perceiver (e.g. chronic anxieties and worries) or in the external environment (e.g. context-specific perceptual reminders of the threat posed by infectious diseases). This information may be veridical (e.g. correctly implying that the perceivers' immunological defences are compromised) but it need not be. Indeed, regardless of the source or veracity of an individual's subjective perception of vulnerability to infection, that subjective perception is likely to influence the activation of the behavioural immune system. Under conditions in which individuals perceive themselves to be more vulnerable to infection, they are expected to be more perceptually sensitive to things (including people) who appear to pose an infection risk [22]; and when those things (including people) are detected, those perceivers are expected to exhibit more exaggerated aversive responses—greater disgust, greater activation of aversive cognitions into working memory, greater motivation for behavioural avoidance and so forth.

The functional flexibility of the behavioural immune system has many implications for human social interaction, and for human sociality more broadly. Many kinds of human social behaviour that serve disease-irrelevant goals (e.g. acquisition of resources, sexual reproduction) also have potential implications for disease transmission too. Consequently, these social behavioural tendencies may vary, depending on the extent to which individuals are (or merely perceive themselves to be) vulnerable to infectious disease. This has important consequences for a wide variety of social attitudes, social perceptions and social activities.

3. SOCIAL GREGARIOUSNESS

Research on the structure of human personality reveals a small handful of fundamental trait dimensions that characterize individual differences in psychological functioning. One of those dimensions is extraversion—the extent to which individuals are socially gregarious or not.

Gregariousness is typically considered to be beneficial. Indeed, empirical research shows that extraversion is associated with many positive outcomes, including higher levels of happiness and increased opportunities for sexual reproduction [23,24]. But gregariousness may have infection-specific costs as well. People who are more gregarious tend to come into interpersonal contact with a relatively larger number of people, with the implication that they are more likely to be exposed to interpersonally transmitted pathogens [25,26]. These costs of gregariousness are relatively

greater (and more likely to outweigh the social benefits) under conditions in which individuals are more vulnerable to infection. Therefore, when people feel relatively invulnerable to infection, they may show a natural tendency towards gregariousness. However, under conditions in which people perceive themselves to be vulnerable, it follows that they will be less sociably inclined.

Two recent experiments tested and supported this hypothesis [27]. Both experiments included an experimental manipulation designed to make some participants (compared with those in a control condition) especially aware of the threat posed by infectious pathogens. One experiment assessed participants' personality traits, and found that the pathogen-salience manipulation caused participants to rate themselves as relatively less extraverted. The second experiment measured actual motor movements in response to the visual perception of other people, and found that the pathogen-salience manipulation caused participants to engage in relatively more socially avoidant motor movements. Together, these findings indicate that the perceived threat of infectious disease has predictable implications for individuals' basic behavioural tendency towards social gregariousness.

A parallel pattern of variation in social gregariousness is found when treating entire populations—rather than individuals—as units of analysis. There is considerable worldwide ecological variability in the historical prevalence of infectious diseases. There is also considerable worldwide cultural variability in dispositional tendencies towards extraversion. These two variables are linked: among populations living in regions that historically have had a high prevalence of pathogens, the mean level of extraversion is lower [28].

4. DISCRIMINATORY SOCIALITY (PREJUDICE)

The inhibition of social gregariousness may offer one means of reducing infection risk, but it is a rather blunt tool for doing so. In many animal species, behavioural disease avoidance is indicated not so much by unsociable behaviour in general, but by *discriminatory* unsociable behaviour: the use of diagnostic perceptual cues to selectively avoid particular conspecifics that appear to pose a particularly high risk of infection [13–15]. The same sort of discriminatory sociality is observed in humans. Aversive emotional and cognitive responses are aroused by the perception of other individuals who are known to be diseased, or who are judged to be at greater risk of being diseased; and these aversive responses are especially pronounced when the diseases are perceived to be especially infectious [29,30]. Thus, as a result of the behavioural immune system and its implications for discriminatory sociality, many people suffering from infectious diseases also suffer from prejudice and social stigmatization as well.

