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Healthy Out-Group Members Are Represented Psychologically as Infected In-Group Members

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Abstract

A range of studies have demonstrated that people implicitly treat out-groups as the carriers of pathogens and that considerable prejudice against out-groups is driven by concerns about pathogens. Yet the psychological categories that are involved and the selection pressures that underlie these categories remain unclear. A common view is that human pathogen-avoidance psychology is specifically adapted to avoid out-groups because of their potentially different pathogens. However, the series of studies reported here shows that there is no dedicated category for reasoning about out-groups in terms of pathogens. Specifically, a memory-confusion experiment conducted with two large-scale samples of Americans (one nationally representative) yielded strong, replicable evidence that healthy out-group members are represented using the same psychological category that is used to represent manifestly infected in-group members. This suggests that the link between out-group prejudice and pathogen concerns is a by-product of general mechanisms for treating any unfamiliar appearance as an infection cue.

Keywords

out-group prejudice, pathogen avoidance, disgust sensitivity, social categories, open data, open materials

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There is increasing evidence that xenophobia and people's prejudice toward the members of groups other than their own are partially driven by psychological motivations designed to avoid pathogens. Out-group members appear to be implicitly tagged as pathogenic risks, especially by individuals with high sensitivity to such risks, and as a result elicit a number of protective behaviors relating to stigmatization, avoidance, and active ostracism (e.g., Aarøe, Petersen, & Arceneaux, 2017; Faulkner, Schaller, Park, & Duncan, 2004; Navarrete & Fessler, 2006; Tybur et al., 2016). These and related psychological processes have been established experimentally and observationally, within and outside of Western populations (e.g., Adams, Stewart, & Blanchar, 2014; Tybur et al., 2016).

Yet although there seems to be robust empirical evidence for an association between pathogen concerns and out-group prejudice, we still lack a clear theoretical understanding of why this association exists. What are the exact psychological mechanisms involved, and what evolutionary selection pressures shaped them? Some work within the psychological sciences has promoted the view that the association between out-groups and perceptions of pathogen risk reflects a dedicated adaptation (Fincher & Thornhill, 2012). Because the pathogen ecology might differ from group to group, the innate or adaptive immune system of one group might not be prepared for the pathogens of other groups; this would increase the virulence of out-groups' pathogens. Past studies (e.g., Faulkner et al., 2004; Tybur, Merriman, Hooper, McDonald, & Navarrete, 2010) have referred to the European conquest of the Americas as a case in point: Ninety percent of the native population was killed by the smallpox virus that the Europeans carried with them (Diamond, 1999). From this perspective, when contemporary individuals avoid out-groups for pathogen-related reasons, they do so because such

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avoidance has been adaptive over the course of human evolutionary history.

Given the current knowledge of ancestral migration and disease patterns, however, the European conquest of the Americas is not an optimal model for understanding the selection pressures operating on human pathogen-avoidance psychology (Aarøe, Osmundsen, & Petersen, 2016; Fessler, Clark, & Clint, 2015). First, ancestral migration happened on foot (Kurzban, Tooby, & Cosmides, 2001); hence, frequent contact with individuals from completely different pathogen ecologies was unlikely. Second, highly infectious diseases first evolved recently with the emergence of high population density (Diamond, 1999), and although different pathogen ecologies have been observed within small distances in present-day tribal societies, they have been observed only for modern disease types (Black, 1975). Finally, it is not clear that the pathogens of other groups would be more dangerous than those of the local group; local pathogens might be adapted to, and hence more dangerous to, the local host group (de Barra & Curtis, 2012).

