

THINK AGAIN

Insights & Perspectives

Strangers look sicker (with implications in times of COVID-19)

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Abstract

We animals have evolved a variety of mechanisms to avoid conspecifics who might be infected. It is currently unclear whether and why this “behavioral immune system” targets unfamiliar individuals more than familiar ones. Here I answer this question in humans, using publicly available data of a recent study on 1969 participants from India and 1615 from the USA. The apparent health of a male stranger, as estimated from his face, and the comfort with contact with him were a direct function of his similarity to the men in the local community. This held true regardless of whether the face carried overt signs of infection. I conclude that our behavioral immune system is finely tuned to degrees of outgroupness – and that cues of outgroupness are partly processed as cues of infectiousness. These findings, which were consistent across the two cultures, support the notion that the pathogens of strangers are perceived as more dangerous.

KEYWORDS

behavioral immune system, COVID-19, disgust, infection avoidance, outgroup, pathogen transmission, perceived health

Better the devil you know.

– Irish proverb

INTRODUCTION: THE IMPORTANCE OF LOOKING ORDINARY

Our long cohabitation with pathogens has shaped – along with an immune system designed to fight infections – a suite of psychological mechanisms that help us to avoid them. This “behavioral immune system”^[1,2] directs us away from potential sources of contamination, such as feces, foul-smelling food, rats, or individuals who already appear infected.

People who are harboring an infection tend to look,^[3,4] smell,^[5,6] and move^[7] differently from people who are not. Because misjudging a contagious person as healthy has dire consequences than misjudging

a healthy one as infected, the behavioral immune system appears to err on the side of caution.^[8,9] Hence, avoidance mechanisms end up being triggered not just by symptoms that are truly diagnostic of infection, but also more generally by physical anomalies that our reason (as opposed to our instinct) can recognize as unrelated to infection – such as obesity,^[10] facial birthmarks,^[11] and all manner of disabilities.^[12–14]

It is now widely believed^[15] that our behavioral immune system sits at the root of prejudice against people who belong to a group (outgroup) other than our own (ingroup). Of course, it might be adaptive to exclude outgroup members inasmuch as they are more likely to violate local norms and rituals that happen to hinder pathogen transmission.^[16,17] But a more radical, biologically grounded, idea is that we have evolved to prefer contact with the ingroup, over the outgroup, because we are better placed to combat parasites (here broadly defined to include all pathogens^[18]) that are widespread in our own community, having coevolved with them for longer.^[19,20] Trapped in

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what is known as the Red Queen effect (“it takes all the running you can do, to keep in the same place”^[21,22]), hosts and their parasites perpetually adapt, counteradapt, and counter-counteradapt. The strict geographical localization of such evolutionary arms races^[23] may render host defenses less effective against pathogens evolving in nearby groups — the unfamiliar pathogens that outgroup members are likely to carry with them.

Notwithstanding its immunological plausibility and vast scope of application (its relevance to other species comes to mind^[24–26]), the unfamiliar-pathogens theory has repeatedly attracted criticism. For example, it has been argued that separate bands would interact often enough for parasites to spread rapidly from one to the other, hence defying the opportunities for local host-parasite coevolution (but see **Box 1**).^[27] And individuals who avoid outsiders would reap no benefits from them (no trading, no mating, no knowledge exchange) while still contracting the diseases introduced by the group members who did interact — and acquired the pathogens, but the benefits too.^[27]

There’s no place like home

If strangers were indeed perceived as infectious, first, people should feel less comfortable when shaking hands with, or sitting close to, ostensibly healthy members of the outgroup as opposed to the ingroup. Second, because activation of the behavioral immune system produces disgust, people who feel more easily disgusted may show a stronger tendency to avoid members of the outgroup. And third, if they truly reflect an evolved component of the human behavioral immune system rather than some cultural accident, both effects should replicate across cultures. A recent large, well-designed, and thoroughly analyzed online study by van Leeuwen and Petersen^[31] tested all three predictions. None of them were supported.

The inevitable conclusion was that the behavioral immune system includes no dedicated adaptation to respond to cues of outgroup membership, such as a “nonlocal” appearance. Yet this notion is at odds with an extensive literature on pathogen avoidance being linked to prejudice against foreign-looking individuals^[1,32–36] and with the peculiarities of outgroup responses in other species, too.^[24,26] Here I intend to solve this contradiction.

In the study,^[31] Indian and American residents rated their imagined comfort with contact with a man portrayed in a photo, who either carried a pathogen cue (a severe facial rash added digitally to the image) or not. The man was either a dark-skinned Indian or a white-skinned American (**Figure 1**). So, the ingroup-outgroup manipulation was based on ethnicity; for the purpose of checking the manipulation’s validity, each participant was also asked to evaluate the depicted individual in terms of how similar he was to the people in the participant’s local community. This sort of information happens to be of extraordinary interest. For long stretches of evolutionary time, people’s behavioral immune system would have been engaged by the parasites of individuals living in the same or separate bands. Any selective pressure on it would have come from folks encountered more or less often — individuals belonging to the local community (with their familiar germs) as