Importantly (and troublingly), the evolved design of the behavioural immune system can not only lead to the social stigmatization of people who truly are infectious but also to equally pernicious prejudices directed against people who are not. Here is why:

Most disease-causing organisms (e.g. bacteria) are so tiny as to be imperceptible to human perceptual processes, and so it is largely impossible for people to directly detect the presence of pathogen infection in others. Because of this fact, the behavioural immune system responds to the *inferred* presence of parasites as indicated by superficial sensory cues (e.g. coughing spasms, skin discolorations). These cues may be probabilistically predictive of the presence of infection, but are still imperfectly diagnostic. This results in a signal-detection problem, with the potential to make both false-positive errors (a healthy person is erroneously perceived to be infectious) and false-negative errors (an infectious person is erroneously perceived to be healthy). From an adaptive perspective, one would expect a particular form of signal-detection bias to emerge: a bias that minimizes the likelihood of making the error with the greatest potential fitness cost, even though that bias inevitably leads to many errors of the opposite kind [31,32]. As with other psychological systems designed for self-protection, false-negative errors are likely to be especially costly. And so the behavioural immune system errs on the side of making false-positive errors instead [4,5,21]. The upshot is that the behavioural immune system is perceptually sensitive to any superficial cue that appears likely to be a symptom of infection—even if it is objectively not.

Furthermore, there is no finite category of superficial cues to which the behavioural immune system is sensitive. This is because there is no finite set of symptoms associated with infection. (Different kinds of parasitic organisms produce different kinds of symptoms. Different people may show somewhat different symptoms even if they are infected with the same species of parasite. And parasitic species can evolve rapidly, with the consequence that their symptomatic manifestations may be highly variable over time.) Thus, to avoid costly false-negative errors, the behavioural immune system must be sensitive to a very broad range of cues that might be potential indicators of infection. Indeed, it has been suggested that any perceived deviation from prototypical human morphology and motor behaviour may implicitly connote potential infection risk [4,5,33].

Therefore, just as the 'real' immune system responds not only to actual pathogenic infection but also to intrusion by benign organic matter (as in the case of organ transplants, for example), the behavioural immune system also responds to an over-general set of superficial social cues. The result is a set of predictable prejudices directed at people who may be objectively non-infectious, but who simply have some sort of non-prototypical physical appearance. Furthermore (in keeping with the principle of functional flexibility), these prejudices are likely to be especially pronounced under conditions in which perceivers feel especially vulnerable to infection.

This line of reasoning is supported by many empirical studies, some of which employ the methodological tools of cognitive psychology to assess the automatic activation of semantic concepts into individuals' working memory [4,5]. It has been found, for example, that aversive semantic concepts (such as 'disease') are more readily activated into working memory upon encountering people with physical disabilities and people

bearing the characteristic features of old age—and that these implicit prejudices occur especially strongly among individuals who feel especially vulnerable to infection [34,35]. Similarly, people who feel more vulnerable to infection express stronger anti-fat attitudes and are more likely to implicitly associate obese people with aversive concepts connoting disease [36]. This latter finding is perhaps especially revealing about the manner in which the behavioural immune system guides discriminatory sociality. Obesity is not objectively diagnostic of pathogen infection (if anything, the opposite is more likely to be true: infectious diseases are more likely to cause weight loss than weight gain) but it does represent a substantial deviation from prototypical human morphology. This finding therefore attests to the behavioural immune system's sensitivity to a very broad category of superficial cues connoting non-normative physical appearance.

An additional programme of research reveals that the behavioural immune system produces a somewhat different form of discriminatory sociality as well: aversive responses to subjectively foreign peoples. There are, of course, many different psychological sources of xenophobia and ethnocentrism, and some of these psychological processes have nothing to do with infectious disease; still, disease-avoidant processes apparently contribute to these discriminatory outcomes. There are at least two distinct reasons why subjective 'foreign-ness' may implicitly connote an increased infection risk. First, exotic peoples may be host to exotic pathogens that can be especially virulent when introduced to a local population. Second, exotic peoples may be more likely to violate local behavioural norms (in domains pertaining to hygiene, food preparation, etc.) that serve as barriers to pathogen transmission. Thus, perceivers are likely to be hypersensitive to inferential cues that discriminate between familiar and foreign peoples and, when those cues are detected, they are likely to trigger the aversive, discriminatory responses associated with the behavioural immune system. This is especially likely to occur when perceivers feel especially vulnerable to infection.