Given these concerns, the association between out-group prejudice and pathogen concerns might reflect not an adaptation but rather a by-product or side effect. Because of the difficulties in detecting infections and the costs of false negatives, human pathogen-avoidance psychology is hyperactive and manages errors in a "better safe than sorry" manner (Tybur & Lieberman, 2016). Unfamiliar or atypical appearances are generally treated by this psychology as potential symptoms of infection. Consequently, research shows that individuals who deviate from the normal phenotype for nonpathogenic reasons (e.g., people with birthmarks, weight problems, or physical disabilities) are perceived as infected (Park, Faulkner, & Schaller, 2003; Park, Schaller, & Crandall, 2007; Ryan, Oaten, Stevenson, & Case, 2012). In modern societies, group boundaries are often correlated with racial or ethnic differences in physical appearance or behavior and, hence, could elicit this hyperactive psychology. This would not be because this psychology is specifically adapted to manage between-group relations so as to reduce pathogen risk but rather would be a side effect of its vigilance toward any type of deviation.

These two accounts—the adaptation and the by-product accounts—differ not just in terms of the selection pressures they specify but also in terms of the psychological mechanisms that they predict. In this regard, a distinction between *potential* and *manifest* infection becomes important. If pathogen-avoidance psychology is adapted to manage between-group relations so as to reduce pathogen risk, there should be a dedicated psychological category for tagging out-group members as potentially infected individuals who might carry pathogens that the local group has not been immunized against. Such individuals not only should be avoided, but also should be distinguished from manifestly infected in-group members. According to the adaptation account, sick in-group members carry less dangerous pathogens and, given the evolutionary importance of health care for band-wide risk buffering (Jensen & Petersen, 2017; Sugiyama, 2004), would have been key targets of help rather than avoidance (Fincher & Thornhill, 2012; Steinkopf, 2017). As argued by Fincher and Thornhill (2012), pathogen concerns trigger not only out-group avoidance but also in-group affiliation "for coping with present infections in members of the in-group" (p. 63). In contrast, if the association of out-groups members with pathogen threat is a by-product of the fact that differences in appearance elicit pathogen avoidance, there should be no dedicated categories for distinguishing between in- and out-group members on pathogen-related grounds. Instead, physically different out-group members should be processed in the same way as manifestly infected in-group members, even if they are healthy. Hence, according to this account, the physical appearance of an out-group member is processed as a sign of infection in exactly the same way as a rash on the face of an in-group member.

Method

These arguments imply that it is possible to discern between the adaptation and the by-product accounts by testing whether people utilize the same psychological category to mentally represent manifestly infected in-group members and healthy out-group members. Should this be the case, there is reason to infer that the perception of out-group members as pathogen threats is an evolutionary by-product of a psychology designed to avoid manifestly infected individuals in general.

To test this possibility, I used a memory-confusion protocol, the "who said what?" (WSW) paradigm. This protocol is specifically geared toward testing whether impressions of two different persons are formed by the same or different psychological categories (Taylor, Fiske, Etcoff, & Ruderman, 1978) and has previously been used to test adaptive and by-product accounts of group psychological processes (Kurzban et al., 2001; Pietraszewski, Cosmides, & Tooby, 2014). To ensure replicability, I conducted two experiments using identical surveys. In Study 1, the survey was administered to a socially diverse sample of Americans using a crowdsourcing platform, CrowdFlower (N = 594). (Because the goal was to collect data from 600 participants, payments for this number of participants were provided, but for unknown reasons, 6 individuals were able to take payment without completing the study.) With a sample of this size, even effects of a relatively small

size should be identifiable. To increase external validity in Study 2, I surveyed a representative sample of White Americans, through the services of the YouGov survey agency (N = 607). Although the WSW paradigm was originally designed to be implemented in laboratory settings, I utilized an abbreviated version that can be implemented in online surveys of the general public (Petersen, 2012).

The WSW paradigm is organized in three distinct phases. First, participants are presented with a slide show that introduces eight individuals, one at a time. The photo of each individual is presented along with a statement made by the individual, and participants are asked to simply pay attention. Second, participants engage in a distractor task. Third, they are presented with a surprise recall task. One by one, each statement from the presentation phase is presented with the eight photos, and participants are asked to indicate the individual who made the statement (i.e., "who said what?").