Box 1: Why Nonlocal Pathogens Spell More Danger

The findings I report here endorse the principle (nonlocal parasites are treated as though they were more dangerous) but not necessarily the underlying mechanism — coevolution between host populations and their parasites — as originally outlined by Fincher and Thornhill.^[19] For example, it has been objected^[28] that coevolution might often work in a direction contrary to that presupposed by the theory. Many pathogens are selected to spread best, or do most damage, within their current host population (i.e., to be “locally adapted”;^[29] but see^[30]) and would cause less, rather than more, trouble in neighboring groups. True, contact between separate groups has led to disastrous epidemics,^[20] but such occurrences would have been rare and only relevant to totally isolated populations, rather than to the adjoining communities typical of our evolutionary history.^[28] Yet the unfamiliar-pathogens theory could work even if coevolution entered the picture only occasionally or not at all. We may prove more vulnerable to outgroup parasites not because these are more virulent due to our maladaptation to them, but simply because numerous illnesses leave us with immune cells that respond efficiently to subsequent exposure to the same pathogens as opposed to novel ones (see also^[20,28]). It is indeed telling that, in many species, exposure to new parasites is meticulously regulated. For example, several primates keep a newcomer at the periphery of the group for weeks or months before allowing it in. This admission practice not only makes it likely that any latent infection will reveal itself, but also ensures lengthy low-level exposure to the alien pathogens — permitting residents to develop immunity to them before the stranger carries them into the group in large numbers.^[24,25]

opposed to other, gradually more distant groups (with their increasingly unfamiliar appearances and germs). Genetic distance between humans increases neatly with geographical distance, and this holds for long distances as well as for short ones.^[37] Because facial traits are largely based on genetically coded information, their degree of unfamiliarity can thus effectively serve as a cue of a stranger’s geographical distance — and with it, of one’s unfamiliarity with the stranger’s parasites.

In this paper I reanalyze van Leeuwen and Petersen’s^[31] data, defining “outgroupness” as dissimilarity from the individuals in the local community. I show that, under this more ecological light, the data are clearly in favor of the idea that the behavioral immune system features a specific adaptation to avoid outgroups. As it turns out, this adaptation senses more than the conventional, binary difference between ingroup and outgroup: it is finely tuned to *degrees* of outgroupness. Importantly, the effects of outgroupness on discomfort with close contact are partly



FIGURE 1 Examples of the type of stimuli used in van Leeuwen and Petersen's^[31] study. Participants saw the face of a White (left panels) or Indian (right panels) man without (top panels) or with (bottom panels) a severe rash. Participants indicated (on a scale from -5 to $+5$, where 0 was "neutral") how comfortable they would feel about shaking hands with, and sitting next to, the man in the photo. The two responses were averaged to form a measure of "comfort with contact." Two additional questions were "manipulation checks" meant to control whether the ingroup-outgroup and pathogen-cue manipulations had worked as intended. These were: "Does the man look ill or healthy?" on a scale from -5 (very ill) to $+5$ (very healthy), and "Does this man look like the men in your local community?" on a scale from 0 (very different from the men in my community) to 10 (very similar to the men in my community). Both checks produced the expected result: faces with the rash tended to look less healthy, and faces of a different ethnicity tended to look less familiar. After being used to confirm manipulation validity, the responses to these questions were not considered further.^[31] The faces portrayed here have been created digitally for purposes of illustration, by morphing real faces presented in the study. Original photos used for the morphs: Center for Vital Longevity Face Database^[38]; courtesy of van Leeuwen and Petersen. Image copyright by Paola Bressan

mediated by the outgroups being perceived as sicker. As we will see, these findings plainly support the unfamiliar-pathogens theory over all the alternatives.

LOOKING AT THE DATA

What's in a face

On comfort-with-contact ratings, van Leeuwen and Petersen^[31] ran an ANOVA whose between-subjects factors were Group (ingroup, outgroup), Pathogen cue (yes, no), and Country (USA, India). As expected, more discomfort was felt at the idea of touching a man when he had a rash on his face than when he had not. Remarkably, however, no significant effect emerged for Group ($F < 1$): whether the stranger shared one's ethnicity or not appeared irrelevant. Group did interact with Pathogen cue, but in an odd way: rather than feeling less comfortable with outgroup (than with ingroup) men with the rash, people felt more comfortable with outgroup (than with ingroup) men without it. These results dealt a blow to the notion that the behavioral immune system tracks cues of outgroup membership for the purpose of avoiding unfamiliar parasites.

I re-ran exactly the same ANOVA, but redefined Group in terms of similarity to local men (below the median, above the median) rather than ethnicity (same, different). Now Group had a striking effect on comfort with contact, $F(1, 3611) = 191.41$, $P < 0.0001$: people felt less comfortable touching outgroup members, whether or not they appeared visibly contagious. (Means were separated by over half a standard deviation, Cohen's $d = 0.6$ — an effect size larger than that of the difference in weight between men and women.^[39]) Group interacted with Pathogen cue too, $F(1, 3611) = 7.12$, $P = 0.008$, but this time the interaction was indeed consistent with the parasite-avoidance account, since it was driven by the rash causing a steeper decrease in comfort with outgroup — as opposed to ingroup — men. Country participated in no interactions, indicating that the effect of outgroup membership on comfort with contact replicated across USA and India (as also shown by separate ANOVAs: both P s $< .0001$).