Many studies now support this hypothesis. One provocative study revealed that women in their first trimester of pregnancy—when the 'real' immune system is naturally suppressed—reported exaggerated ethnocentrism and xenophobia [37]. Similar exaggerations in xenophobia occur among people who merely perceive themselves to be especially vulnerable to infection [38]. In one experiment, students at the University of British Columbia (in Canada) watched one of two slide shows: in a control condition, the slide show made salient the threat posed by accidents and mishaps (e.g. electrocution); in the other condition, the slide show made salient the threat posed specifically by infectious pathogens. Participants then completed a task that assessed their interest in attracting, to Canada, immigrants from a variety of countries that were either subjectively familiar (e.g. Poland and Taiwan) or subjectively foreign (e.g. Mongolia and Peru). The pathogen-salience manipulation influenced responses on the immigration attitudes task: compared with the accident-salient control condition, when the threat of pathogen infection was salient, participants indicated a stronger preference for immigrants from familiar places, to the exclusion of those from subjectively foreign places [38].

Intriguingly, there is also cross-cultural evidence linking xenophobia and intergroup prejudice to worldwide ecological variation in the prevalence of pathogenic diseases. Ecological variation in pathogen prevalence is correlated with the percentage of people in a population who explicitly express intolerance for 'people of a different race' in their neighbourhood [39], and with regional frequency of ethnopolitical warfare [40]. Additionally, collectivistic value systems—which emphasize sharp boundaries between 'us' and 'them'—are especially likely to exist in social ecologies characterized historically by especially high levels of pathogen prevalence [41]. Thus, just as with sociality in general, discriminatory sociality is predicted by infection risk not only at an individual level of analysis, but also at a population level of analysis.

5. MATE PREFERENCES AND MATING BEHAVIOUR

Specific forms of social behaviour increase individuals' susceptibility to pathogen transmission. Mating behaviour is one obvious example.

Sexual contact with others exposes individuals to a much higher risk for contracting sexually transmitted diseases (e.g. syphilis). In addition, given the intimate physical proximity associated with mating behaviour, it also facilitates transmission of other pathogens as well. Sexual promiscuity therefore poses a potential problem: the more sexual encounter partners an individual has, the greater is that individual's infection risk. Of course, these infection-specific costs of promiscuity must be balanced against potential fitness benefits associated with multiple mating partners (benefits that may be especially pronounced among men [42]). Following the principle of functional flexibility, one might expect an attitudinal disposition towards sexual promiscuity to be inhibited among people who feel vulnerable to pathogen infection, whereas a preference for promiscuity may be more pronounced among people who feel relatively invulnerable to infection. Consistent with this hypothesis is a negative correlation between individuals' perceived vulnerability to infection and their endorsement of an 'unrestricted' (i.e. more promiscuous) sociosexual style [43].

This inverse relation between vulnerability to infection and preference for promiscuity may apply not just only to one's own sexual behaviour but also to preferred behavioural dispositions of potential mates. This is because one's risk of infection is a function not only of one's own sexual promiscuity but also a function of the sexual promiscuity of anyone with whom one has sexual contact (i.e. a sexually monogamous woman is at minimal risk of infection if her one sexual partner is also monogamous, but at greater risk of infection if her one partner is sexually promiscuous). Therefore, in mating contexts, the sexual promiscuity of other people can be considered a dispositional trait connoting infection risk, and, as such, it is likely to trigger an aversive response from the

behavioural immune system—especially under conditions in which perceivers feel especially vulnerable to disease. Recent empirical evidence provides preliminary support for this hypothesis: people who feel more chronically vulnerable to infection indicate a stronger preference for non-promiscuous mates, and this effect itself is especially pronounced under conditions in which the threat of infectious diseases is psychologically salient (D. R. Murray, D. N. Jones & M. Schaller 2010, unpublished raw data).

The behavioural immune system may also influence the extent to which other kinds of traits are valued in a mate. Physical attractiveness is one such trait. Subjective assessments of facial attractiveness are influenced by specific aspects of facial physiognomy that are associated with genetic quality [44]. Consequently, subjective assessments of physical attractiveness may be somewhat diagnostic not only of a potential mate's own immunological competence but also diagnostic of the immunological competence of any offspring produced by that potential mate. This line of reasoning offers a partial explanation for the high value that people place on the physical attractiveness of a mate. It also implies that the typical preference for physically attractive mates may be exaggerated even further under conditions in which people feel more vulnerable to infectious diseases. Results from a recent experiment support this hypothesis: romantic interest in physically attractive (compared with unattractive) opposite-sex individuals is exaggerated under circumstances in which the threat posed by infectious diseases is temporarily salient (A. Beall & M. Schaller 2010, unpublished raw data).

