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Mark Schaller, Gregory E. Miller, Will M. Gervais,
Sarah Yager, and Edith Chen

University of British Columbia

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Abstract

An experiment ($N = 28$) tested the hypothesis that the mere visual perception of disease-connoting cues promotes a more aggressive immune response. Participants were exposed either to photographs depicting symptoms of infectious disease or to photographs depicting guns. After incubation with a model bacterial stimulus, participants' white blood cells produced higher levels of the proinflammatory cytokine interleukin-6 (IL-6) in the infectious-disease condition, compared with the control (guns) condition. These results provide the first empirical evidence that visual perception of other people's symptoms may cause the immune system to respond more aggressively to infection. Adaptive origins and functional implications are discussed.

Keywords

disease, health, immunity, perception, threat

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People are sensitive to visual stimuli connoting the potential presence of infectious pathogens in others. These stimuli include anomalous morphological and behavioral characteristics (e.g., skin discolorations, sneezing) that suggest infection with disease-causing microorganisms. When perceived, these stimuli trigger psychological responses—such as disgust and the activation of aversive cognitions into working memory—that inhibit interpersonal contact (e.g., Curtis, Aunger, & Rabie, 2004; Oaten, Stevenson, & Case, 2009; Park, Faulkner, & Schaller, 2003; Park, Schaller, & Crandall, 2007). These perceptual processes are part of an integrated set of psychological mechanisms that facilitate prophylactic behavioral defense against pathogens—a sort of *behavioral immune system* (Schaller & Duncan, 2007). Previously unexplored, however, is the intriguing possibility that these processes might also have an influence on the real immune system.

In a recent review article on disgust as a disease-avoidance mechanism, Oaten et al. (2009) suggested that “immune function, especially the innate (i.e., rapid) component, may be directly mobilized by cues that are disgust-evoking,” but also noted that “as yet there are no data in humans to confirm or refute this possibility” (p. 315). Here, we report a study that empirically tested (and supports) the specific hypothesis that

mere visual perception of other people's disease-connoting cues can cause the immune system to respond more vigorously to microbial stimuli that connote infection.

This hypothesis is plausible on functional grounds. Visual perception of other people's apparent symptoms of infection implies one's own immediate vulnerability to pathogen infection. To the extent that visual perception of such stimuli influences perceivers' own immune functioning (by causing perceivers' immune cells to respond more aggressively if, or when, such infection occurs), this response phenomenon may reduce the likelihood of the infection's becoming established.

The hypothesis is plausible on mechanistic grounds as well. There is abundant evidence that immune responses (e.g., the production of proinflammatory cytokines) can be facilitated by stressful psychological experiences. These effects are mediated by hormones such as cortisol and norepinephrine, which are released when people appraise situations as threatening, and subsequently bind to receptors on immune cells (Cohen, Doyle, & Skoner, 1999; Kiecolt-Glaser et al., 2003;

Corresponding Author:

Mark Schaller, Department of Psychology, University of British Columbia,
 2136 West Mall, Vancouver, British Columbia V6T 1Z4, Canada
 E-mail: schaller@psych.ubc.ca

Segerstrom & Miller, 2004). Moreover, visual perception of other people's disease-connoting characteristics can trigger a disgust response, which is supported physiologically through modulation of sympathetic nervous system activity. Sympathetic fibers descend from the brain into most lymphoid organs, where they release neuropeptides and neurotransmitters that can modulate immune functions (e.g., Sternberg, 2006; Webster, Tonelli, & Sternberg, 2002).

Although conceptually plausible, the hypothesis has never been directly tested by empirical data. To provide such data, we conducted an experiment in which we tested the effect that different kinds of visual stimuli have on one commonly used indicator of immunological response: the production of the proinflammatory cytokine interleukin-6 (IL-6). When white blood cells (particularly monocytes) detect foreign microbial bodies (e.g., bacteria), they secrete cellular messengers called cytokines. IL-6 is one of the cytokines released as part of this process, and it plays a key role in the subsequent inflammatory response designed to clear the body of these microbial intruders. We tested the specific hypothesis that visual perception of other people's symptoms primes the perceiver's own white blood cells to produce higher quantities of IL-6 upon contact with a bacterial stimulus.