For the sake of comparability with van Leeuwen and Petersen's ANOVA, here similarity to local men was dichotomized by a median split. However, this is a continuous variable — let's call it "outgroupness" — and such continuity is of great relevance from a theoretical standpoint too. Not only is it the case that our ancestors, traveling primarily by foot, would have been unlikely to come across people of other "races"; but genetic variation between neighboring groups would have been then, as it largely remains now, geographically graded rather than sharp. For this reason, I ran all subsequent analyses with outgroupness as a continuous variable.

A multiple regression analysis showed that participants' imagined comfort with touching a stranger increased with the stranger's similarity to men in the participants' local community (i.e., decreased with his outgroupness: $\beta = .27$, $P < 0.0001$), diminished if the stranger had a rash on his face ($\beta = -.28$, $P < 0.0001$), and

tended to be negligibly higher for Indian than for American participants ($\beta = .03$, $P = 0.030$). Differences between slopes were explored by examining interaction effects (as in^[31], the variables rash vs no-rash and India vs USA were coded by dummy variables). To keep the number of independent variables as small — and hence the error term as large, and the analysis as conservative — as possible, I added only one interaction term at a time, removing it before adding the next.

Comfort with contact decreased more steeply with the stranger's outgroupness if he had a rash ($r = .32$; **Figure 2**, left panel, solid symbols) than if he had not ($r = .26$; **Figure 2**, left panel, open symbols: similarity \times rash, $\beta = .11$, $P = 0.0004$). That is, people were more wary about pathogen cues in outgroup than in ingroup members. Neither similarity nor rash interacted significantly with participants' nationality, indicating that the effects of these two variables replicated across countries.

Comfort with contact with a stranger was a continuously increasing function of his similarity to locals — that is, it diminished with his outgroupness, $r = -.34$, $P < 0.0001$, $N = 3619$. Such a relationship held whether people looked at faces of the same ethnicity as their own ($r = -.43$, $P < 0.0001$, $N = 1816$) or of the other ethnicity ($r = -.31$, $P < 0.0001$, $N = 1803$).

The behavioral immune system is expected to steer us away from individuals who appear ill and might thus be contagious. When not enough information is coming our way (i.e., the other is neither the picture of health nor obviously sick), it is safest to assume illness and behave accordingly. But if the parasites of strangers are more dangerous to us, the assumption of illness should be stronger for outgroup than for ingroup members. That is, outgroup members ought to be perceived as less healthy.

I tested this prediction with a multiple regression identical to the previous one, except that this time perceived health (as measured by the question "Does the man look ill or healthy?") replaced comfort with contact as a dependent variable. As one would expect, strangers with a rash on their face were seen as less healthy (rash: $\beta = -.48$, $P < 0.0001$). More interestingly, strangers who looked less similar to the men in the participants' local community appeared less healthy too (**Figure 2**, right panel: similarity: $\beta = .26$, $P < 0.0001$). Participants' nationality played no role (country: $\beta = -.006$, $P = 0.637$) and was thus removed from the regression model. In a critical twist, adding the interaction term of similarity by rash revealed that the perceived health of a stranger decreased more steeply with his outgroupness if he had a rash ($r = .35$) than if he had not ($r = .24$; similarity \times rash, $\beta = .12$, $P < 0.0001$). That is, people interpreted the same pathogen as more severe — more detrimental to health — in outgroup than in ingroup members.

The unfamiliar-pathogens idea ought to predict that the negative relationship between outgroupness and comfort with contact is partly mediated, or moderated, by the outgroup faces appearing unhealthier. This should be the case both when the stranger features a conspicuous pathogen cue (manifest infection) and when he does not (assumed infection). I tested these predictions via separate linear regressions. Added to the model, perceived health decreased the contribution of

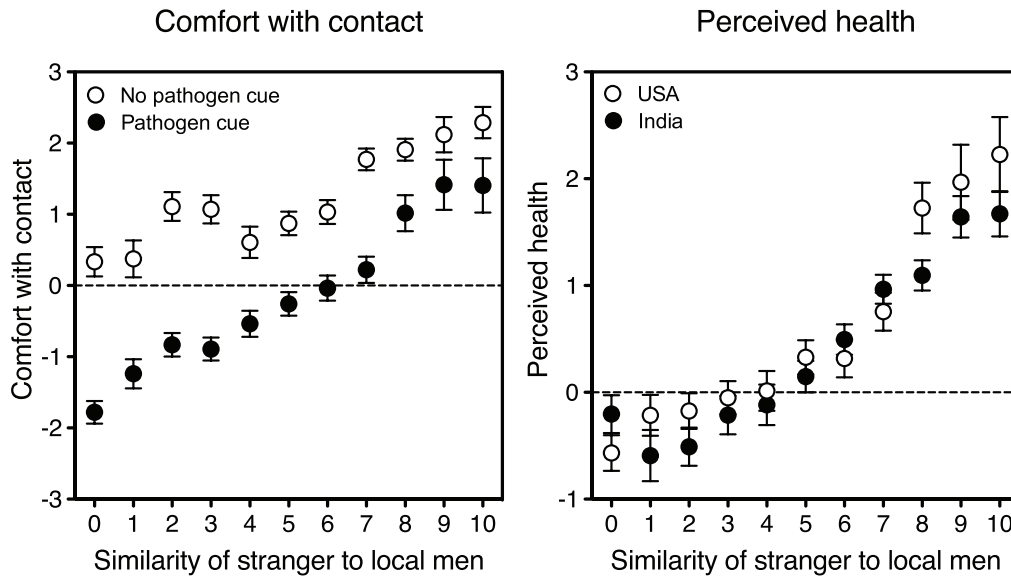


FIGURE 2 Left panel: Comfort with contact with a male stranger who either has a rash on his face (solid symbols) or not (open symbols) as a function of how similar he looks to the men in the participant's local community. Right panel: Perceived health of a male stranger as a function of how similar he looks to the men in the local community, separately plotted for USA (open symbols) and India (solid symbols) participants. Error bars indicate one standard error of the mean