Cross-national analyses reveal conceptually similar linkages between ecological variability in pathogen prevalence and cultural differences in mating behaviour. In countries that historically have had a relatively higher prevalence of pathogenic diseases, people (especially women) report attitudes endorsing relatively more 'restricted' (i.e. less promiscuous) strategies of mating behaviour [28]. Ecological differences in pathogen prevalence also predict cultural variation in mate preferences, with physical attractiveness emerging as an especially prized attribute in populations characterized historically by an especially high prevalence of pathogens [45,46].

6. NORMATIVE AND COUNTER-NORMATIVE BEHAVIOUR

The implications of the behavioural immune system for xenophobia, discussed above, are predicated in part on the possibility that foreign peoples may be especially likely to violate local behavioural practices (e.g. hygiene rituals, food preparation norms) that inhibit the spread of infectious diseases. The potential for norm violation is not specific to foreign peoples; *anyone* might potentially engage in non-normative behaviour. And while non-normative behaviour can have substantial infection-specific costs (e.g. a free thinker who violates normative practices pertaining to defaecation may increase the infection risk of the entire local population), it is also potentially beneficial (especially when that non-normative behaviour produces technological innovations and novel solutions to enduring problems). The ratio of costs to benefits is likely to be a function of the threat posed by infectious pathogens. Under circumstances in which the threat posed by pathogens is rather modest, the benefits of non-normative behaviour may outweigh the costs. But under circumstances in which people are more highly vulnerable to pathogen infection, the costs of non-normative behaviour increase accordingly, and may outweigh the benefits. Drawing on the principle of functional flexibility, it follows that the extent to which individuals favour normative versus non-normative tendencies is likely to vary depending on the extent to which those individuals feel vulnerable to infectious disease. Compared with circumstances in which people feel relatively invulnerable, when people feel more vulnerable to infection they may be relatively more conformist in their own behavioural tendencies and also less tolerant of others' non-normative behaviour. (In addition, these effects may be more pronounced in behavioural domains that are more clearly linked to the transmission of infectious diseases.)

This hypothesis has yet to be rigorously tested in laboratory research, but some preliminary evidence is supportive: people who report higher levels of chronic concern with infection also report more conformist attitudes, and people also show an increased tendency to conform to majority opinion under conditions in which the threat of infectious disease is temporarily salient (D. R. Murray & M. Schaller 2011, unpublished raw data).

In addition, there is now ample evidence linking ecological variation in pathogen prevalence to cultural variation in conformity relevant dispositions and values. The linkage between pathogen prevalence and collectivistic value systems is indirectly supportive, given that collectivism is defined in part by a higher value placed on the conservation of cultural traditions [41]. Also indirectly supportive are results revealing that higher levels of pathogen prevalence are associated with lower population-level scores on the personality trait 'openness to experience', which is associated with novelty-seeking and tolerance for inconsistency [28]. More convincing support emerges from recent findings on more focused measures of cultural conformity pressure and tolerance for non-conformity: the results reveal that, in places characterized historically by a higher prevalence of pathogens, there exist stronger cultural pressures towards obedience and conformity, as well as a reduced tolerance for non-normative behaviour [47].

7. QUESTIONS, SPECULATIONS AND DIRECTIONS FOR FUTURE RESEARCH

These lines of research reveal that the mechanisms of behavioural disease-avoidance influence a diverse set of social psychological phenomena. The behavioural immune system matters not only because of its implications for anti-pathogen defence but also because of its implications for social perception, social cognition and the social lives of human beings.

Along with these wide-ranging implications, there also emerges a diverse set of scientific questions that can only be answered through further scientific investigation.

(a) *Underlying mechanisms*

Most discussions of the human behavioural immune system assume that the psychological mechanisms that define it are likely to have been adaptive throughout long stretches of human evolutionary history [4,19,33]. This assumption does not, however, imply that each mechanism is an adaptation specific to selection pressures posed solely by infectious diseases. Many of the psychological systems employed by the behavioural immune system are likely to have evolved in response to additional selection pressures as well—some of which may have predated threats posed by infectious pathogens. An obvious example: the human behavioural immune system employs sensory organs (such as the eyes) in the service of detecting infection-connoting cues, but this hardly implies that these organs evolved specifically as adaptations 'for' anti-pathogen defence. Similarly, while the emotional experience of disgust is integral to the suite of adaptive responses associated with the behavioural immune system (and is causally linked to social outcomes such as xenophobia and the moral condemnation of norm violators [48,49]), the physiological substrates of disgust may have evolved originally as a means of facilitating the expulsion of harmful things that have been ingested orally [50]. So, while it is sensible to assume evolutionary origins for the various mechanisms that define the behavioural immune system, it is probably sensible also to assume that some of these mechanisms have ancient evolutionary origins that predate the behavioural immune system, and were adaptively 're-purposed' in response to selection pressures imposed by infectious diseases.