Method

Participants and design

Twenty-eight human participants (9 men, 19 women) were randomly assigned to one of two experimental conditions. In one condition, participants watched a slide show depicting furniture (*neutral slide show*) and later, in a separate session (on a different day), watched a *disease slide show* depicting people who displayed morphological and behavioral characteristics associated with infectious diseases (e.g., pox, skin lesions, sneezing). In the other condition (designed to control for any effects due to stress-inducing threatening stimuli in general), participants first watched the neutral slide show and then, in the later session, watched a *guns slide show* depicting people brandishing firearms, most of which were aimed directly at the participants. Peripheral blood was collected before and after each slide show, allowing us to measure the effects of each slide show on white blood cells' production of IL-6 after stimulation by a model bacterial stimulus (lipopolysaccharide). We also assessed participants' self-reported emotional state following each slide show.

Procedure

Slide-show stimuli. Each slide show comprised 10 photographs, displayed multiple times, in random order. When displayed, a photograph appeared for 8 s, followed by 4 s of blank screen before the next photograph appeared. Slide shows lasted 10 min and were presented on a flat-panel LCD

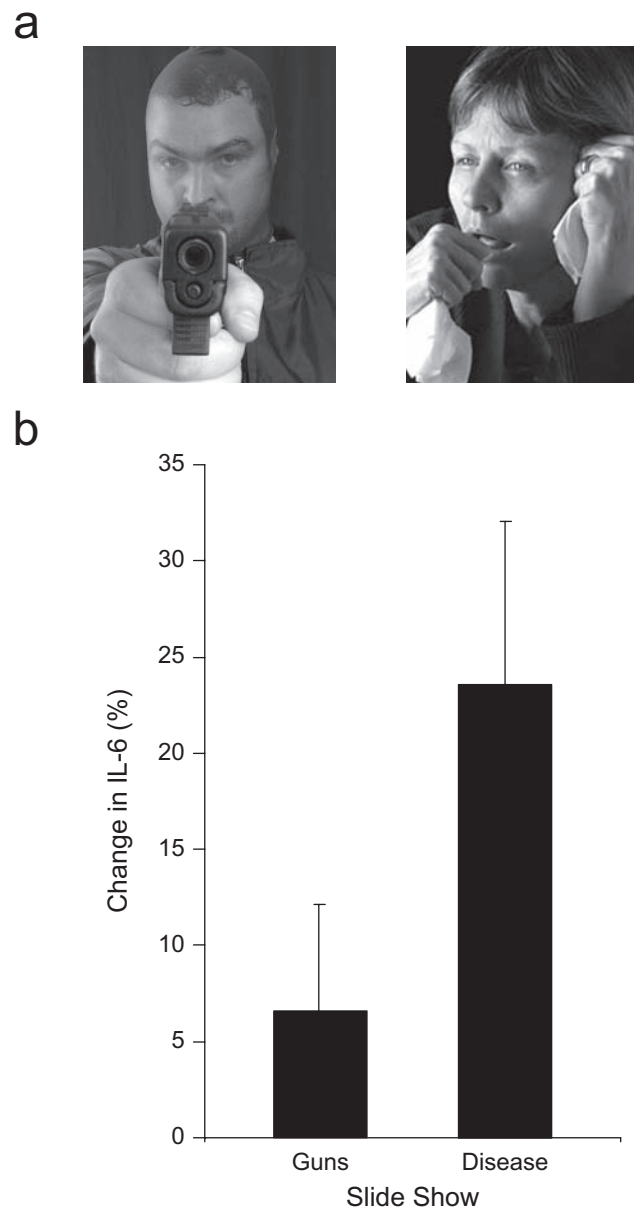


Fig. 1. Examples of the visual cues and immune response to those cues. In (a), an example of a photograph from the guns slide show is shown on the left, and an example of a photograph from the disease slide show is shown on the right. The graph (b) shows the mean percentage increase in interleukin-6 (IL-6) from pretest to posttest as a function of slide-show condition. Error bars represent standard errors.

computer monitor. Sample photographs from the disease slide show and the guns slide show are presented in Figure 1a.

Assessment of stimulated IL-6 production. Standard venipuncture techniques were used to draw approximately 10 ml of blood from participants 30 min prior to the start of each slide show (pretest sample) and again immediately after the completion of each slide show (posttest sample). Stimulated production of IL-6 was then measured using standard

immunological assay procedures (e.g., Deering & Orange, 2006; Rose, Hamilton, & Detrick, 2002; for an example in the psychological sciences, see Miller, Rohleder, Stetler, & Kirschbaum, 2005). In each sample, 200 μ l of whole blood was diluted with saline at a ratio of 10:1. The suspension was incubated with the model bacterial stimulus (0.5 ng/ml lipopolysaccharide, *E. coli* 055:B5, from Sigma Chemicals, St. Louis, MO) for 6 hr at 37 °C in a 5% CO₂ atmosphere. Supernatants were harvested and frozen at -80 °C. The samples were later assayed in duplicate for IL-6 (measured in pg/ml) using commercially available ELISA development kits (DY206E, R&D Systems, Minneapolis, MN). These kits have detection thresholds of 5 pg/ml and intra- and interassay coefficients of variation less than 5%.