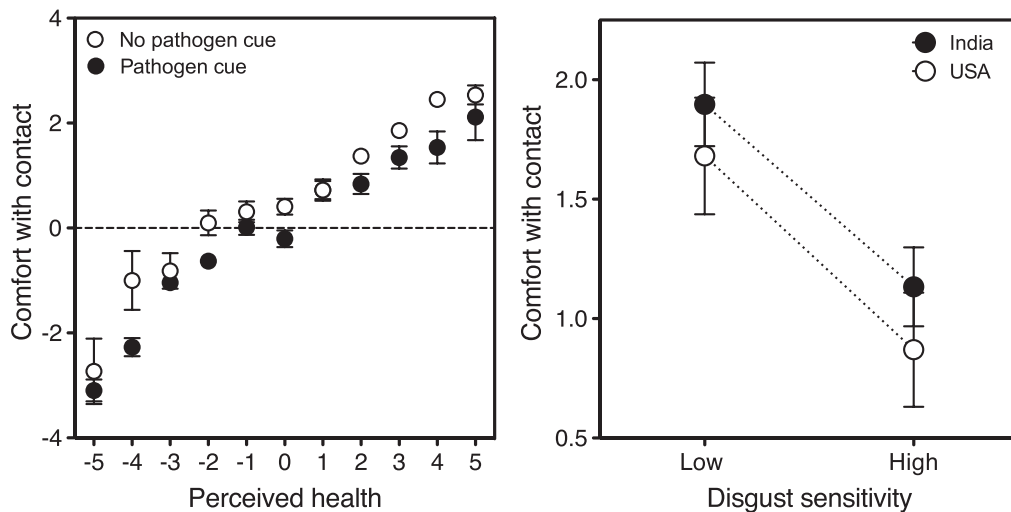


FIGURE 3 Left panel: Comfort with contact with a male stranger as a function of his perceived health, as estimated from his face. The healthier the stranger appears, the more comfortable people feel with touching him or sitting nearby, whether he has a visible rash (solid symbols) or not (open symbols). Note incidentally that, health ratings being equal, the rash consistently reduces comfort (solid symbols tend to sit lower than open ones), suggesting a further unconscious component to contact comfort that is not picked up by health ratings. Right panel: Comfort with contact with an Indian stranger with no facial pathogen cue as expressed by participants in the bottom ("low") and top ("high") tertiles of pathogen disgust sensitivity. The figure illustrates how being prone to disgust decreases comfort with touching an ostensibly healthy person not only in the USA, as already shown by van Leeuwen and Petersen,^[31] but in India too. [Section "Two shades of disgust (black and white)" explains why the Indian face is the only no-pathogen-cue condition in which India and USA data can be meaningfully compared.] India (solid symbols): participants who identified themselves as South Asians. USA (open symbols): participants who identified themselves as White. Means are covariance-adjusted (i.e., the stranger's similarity to local men and his perceived health are controlled for). Error bars indicate one standard error of the mean

outgroupness and became the main predictor of comfort with contact (see Figure 3, left panel), whether the face had a rash (similarity: $\beta = .19$; health: $\beta = .37$) or not (similarity: $\beta = .16$; health: $\beta = .38$). These results suggest that cues of outgroupness in a man's face are partly processed as cues of infectiousness.

Two shades of disgust (black and white)

If strangers are seen as health hazards even when they are not manifestly ill, people who are especially disgusted by pathogens would be expected to shun "healthy" strangers more than do people who are less

prone to disgust. White Americans' comfort with touching Indians was indeed reduced by pathogen disgust sensitivity, but Indians' comfort with touching Whites was not. This mixed result appeared to lend no support to the unfamiliar-pathogens account.

However, the Indian faces meant as ingroup members for Indian participants had dark skin, on the apparent assumption that the participants themselves would be dark-skinned too. Yet in India skin color ranges from white to black, partly depending on caste. The higher their caste, the genetically closer Indians tend to be to the (light-skinned) West Eurasians who invaded India early in its history.^[40] The lower their caste, the closer they tend to be to the tribal people (as dark as the darkest Africans) who presumably populated India before then.^[41] Because of the hot monsoon climate, infection risk has been high in India throughout its history. The caste system may thus have emerged partly as a way to reduce the exposure of the West-Eurasian invaders to pernicious infections to which local people had acquired tolerance.^[20] Today, also because the lowest castes are normally stuck with the dirtiest jobs, this system may continue to minimize the pathogen exposure of the invaders' light-skinned descendants. Indeed, in a malevolent and poorly disguised hint to infection risk, the typically dark-skinned outcaste Dalits are also known as "the untouchables."