Any speculations about the evolutionary origins of these psychological mechanisms, and the manner in which they have been adaptively coordinated in the service of anti-pathogen defence, can be usefully buttressed by enquiry into the underlying physical substrates—at anatomical, neurochemical and genetic levels of analysis. It will therefore be useful for future research on the human behavioural immune system to follow the lead of behavioural neuroscientists who study disease-avoidant behaviour in other species. For instance, many mammals are sensitive to olfactory cues that are diagnostic of infection risk; these olfactory cues are employed to identify infected conspecifics and trigger avoidant responses. Correlates of these behavioural processes have been identified at both genetic and neurochemical levels of analysis, in the form of specific genes coding for neuropeptide, oxytocin and oestrogenic mechanisms [51].

Of course, while it is instructive to examine disease-avoidant behaviour in other animals to inform enquiry into the human behavioural immune system, it is also important to recognize elements of human psychology that are unique, and thus may have unique implications for disease-avoidant behaviour. Compared with other animals, humans have unusually massive neocortical brain structures; consequently, humans have relatively greater capacities for perspective-taking, deliberative thought and the intentional inhibition of behavioural impulses. Thus, while the perception of another person's infection-connoting features may automatically trigger aversive affective and cognitive states, these affective and cognitive states may not necessarily manifest in avoidant motor behaviour. These behavioural impulses may be muted by more deliberative cognitive processes—which, for instance, allow people to sympathize with conspecifics who are sick or disabled, and facilitate behaviour that is nurturant rather than neglectful [52]. A fuller understanding of the behavioural immune system, and its impact on human sociality, must consider its highly automated mechanisms in conjunction with the more deliberative and controllable cognitive processes that also influence human behaviour.

An additional kind of enquiry into underlying mechanisms is implied by results that test disease-avoidance predictions at a cultural rather than at an individual level of analysis. As reviewed above, many of the findings observed in laboratory experiments (e.g. the psychological salience of infectious diseases leads individuals to be less gregarious and more xenophobic) have parallels in comparisons between different cultural populations (e.g. in geographical regions characterized by high pathogen prevalence, human populations tend to be characterized by lower mean levels of extraversion and more xenophobic cultural values). It is perhaps tempting to assume that these cultural differences simply represent population-level outcomes of the same psychological processes that account for the laboratory findings (e.g. the operation of functionally flexible neurocognitive systems). But this single explanation cannot account for the full pattern of cross-cultural findings (including the fact that these cultural differences are typically predicted *less* strongly by contemporary pathogen prevalence than by historical pathogen prevalence [28,41]). A variety of additional explanatory mechanisms must therefore be considered as well, including cultural transmission processes, developmental processes and genetic selection processes too. These different kinds of explanatory mechanisms are mutually compatible with one another and each is associated with some empirical support, either direct or indirect [53]. For example, recent evidence suggests that the relationship between pathogen prevalence and cultural collectivism may be partially mediated by population-level differences in the frequency of a specific genetic polymorphism in the serotonin transporter gene-regulatory region [54].

(b) *Additional consequences for additional kinds of social behaviour*

A more complete understanding of the mechanisms that underlie disease-avoidant behaviour in humans is likely also to be accompanied by a fuller appreciation for the varieties of social behaviour that may serve a disease-avoidant function.