In the present version of the WSW paradigm, all of the statements were neutral statements that conveyed no substantial information (Pietraszewski et al., 2014). The pictured individuals were four White Americans and four individuals with an East Indian background, who thus were in-group and out-group members, respectively, for the majority of participants in Study 1 and all the participants in Study 2. In both studies, participants were randomly assigned one of two versions of the experiment. In the control condition, all of the pictured individuals were healthy, with no signs of infection. In the treatment condition, although the out-group members were healthy in appearance, the in-group members were presented with significant facial rashes, an indication of infection. These rashes were applied to the original photos using Photoshop (see the Supplemental Material available online for additional information, including validation information, on the stimuli).

By analyzing the errors made in the recall phase, I was able to test whether healthy but physically different out-group members and manifestly infected in-group members were mentally represented using the same psychological category. In the WSW paradigm, if two individuals are confused during the recall phase—that is, if the statement made by one is misattributed to the other—this suggests that they were identified as alike in the presentation phase and confused in memory (Kurzban et al., 2001; Pietraszewski et al., 2014; Taylor et al., 1978). Previous research has identified a clear tendency for participants to categorize individuals along group lines in this paradigm (Kurzban et al., 2001; Pietraszewski et al., 2014; Taylor et al., 1978). For example, participants tend to confuse Black individuals with other Black individuals and White individuals with other White individuals, but not Blacks with Whites. Similarly, in the present experiment, the participants in the control condition were expected to confuse individuals along group lines. Yet to the extent that healthy out-group members fall in the same mental category as manifestly infected in-group, between-group confusion would increase in the treatment condition. Note that this is far from a trivial prediction. It implies that by making in-group members visually more similar to each other because of a shared rash, they will become psychologically less distinct relative to the out-group.

From the answers provided in the recall phase, three measures were created for each participant: the number of correct recalls, the number of within-group recall errors (confusion of a White person with another White person or of an East Indian person with another East Indian person), and the number of between-group recall errors (confusion of an Indian person with a White person). Because each response in the recall phase was necessarily a correct attribution or one of these two types of errors, all three measures could range from 0 to 8. In Study 1, the mean number of correct answers was 3.41 (SD = 2.14), the mean number of within-group errors was 2.30 (SD = 1.48), and the mean number of between-group errors was 2.19 (SD = 1.71). In Study 2, the mean number of correct answers was 2.71 (SD = 1.95), the mean number of within-group errors was 2.61 (SD = 1.49), and the mean number of between-group errors was 2.54 (SD = 1.59).

In addition to performing the WSW task, participants completed the Pathogen Disgust Scale, a measure of individual differences in attention and responsiveness to pathogen risks (Tybur, Lieberman, & Griskevicius, 2009). Higher scores reflect motivated avoidance of "substances associated with disease-causing agents in ancestral environments" (Tybur et al., 2009, p. 105; Study 1: α = .85, M = 5.13, SD = 1.03; Study 2: α = .89, M = 5.09, SD = 1.05).

Results

Figure 1 summarizes the effects of the treatment condition on the three dependent measures of recall. If out-group members are psychologically represented as manifestly infected in-group members, the addition of manifest infection cues to the photos of in-group members would be expected to trigger more confusion in the recall phase of the experiment. This is exactly what happened: The number of statements that were correctly attributed to individuals was reduced in the treatment condition relative to the control condition in both Study 1, b = −0.93, p < .001, 95% confidence interval (CI) = [−1.25, −0.58], and Study 2, b = −0.73, p < .001, 95% CI = [−1.04, −0.43].