In India, therefore, white faces should not activate disgust mechanisms nearly as much as dark faces do, if at all. This asymmetry introduces a large confound in the Indian data: other-ethnicity strangers (light skinned) are historically far less likely to be sources of pathogens than are same-ethnicity strangers (dark skinned). Compounding the problem, light skin — because of its connection with high caste and thus status and wealth — is probably associated today with greater internet access. Light skin may therefore be more common among the Indian participants in this online study than it is among Indians generally. Indeed, far more Indian than American participants pronounced themselves to be well-off (respectively, 70% vs 42% chose values higher than 5 on a 1–10 scale from worst- to best-off), and nearly 90% of the Indian participants described themselves as more light- than dark-skinned (selecting values up to 5 on a 1–10 scale from albino to the darkest possible skin).

To test the unfamiliar-pathogens idea in the Indian sample, then, we should look at the no-pathogen condition in which *Indian* faces were shown. Disgust sensitivity ought to decrease comfort with dark-skinned Indians with no visible pathogen cue. This was indeed the case (Figure 3, right panel), even after outgroupness and perceived health were controlled for (participants who identified themselves as South Asians: outgroupness: $\beta = -.23$; perceived health: $\beta = .34$; pathogen disgust: $\beta = -.14$, $P = 0.0008$). In the US, where this unique confound is absent, people proner to disgust felt instead less comfortable with touching "healthy" strangers whichever the strangers' ethnicity (participants who identified themselves as White: outgroupness: $\beta = -.30$; perceived health: $\beta = .27$; pathogen disgust: $\beta = -.12$, $P = 0.018$ for White faces; outgroupness: $\beta = -.17$; perceived health: $\beta = .38$; pathogen disgust: $\beta = -.17$, $P = 0.001$ for Indian faces).

THE FAR VIEW

Here I have shown that the more dissimilar strangers are from the people in our local community, the more we perceive them as threats to our health. As I will now explain, this finding supports the strong version of the unfamiliar-pathogens theory and none of the current alternatives to it.

These data do not support the idea that the behavioral immune system avoids outgroup members just because they are likely to violate local anti-parasite traditions

This notion — which some deem superior to the unfamiliar-pathogens one,^[17] although Fincher et al.^[42] originally proposed that the two coexist — does predict higher discomfort with nonlocal than with local people. Such discomfort, however, would be due not to the outgroups' infectious threat, but entirely to their tendency to depart from the ingroup's practices and their protective function. There is no denying the relevance of local hygiene norms, especially those concerning food and bodily waste. Such rules have evolved to keep pathogens at bay and are thus fully expected to play some role in the antipathy towards nonlocals, who, being less likely to conform to the community's anti-pathogen behaviors, are better placed to help contagious diseases spread. Yet the traditional-norms view, by itself, is clearly unable to account for all the findings. First, it contends that nonlocals' threat has nothing to do with their harboring worse pathogens;^[17] yet I have shown that nonlocals *are* perceived as sicker and this very feeling partly mediates the compulsion to avoid them. Second, this perspective fails to explain why American participants did not feel less comfortable with touching Indians as opposed to Americans (no effect of ethnicity^[31]), even though it is exceedingly likely that Americans perceive the hygiene practices of Indians as more different from their own than those of fellow Americans. The unfamiliar-pathogens theory has no problem with ethnicity being ineffective *per se* — because the primary evolutionary pressure for outgroup avoidance is assumed to come from lack of familiarity, not from a cultural mismatch.

These data do not support the idea that the behavioral immune system requires socially transmitted information about the outgroup's association with infections

This line of thought maintains that outgroup membership is only weakly, *if at all*,^[43] interpreted as diagnostic of a pathogen threat — on account of evidence (furnished, however, by a series of mutually inconsistent studies^[43]) that people are afraid of strangers solely if these come from countries explicitly described as "wracked with infectious diseases." No information of the kind was provided in van Leeuwen and Petersen's^[31] study. As to preexistent associations learned by participants in real life, while light-skinned Indians well associate dark

(hence, “outgroup”) Indian faces with infections, for USA participants any association of nonlocals with infections would arguably involve Indian faces as a category and not White faces. Yet comfort with contact depended on the perceived level of outgroupness (as opposed to ethnicity) and this applied to Indian *and* White faces both. For precisely the same reason, the socially-transmitted-information concept is unable to explain why nonlocal faces — whether Indian or White — were perceived as sicker. Note, incidentally, that the finding that people who feel vulnerable to disease are more averse to immigrants from less familiar countries^[32] squares tidily with the unfamiliar-pathogens account.

These data do not support the idea that the behavioral immune system includes no adaptation to respond to cues of outgroupness

According to this view, although our hypervigilant concern about pathogens leads us to treat any “physical or behavioral deviation from the expected phenotype” as a possible sign of infection (as suggested by Kurzban and Leary^[12]), infected outgroup members are neither particularly dangerous nor special in any other way.^[31,44,45] This notion, however, cannot explain either of the core results. Both comfort with contact with a male stranger (Figure 2, left panel) and the stranger’s perceived facial health (Figure 2, right panel) rise with his similarity to the men in the participants’ local community, and thus diminish with his outgroupness rather than remaining constant. In both panels of Figure 2, the no-adaptation-against-outgroups account would predict flat lines instead of curves.