For instance, while chimpanzees have been observed to act aggressively towards a diseased conspecific [15], no rigorous experimental research has yet explored potential connections between the behavioural immune system and overt aggressive behaviour (in either humans or in other animal species). Aggression is a particularly intriguing behavioural response to perceived infection. To aggress physically against another individual typically requires some sort of approach rather than avoidance behaviour; and so if that individual is infected,

one's own risk of infection may be temporarily increased during the act of aggression. However, aggression may be an effective means by which to compel the infected individual to leave the immediate area, or to stay at a distance, which may functionally reduce infection risk (for oneself, and for others within the immediate vicinity) in the long term. These considerations suggest that aggression may sometimes be a behavioural outcome of the behavioural immune system, but this behavioural consequence may depend greatly on additional context-specific variables that influence the relative salience of these short-term costs and long-term benefits. In addition, given that aggressive responses to infection risks impose costs primarily on the actual aggressors, while potentially benefiting others within the immediate vicinity, aggressive responses may be highly influenced by social pressures. For example, when the threat of disease is great, normative prohibitions against interpersonal aggression may be relaxed; instead, people may be especially likely to encourage others within their social community to act aggressively against anyone who appears to pose an infection risk, or to be especially tolerant (and perhaps even rewarding) of such acts of aggression when they occur.

More broadly, within any social community, one might expect the emergence and persistence of societal norms that encourage specific individuals to engage in any kind of approach-oriented behaviour that increases the infection risk of those particular individuals while simultaneously reducing the infection risk of others in the community. For this reason, perhaps, unusually high levels of prestige and economic resources are accorded to those individuals who habitually undertake the task of attempting to cure others' infections (i.e. physicians and other 'healers'). People may be especially supportive of these societal inequities under conditions in which they personally feel especially vulnerable to infection.

(c) *Implications for human health and well-being*

Following the principles of evolutionary psychology, research on the human behavioural immune system is guided by logical considerations regarding specific forms of social behaviour that are likely to have either amplified or reduced individuals' risk of pathogen infection, and thus had implications for reproductive fitness within ancestral social ecologies. The bulk of this research reveals that vulnerability to infection (either real or perceived) has consequences for social cognitive and behavioural outcomes. In contrast, very little research has tested whether these social cognitive and behavioural outcomes actually do have consequences for reproductive fitness, or even whether they have measurable consequences for individuals' health. Of course, there is no necessary reason to assume that, just because a particular behavioural tendency reduced infection risk within the context of ancestral ecologies, that it also reduces infection risk in contemporary ecologies. Indeed, given the substantial changes that have occurred in social ecologies during recent centuries (e.g. demographic shifts, advances in public health infrastructure), there are abundant reasons to presume that behavioural strategies that once functioned as effective defences against pathogen infection may no longer do so [55]. It is an empirical question as to whether the behavioural outcomes produced by the behavioural immune system reduce infection risk in contemporary environments—and if so, which specific behavioural outcomes might do so. There is some evidence pertaining to the infection risks associated with gregariousness and sexual behaviour [26,56], but little is yet known about possible risk-reducing consequences of xenophobia, conformity or other behaviours implicated in this line of enquiry.

To the extent that these behavioural responses do reduce individuals' risk for infection, there are likely to be large-scale epidemiological implications as well. The size, scope and speed with which many infectious diseases become epidemic depends, in part, on the geometric properties of the social networks through which those diseases are transmitted. These geometric properties are themselves products of the social behaviour of the individuals within those networks (the number of sexual partners that people tend to have, the number of acquaintances with whom people interact socially, the frequency of those interactions, etc.). Therefore, as these behaviours vary (in response to vulnerabilities both real and imagined), epidemiological outcomes are likely to vary predictably as well. One intriguing implication is that, because of existing cultural differences in transmission-relevant behavioural attitudes [28], there may be predictable differences in the epidemic spread of emerging infectious diseases within different cultural populations. More broadly, this research suggests that the psychological mechanisms that characterize the behavioural immune system might fruitfully be included into the mathematical models employed in the service of epidemiological prediction.

Finally, it is worth considering the possibility that there may be important functional connections between psychological mechanisms employed by the behavioural immune system and the biochemical processes through which immunological defences respond to actual infection [18]. One plausible hypothesis is that sensory perception of infection-connoting stimuli may trigger a more aggressive immunological response. Results from two recent experiments provide preliminary support for this hypothesis. One experiment showed that the subjective experience of disgust influences several markers of oral immune function (e.g. increased salivary tumour-necrotizing factor alpha) [57]. The other experiment revealed that immediately after people see photographs depicting symptoms of infectious disease in others, their own white blood cells produce higher levels of the pro-inflammatory cytokine interleukin-6 in response to a bacterial stimulus [58]. The provocative implication (which raises many additional questions requiring further investigation) is that activation of the behavioural immune system may influence the functioning of the 'real' immune system too.

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