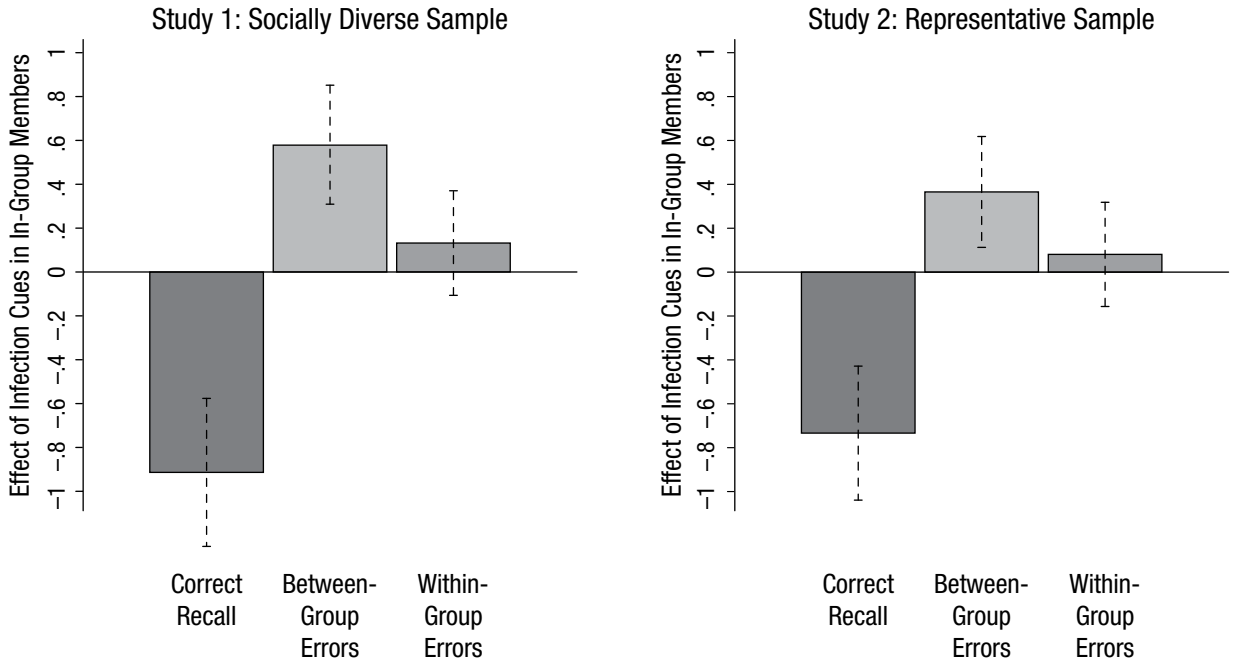

Fig. 1. Results from Study 1 (left; $N = 594$) and Study 2 (right; $N = 607$). The graphs show unstandardized coefficients for the predicted effect of the experimental treatment (presence vs. absence of manifest signs of infection in in-group members) on the number of correct responses, between-group errors, and within-group errors. Error bars represent 95% confidence intervals.

If reduced accuracy in the treatment condition is due to healthy out-group members being categorized with infected in-group members, the increased number of errors should show a particular pattern. Specifically, when in-group members show signs of infection, participants should confuse in-group members with out-group members, and, hence, the number of between-group mistakes should increase. In contrast, the overall salience of the individual groups should not increase, and, hence, the number of within-group mistakes should decrease or remain the same. In both studies, these predicted effects were obtained. The number of between-group errors increased significantly in the treatment condition—Study 1: $b = 0.58$, $p < .001$, 95% CI = [0.31, 0.85]; Study 2: $b = 0.37$, $p < .001$, 95% CI = [0.11, 0.62]. In contrast, the number of within-group recall errors was not significantly modulated by signs of infection—Study 1: $b = 0.13$, $p = .28$, 95% CI = [−0.11, 0.37]; Study 2: $b = 0.08$, $p = .50$, 95% CI = [−0.16, 0.32]. In other words, when in-group members showed signs of manifest infection, they were more often processed as belonging to the same mental category as out-group members. This suggests that this category is designed to process manifestly sick individuals and that the tagging of out-group members as pathogenic risks is a by-product of a hypervigilant system that treats deviations from the expected phenotype as signals of pathogenic risk, rather than the output of a dedicated psychological system for representing individuals who are potentially infected with pathogens that one has not acquired immunization against. From the perspective of the adaptation account, this latter category would have been crucial in managing decisions about approach and avoidance behavior: Potentially sick out-group members should be avoided, whereas manifestly sick in-group members should be targets of health care (Steinkopf, 2017). The results indicate that the human mind does not discriminate in this way.