Of course one could justify those curves by arguing that a man’s dissimilarity from the locals conveys his atypicality (“deviation from the expected phenotype”) just as easily as it conveys his outgroupness: but this is precisely the point. Outgroupness cannot be separated from atypicality because it is inferred on the basis of it. Thus, it is hardly possible to claim in the same breath that, in the context of pathogens, humans rely on atypicality *but* take no notice whatever of outgroupness. Incidentally, outgroupness seems to matter even when phenotypic abnormality does not come into play at all. For instance, revolting statements or unpleasant smells feel more disgusting when they are described as coming from an unfamiliar than from a familiar person.^[46–48]

These data do not support the idea that the behavioral immune system responds simply to individuals’ deviation from the species-typical design

It is utterly reasonable that, since parasites play havoc with the healthy phenotype, we have evolved to treat any physical deviation from the “species-typical design” as a cue of infection.^[12] Because it dispenses with the geographical implications of trait atypicality, however, this view is incapable of explaining the data unless it is supplemented by two assumptions. First, we appear to use not deviations from the species-typical design, but deviations from the locally-typical design.

Second, we treat an atypical appearance as a *stronger* potential sign of disease if a separate, conspicuous infection cue — here in the form of a severe sore — is being advertised as well. These two obligatory requirements effectively turn the species-atypicality theory (and any improvements on it, such as Petersen’s otherwise excellent “deviation from the expected phenotype” proposal^[44,45]) into the unfamiliar-pathogens one. The difference is that atypicality perspectives that dispose of outgroupness lack a functional explanation, let alone a *prediction*, of why should facial atypicality be more (as opposed to less, or equally) important when the rash is there than when it is not. The unfamiliar-pathogens theory predicts this exact result, because a rash is more likely to announce an *unknown* pathogen on a stranger than it does on a community member. From the vantage point of a defense system that remembers its assailants, the immunological novelty of a pathogen that is already colonizing someone’s face (the additional threat presented by a “sick” foreigner relative to a “sick” neighbor) is of course more ominous than the immunological novelty of a pathogen that might or might not be there (the additional threat presented by a “healthy” foreigner relative to a “healthy” neighbor).

The “out” in outgroup

These data support the idea that the behavioral immune system uses outgroupness as a cue to infectiousness (see **Box 2**). I have defined “outgroupness” as looking different from the people in the *local community*: every finding and argument I present here must be understood in light of this definition. It hardly matters what the study’s participants conjured up as their “local community” (their family, their neighborhood, their continent), and whether this meant the same to metropolitans and villagers, to Indians and Americans, to individuals surrounded by assorted ethnicities or just by one. Each of us belongs to a multitude of “local communities” varying in space and time, from the short-lived ingroups created by wearing matching clothes up to the sturdy ones defined by one’s sex and species. If the outgroup are those who look different, people from afar are outgroup, but so are people who are obese, or abnormally thin, or born with a cleft lip or port-wine stain on their face. Rather than supplanting an outgroup-detection module, the identification of phenotypic abnormality is an outgroup-detection module. That is, I am proposing that the behavioral immune system has evolved to use outgroupness (a salient, observable cue) to infer infectiousness (a mutable, unpredictable, specific-pathogen-dependent, and often invisible state). I am further proposing that outgroupness, although it expands and generalizes to all deviations from normality, is rooted in geographical separation and in the gradual lack of proximity with others — with others’ parasites, to be exact — this brings about.

Using outgroupness as a cue to infectiousness affords several advantages. First, outgroupness is salient: we are equipped with an exquisite ability to spot the odd one out — the something or someone that deviates from the rest of the group. Our sensory, perceptual, and attentional machineries are mainly built to detect deviations (and we may be more sensitive to them when our behavioral immune system

Box 2: Friends with Benefits

Every conspecific can transmit pathogens. One can avoid some of one's conspecifics all of the time, and all of them some of the time, but one can't avoid all of them all of the time. So, a balance must be struck between the risk and the need of engaging with others. The system dedicated to finding this balance appears to draw on various strands of information, such as whether others are kin, or suitable sexual mates, or likely to reciprocate our benevolence at some future time ([49,50]; see also [51]).

A cost-benefit analysis may predict that sick ingroup members should not be ostracized but helped.^[19,25] An intriguing finding that has been used against this notion is that, when asked to recall who said what, white participants confused dark Indians with Whites more often when the latter had a rash photoshopped on their face.^[44] This has been taken to imply that people place *healthy outgroup* members and *infected ingroup* members in the same mental category.^[44] If the unfamiliar-pathogens account were correct, the reasoning goes, the two should instead be carefully sorted out: potentially sick outgroup members ought to be marked "avoid" and manifestly sick ingroup members "help with caution." But the evidence of this differential treatment comes up very clearly indeed when one asks the right question. The left panel of Figure 2 shows that healthy outgroup members and infected ingroup members do tend to be regarded unequally, and this disparity's size and direction depend on the degree of in/outgroupness. Of particular interest is the comparison between the endpoints – strangers whose similarity to the local community members was rated 0 or 1 (farthest outgroup, healthy: two leftmost open symbols) versus 9 or 10 (closest ingroup, infected: two rightmost solid symbols). As is clear in the figure, comfort with contact was significantly *lower* for healthy outgroup members than for infected ingroup members, $F(1, 333) = 8.38, P = 0.004$. Also note the relatively high contact comfort with *infected* members of the close ingroup (rightmost solid symbols); this peculiarity lends credibility to the concept that disgust and empathy ("avoid" and "help with caution") work side by side in shaping decisions about approach and avoidance.^[52]