Statistical analyses were conducted on an index indicating the percentage of change in stimulated IL-6 production from pretest to posttest, computed as (posttest - pretest)/pretest. This index controls for individual differences in pretest IL-6, while simultaneously normalizing (i.e., removing positive skew from) raw pretest-to-posttest change values.

Assessment of self-reported emotions. Subjective emotional state was assessed immediately following each posttest blood draw. On 5-point scales ranging from 0 to 4, participants rated the extent to which each of 18 adjectives accurately described their mood. Composite measures of four specific emotional states were computed as mean ratings of 3 adjectives each: *stressed* (stressed, tense, overwhelmed), *relaxed* (relaxed, calm, at ease), *scared* (scared, afraid, fearful), and *disgusted* (disgusted, repulsed, revolted).

Results

Did the disease slide show prime white blood cells to respond more aggressively to the bacterial stimulus? Yes. Participants' cells produced 23.6% more stimulated IL-6 after (relative to before) the disease slide show, $d = 0.74$, $t(13) = 2.78$, $p = .016$ (see Fig. 1b). These same participants showed no increase in stimulated IL-6 in response to the neutral slide show (mean change = -3.6%). Change in stimulated IL-6 was significantly greater for the disease slide show than for the neutral slide show, $d = 0.86$, $F(1, 13) = 9.74$, $p = .008$.

Did this effect occur in response to threatening stimuli in general? No. The guns slide show produced a negligible and nonsignificant increase in stimulated IL-6 (mean change = 6.6%), $d = 0.32$, $t(13) = 1.21$, $p = .249$. A 2 (condition) \times 2 (slide show) mixed-model analysis of variance (which took into account IL-6 changes associated with the neutral slide show in each condition) revealed that, compared with the guns slide show, the disease slide show produced a greater pretest-to-posttest increase in stimulated IL-6, $d = 0.63$, $F(1, 26) = 10.81$, $p = .003$.

We noted that, despite random assignment, the pretest level of stimulated IL-6 was greater in the guns condition than in the

Table 1. Mean Stimulated Production of Interleukin-6 (IL-6) and Self-Reported Mood Before and After the Guns and Disease Slide Shows

Measure	Guns slide show	Disease slide show
Stimulated IL-6		
Pretest (pg/ml)	32,002 (29,974)	22,320 (14,672)
Posttest (pg/ml)	33,964 (30,725)	26,814 (15,771)
Change (pg/ml)	1,962 (3,790)	4,494 (8,249)
Change (%)	6.62 (20.51)	23.62 (31.74)
Self-reported mood		
Stressed	1.57 (0.94)	1.24 (0.96)
Relaxed	1.62 (1.18)	1.67 (1.13)
Scared	1.38 (1.11)	0.88 (0.89)
Disgusted	1.52 (1.19)	1.64 (1.17)

Note: Standard deviations are given in parentheses. Mood was assessed after the slide show only.

disease condition (see Table 1). Does this difference reflect a failure of randomization? It appears not. In addition to the primary measures described earlier, all participants completed a battery of questionnaires assessing dispositional tendencies, including the Big Five personality traits (agreeableness, conscientiousness, extraversion, neuroticism, and openness), as well as six specific traits relevant to perceptions of threat and disease (e.g., perceived vulnerability to disease, health locus of control). On none of these traits was there a significant difference between subjects in the guns and disease conditions (all $ps \geq .10$). (Nor did any of these traits significantly predict changes in stimulated IL-6; because of these noneffects, the trait measures are not discussed further in this article.) Furthermore, the difference between slide-show conditions in pretest levels of stimulated IL-6 was nonsignificant ($p = .288$), and pretest values of stimulated IL-6 had no meaningful relation to the percentage of change in stimulated IL-6 ($rs = -.03$ and $-.18$ in the guns and disease conditions, respectively; both $ps > .54$). Most important, the significant between-conditions difference in relative pretest-to-posttest change in stimulated IL-6 (revealed by the 2 \times 2 ANOVA reported earlier) remained significant even when we statistically controlled for pretest values of stimulated IL-6 ($p = .004$).

Can this latter difference be attributed to greater subjective stress associated with the disease slide show? No. Mean levels of self-reported stress were lower following the disease slide show, compared with the guns slide show (see Table 1 for mean values on the mood measures). Subjective appraisal of stress cannot account for the greater impact of the disease slide show on facilitation of an immune response.