An alternative interpretation of the results, however, is possible. The increased number of recall errors in the treatment condition could suggest that categorization was inhibited in the face of infection cues. Because pathogen-avoidance mechanisms operate via avoidance, the presence of pathogen cues might have decreased processing of the stimuli or even caused participants to look away from the stimuli. One important observation provides evidence against this interpretation: If inhibited processing of the faces with infection cues had driven the decrease in correct recall, the number of within-group errors should also have increased in the treatment condition, and this did not happen. To more completely test this alternative interpretation, however, I designed a third study, which surveyed a socially diverse sample via Amazon's

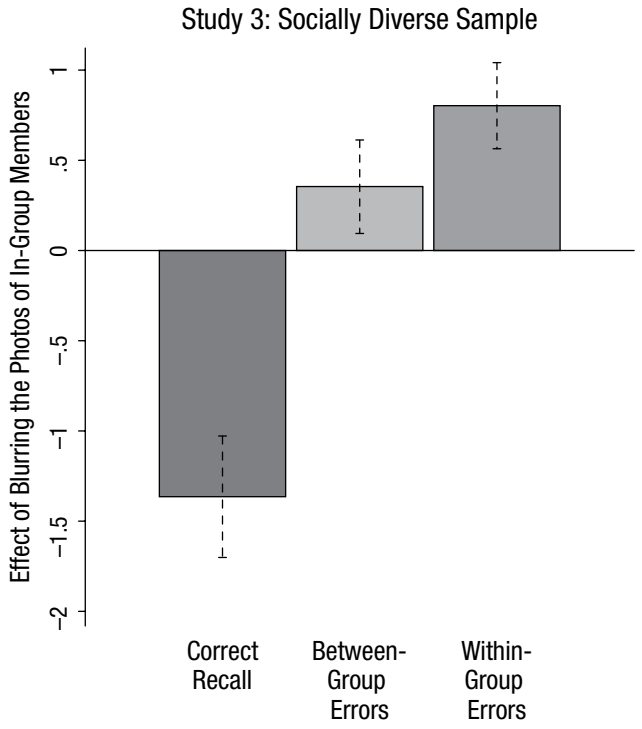

Fig. 2. Results from Study 3 ($N = 600$). The graphs show unstandardized coefficients for the predicted effect of the experimental treatment (presence vs. absence of blurring in photos of in-group members) on the number of correct responses, between-group errors, and within-group errors. Error bars represent 95% confidence intervals.

Mechanical Turk platform ($N = 600$). The design replicated the design of Study 1 and Study 2, with the important difference that infection cues were not introduced in the treatment condition, but instead the White faces were blurred during both the presentation and the recall phases (see the Supplemental Material). In this way, Study 3 experimentally induced the inhibited processing that might account for the results in Study 1 and Study 2.

Figure 2 summarizes the effects of the treatment condition in Study 3. Contrary to what the alternative interpretation of Study 1 and Study 2 predicts, induced inhibited categorization did not result in a replication of the original effects. As in the previous studies, correct recall decreased in the treatment condition, $b = -1.35$, $p < .001$, 95% CI = [−1.70, −1.03], and the number of between-group errors increased in the treatment condition, $b = 0.34$, $p = .007$, 95% CI = [0.10, 0.61]. But, in contrast to Study 1 and Study 2, the decrease in correct recall was due to a very large increase in the number of within-group errors, $b = 0.80$, $p < .001$, 95% CI = [0.56, 1.04]. This increase was significantly larger than the increase in between-group errors, $\chi^2(1, N = 600) = 5.73$, $p = .017$; when the data were adjusted for the increased baseline probability of making a between-group error (see Kurzban et al., 2001), the difference became even more significant, $\chi^2(1, N = 600) = 10.78$, $p = .001$. Thus, the results in Study 3 suggest that inhibited processing of the photos of infected individuals cannot account for the results in Study 1 and Study 2.