The notion that the distance we keep from others reflects a trade-off between the costs of infection and the benefits of interaction was part of the parasite-stress explanation of human behavior all along.^[19,53] Subsequent theorizing, however, has emphasized the supposedly larger benefits expected from, or "interpersonal value" placed on,^[50] close individuals as opposed to distant ones – on the apparent assumption that their pathogens yield identical costs. Yet if avoidance of strangers revolved solely around their fewer perks (their infection threat being equal to neighbors'), it is odd they should look *sicker* – as opposed to simply less attractive, interesting, or likable. But suppose for a moment that the net profit expected from others is translated into their perceived health. The benefits of strong ties with the ingroup (which include, prominently, support during illness) are arguably higher in regions with more parasites,^[19] such as India compared to the USA. So the "apparent-health-as-a-proxy-for-benefits" perspective would appear to predict that close ingroups look healthier in India than in the USA, but this is not the case (Figure 2, right panel, rightmost symbols: solid symbols do not sit higher than open ones). The similarity between India's and USA's curves comes as less of a surprise if estimated illness represents not expected profits but, in fact, estimated illness, and thus the expected costs of contracting the underlying pathogen – which go up with its unfamiliarity, in line with immunological wisdom (Box 1). As to the idea that our attitudes toward others are shaped by "perceptions of interpersonal value rather than perceptions of infectiousness,"^[50] it fails to explain why such attitudes, in the absence of pathogen cues and regardless of others' "interpersonal value," depend (a) on one's pathogen disgust sensitivity (Figure 3, right panel) and (b) on how sick others appear (Figure 3, left panel).

is activated^[54]. Second, an outgroupness rule of thumb spares individuals from the risks of having to learn the visible symptoms of each novel infection before activating countermeasures such as social distancing. (The uncontacted tribes that have managed to survive, like the Sentinelese of North Sentinel Island, are those that have made it abundantly clear that outsiders are *never* welcome.^[55]) And of course, not all pathogens advertise themselves in ways we have evolved to recognize, in which case no telltale symptoms will be there to be learned. The behavioral immune system does feature a collection of automatic responses to signals that, over evolutionary time, were statistically associated with disease. Pallor, red eyes, a tired expression are interpreted as indicators of sickliness.^[3] Yet, unlike outgroupness, such cues convey no information about a pathogen's potential harmfulness – that is, the likelihood we have encountered and defeated it before. Note that deviations from the species-typical design do not pass on that information either.

Third, outgroupness is a supremely adaptable yardstick. Outgroupness is based on the looks (or, depending on the species, the scent or other chemical cue) of the conspecifics to which one is habitually exposed – the current "local community." Because of this plasticity, it permits individuals to adjust their defenses to capricious circumstances. Should our local community – with its familiar parasites – change, so should the ingroup/outgroup label we attach to others. A response to outgroupness, unlike a response to mere deviations from the species-typical design, keeps us up to speed.

IT'S A SMALL WORLD

Unfamiliar faces look sicker; familiar ones look healthier; and during a pandemic the ramifications of neither proposition look good. In times of COVID-19, the only comforting thing that can be said of these

findings' implications is that they are scientifically very interesting. The sicker our fellow humans appear, the more we feel compelled to keep them out of the way — which we do also by becoming unkind, prejudiced, intolerant, and aggressive. There is no escaping the pressure of an infection threat as formidable as COVID-19 on millions of behavioral immune systems, to the effect that our prospects for world peace and universal harmony are unlikely to take an upturn anytime soon.

Yet the other side of the coin — familiar faces seem healthier — is hardly a compensation when the pathogen we are up against is entirely novel. In unremarkable times the special intimacy we enjoy with our ingroup serves us quite nicely, because we are better adapted to their parasites than to the outgroup's. No human, however, is pre-adapted to viruses that jump out overnight from bats or camels or pangolins^[56] and quickly proceed to invade a globalized and hyper-connected world. Being ingroup or outgroup matters no longer: but not in the sense that one would have hoped for. And of course our ingroup-loving instincts continue to run as blindly as they always have done. Impressively, comfort with contact with familiar-looking individuals was higher than the neutral point even in front of a glaring contagion cue (infected ingroup: left panel of Figure 2, rightmost solid symbols). That is, provided they look like community members, unmistakably infectious strangers appear fine to shake hands with and sit close to; strict proximity to them feels more comfortable than uncomfortable. It is not a matter of contagiousness being misconstrued either, because the very same signs keep us well away from those who look different from the locals (infected outgroup: left panel of Figure 2, leftmost solid symbols). With a pandemic ongoing — let alone one that presents with a lack of obvious symptoms^[57] rather than with a disgusting facial rash — these findings are disquieting.