In addition, among participants who watched the disease slide show, self-reported disgust was inversely correlated with change in stimulated IL-6, $r = -.42$ ($p = .134$). Thus, there is no evidence that the effects on stimulated IL-6 production resulted from subjective appraisals of disgust.

Discussion

These results provide the first empirical evidence that the mere visual perception of other people's disease symptoms can cause the immune system to respond more aggressively to microbial stimuli connoting infection. It is important to emphasize that this effect was specific to the perception of disease-connoting social cues, and that it did not occur in response to a different category of stress-inducing interpersonal threat.

This linkage may have been adaptive in ancestral ecologies, as individuals characterized by perception-facilitated immune responses would have had reduced likelihood of succumbing to pathogenic infections. This immune-response phenomenon may also have had additional beneficial consequences for human social interaction. Reducing one potential cost associated with interpersonal proximity (pathogen infection) may have made it easier to reap other benefits of social groupings, such as access to material resources and protection from other threats.

Although presumably adaptive in origin, this phenomenon may nonetheless have nonadaptive (and potentially even costly) consequences in contemporary ecologies. Recent research has revealed that a wide range of morphological anomalies—even those that are not symptoms of infectious disease—can elicit emotional, cognitive, and behavioral responses that mimic those associated with the perception of disease symptoms (e.g., Park et al., 2003, 2007). To the extent that these perceptual cues also influence immune functioning, the immune system may often be primed to respond aggressively to infection even under conditions in which there is no imminent threat of infection. Persistent priming of immune responses can have detrimental effects on individuals' immune functioning (Segerstrom & Miller, 2004). The overall implication is that the link between perceived disease cues and immune responsiveness may have important consequences for human health and welfare.

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Declaration of Conflicting Interests

The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

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References

- Cohen, S., Doyle, W.J., & Skoner, D.P. (1999). Psychological stress, cytokine production, and severity of upper respiratory illness. *Psychosomatic Medicine, 61*, 175–180.
- Curtis, V., Aunger, R., & Rabie, T. (2004). Evidence that disgust evolved to protect from risk of disease. *Proceedings of the Royal Society B: Biological Sciences, 271*, S131–S133.
- Deering, R.P., & Orange, J.S. (2006). Development of a clinical assay to evaluate toll-like receptor function. *Clinical Vaccine Immunology, 13*, 68–76.
- Kiecolt-Glaser, J.K., Preacher, K.J., MacCallum, R.C., Atkinson, C., Malarkey, W.B., & Glaser, R. (2003). Chronic stress and age-related increases in the proinflammatory cytokine IL-6. *Proceedings of the National Academy of Sciences, USA, 100*, 9090–9095.
- Miller, G.E., Rohleder, N., Stetler, C.A., & Kirschbaum, C. (2005). Clinical depression and regulation of the inflammatory response during acute stress. *Psychosomatic Medicine, 67*, 679–687.
- Oaten, M., Stevenson, R.J., & Case, T.I. (2009). Disgust as a disease-avoidance mechanism. *Psychological Bulletin, 135*, 303–321.
- Park, J.H., Faulkner, J., & Schaller, M. (2003). Evolved disease-avoidance processes and contemporary anti-social behavior: Prejudicial attitudes and avoidance of people with disabilities. *Journal of Nonverbal Behavior, 27*, 67–87.
- Park, J.H., Schaller, M., & Crandall, C.S. (2007). Pathogen-avoidance mechanisms and the stigmatization of obese people. *Evolution and Human Behavior, 28*, 410–411.
- Rose, N.R., Hamilton, R.G., & Detrick, B. (Eds.). (2002). *Manual of clinical laboratory immunology* (6th ed.). Washington, DC: American Society for Microbiology Press.
- Schaller, M., & Duncan, L.A. (2007). The behavioral immune system: Its evolution and social psychological implications. In J.P. Forgas, M. Haselton, & W. von Hippel (Eds.), *Evolution and the social mind* (pp. 293–307). New York: Psychology Press.
- Segerstrom, S.C., & Miller, G.E. (2004). Psychological stress and the human immune system: A meta-analytic study of 30 years of inquiry. *Psychological Bulletin, 130*, 601–630.
- Sternberg, E.M. (2006). Neural regulation of innate immunity: A coordinated nonspecific host response to pathogens. *Nature Reviews Immunology, 6*, 318–328.
- Webster, J.I., Tonelli, L., & Sternberg, E.M. (2002). Neuroendocrine regulation of immunity. *Annual Review of Immunology, 20*, 125–163.