Finally, it is relevant to consider another alternative interpretation of the results of Studies 1 and 2: that the categorization of out-group members with manifestly sick in-group members reflects the perceived dangerousness of these people—that is, that a manifestly sick in-group member is just as dangerous as a potentially sick out-group member. First, let us consider this alternative interpretation from the perspective of the adaptation account. On the one hand, the two types might, in some abstract sense, be equally dangerous: The in-group member is certainly infected, whereas the out-group member is only potentially infected, but the in-group member's pathogens are less lethal than those the out-group member might be carrying. On the other hand, as noted in the introduction, researchers within this tradition have emphasized that potentially sick out-group members and manifestly sick in-group members are reacted to in distinctly different ways: Out-group members are to be avoided, but sick in-group members require cautious support (Steinkopf, 2017). This is why, according to this line of research, pathogen-avoidance motivations seemingly are related to both in-group affiliation (ethnocentrism) and out-group prejudice (xenophobia; Fincher & Thornhill, 2012). Hence, from the outset, this alternative interpretation is not consistent with at least some versions of the adaptation account.

Second, we can evaluate this alternative interpretation empirically by utilizing the data from the Pathogen Disgust Scale. This measure tracks individual differences in perceptions of the threat associated with disease vectors and, hence, motivations to avoid them. Individual differences in pathogen disgust did not correlate with any of the three dependent variables in the treatment condition of either Study 1 or Study 2—correct responses: $r = -.04$, $p = .49$, 95% CI = [−.14, .07] in Study 1 and $r = -.02$, $p = .77$, 95% CI = [−.13, .10] in Study 2; between-group errors: $r = .03$, $p = .58$, 95% CI = [−.08, .15] in Study 1 and $r = .05$, $p = .35$, 95% CI = [−.06, .17] in Study 2; within-group errors: $r = .01$, $p = .91$, 95% CI = [−.11, .12] in Study 1 and $r = -.04$, $p = .45$, 95% CI = [−.16, .07] in Study 2. Moreover, there was no evidence that individual differences in pathogen disgust interacted with the presence of signs of infection in predicting recall errors ($p > .40$ for each of the three interactions in each study).

Hence, independently of the danger that participants attributed to pathogens, they tended to mentally categorize healthy out-group members and manifestly infected

in-group members together. If the cocategorization of out-group members and manifestly sick in-group members simply reflects their perceived pathogenic dangerousness, individual differences in the perceived danger of pathogens should predict categorization, but they did not. The fact that individual differences in pathogen disgust did not predict categorization is less surprising from the by-product perspective. According to this account, categorization of manifestly infected in-group members with outwardly healthy out-group members is not the result of the perceived danger associated with pathogens; it is rather a result of the more basic perception of deviations from the expected phenotype, which is independent of individual differences in avoidance motivations.

Discussion

Prejudice toward out-groups partly reflects the fact that out-group members are perceived as pathogen risks. Although it is widely argued that this perception reflects a dedicated adaptation, as out-groups can carry pathogens that in-group members have not acquired immunization against, the present results constitute evidence against this view. Instead, the association between out-groups and pathogens seems to be a by-product of a general hypervigilance against deviations from the expected phenotype, such that physical (and potentially cultural and behavioral) differences are processed as signs of manifest infection in the same way as is a rash on the face of an in-group member. These findings are important, as they bridge observations of an association between out-group prejudice and pathogenic concerns with observations about human evolutionary history indicating that an adaptationist account of this association is implausible. The findings also provide an important illustration of the relevance of evolutionary theorizing for psychological science. By utilizing existing evolutionary knowledge, it was possible to specify a more well-grounded theory of the psychological processes involved in out-group prejudice. At the same time, these findings also show how psychological experiments can inform evolutionary theory. Hence, the present results provide yet another item of evidence that the pathogens from other groups most likely have not constituted a fundamental, adaptive problem over the course of human evolutionary history.

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Author Contributions

M. B. Petersen is the sole author of this article and is responsible for its content.

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Supplemental Material

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Open Practices

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