The behavioral response of individuals to an epidemic is capable of altering its dynamics with catastrophic consequences.^[58,59] The most effective measure to stop or slow down the spread of airborne diseases like COVID-19 is to avoid person-to-person proximity (in conjunction with wearing masks whenever spatial or temporal separation is less than ideal,^[60] which in places shared with others means virtually *always*^[61,62]). In the face of infection, social distancing is practiced in nature by mostly every species except superlatively social ones, such as bats^[63] and mongooses,^[64] where group members are connected so tightly that isolation might prove undesirable, and pathogen exposure is inevitable. Of course, spontaneous social distancing is driven by the detection of physical or behavioral signs of infection, which in humans is largely unconscious^[5] and not necessarily accurate. For example, we are unable to judge from the sound alone whether a cough comes from someone who is infected or not; disgusting coughs appear more alarming regardless.^[65] Here I have shown that identical infection cues can be perceived as more or less threatening, and lead to a stronger or weaker avoidance response, depending on whether they show up on unfamiliar or familiar faces. A weaker avoidance response translates, needless to say, into reduced spontaneous social distancing and reduced compliance with enforced social distancing.

Mathematical models of human epidemics have begun to recognize that not everyone in a population has an equal chance to become infected or infect others. Infections propagate primarily through

networks that, being formed by individuals who are habitually in contact, tend to be clustered in space.^[66] Yet, unless one is modelling the inhabitants of North Sentinel Island, there also exist rare random links to distant individuals ("small-world" networks^[67]), which permit infection to expand relatively quickly — allowing indeed for multiple epidemics or even pandemics. The findings I have presented hold two implications for disease transmission. First, individuals are more likely to infect and be infected by community members, as opposed to nonmembers, not only because they meet them more often and for a longer time,^[68] but also because — on account of perceiving them as healthier and hence safer — they are bound to take fewer precautions upon meeting them. And second, individuals are more likely to infect, and be infected by, strangers who are not even community members but just look similar to them. This bears evident potential repercussions on contagion patterns: predicting as it does, for instance, that at the start of the COVID-19 outbreak in the USA white Americans might have been less guarded toward (possibly infected) white European tourists than toward healthy fellow Americans of African origin. Thus, perceived familiarity effectively reduces the distance between individuals within a network, changing their probability of both acquiring and transmitting infection. Incorporating properties such as familiarity or outgroupness to alter the weight of the links between individuals may increase models' realism, with immediate relevance to epidemiology.

I have proposed that facial familiarity decreases infection cues' perceived threat by serving as a proxy for previous exposure to the same pathogens. Mandrills abstain from grooming contagious mates unless these are close maternal kin (mother, offspring, maternal half-siblings) and it has been suggested they might be less sensitive to infection cues associated with kin.^[69] Note, however, that they treat infected *paternal* half-siblings exactly as they treat infected distant kin or nonkin, even though paternal half-siblings are every bit as related to them as are maternal ones, and can be recognized as kin.^[70] Yet of course maternal half-siblings are exposed to one another a great deal because they are raised together from birth, whilst paternal half-siblings grow up in entirely different families.^[70] I suggest, then, that mandrills' disregard of contagion when attending to maternal kin might reflect not their genetic relationship, but their larger familiarity with them and hence with their parasites.

CONCLUSIONS: TO THE BEHAVIORAL IMMUNE SYSTEM ALL STRANGERS ARE EQUAL, BUT SOME STRANGERS ARE MORE EQUAL THAN OTHERS

The results described in this paper fit effortlessly what I have called the "unfamiliar-pathogens" theory — a facet of the general parasite-stress explanation of human behavior (^[19,53]; see also^[16]). Building upon new data, here I have unpacked the basic idea and stretched it slightly in depth and breadth. I have argued that our treacherous cohabitation with parasites has forced on us the compulsion to assess others' dissimilarity from the people to whom we are usually exposed (our quintessential "ingroup"). Individuals who do not look like them are

likely to be coming from elsewhere, carrying pathogens that are novel to us and thus more dangerous. Therefore, our behavioral immune system has specifically evolved to pay “outgroups” the greatest attention, perceiving them as sicker from the start. And because the members of our local, familiar community embody “normality,” and outgroupness is detected as a deviation from that, our aversion generalizes to all deviations from normality. This can only deepen and widen our disinclination to engage with people who are (or we perceive as) malformed, disfigured, disabled, or just “strange” — anomalies that happen to be statistically associated to disease on their own merits.

If the idea laid out here is correct, discomfort with contact should not be confined to actual strangers or atypical individuals but extend to familiar, ordinary-looking community members we seldom bump into. Indeed, when 30,000 people from 165 countries were asked who was the least likely person they would share a toothbrush with, 2% indicated their spouse, 25% the boss at work, and 60% the postman.^[71] Our spouses, bosses, and postmen do not represent increasing degrees of outgroupness in terms of which tribe or village or ethnicity they belong to. They do, however, in terms of how regularly they happen to lavish their own parasites on us.

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CONFLICT OF INTEREST

The author declares no conflict of interest.

DATA AVAILABILITY STATEMENT

All data, analysis scripts and annotated outputs are publicly available via the Open Science Framework and can be accessed at <https://osf.io/qx74g>. The data originally posted by F. van Leeuwen and M. B. Petersen, along with their own analyses and the variables’ descriptions, can be found at <https://osf.io/md7nb>.

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