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The Behavioral Immune System: Homelessness as a Disease Marker

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Abstract

Alongside the biological immune system is the behavioral immune system that works to avoid infection by avoiding contact with potential disease. The behavioral immune system reacts to visual cues of potential disease, such as coughing or skin lesions, and promotes avoidance behavior via disgust reactions. It interacts with biological immune responses by influencing hormones such as Interleukin-6 and cortisol. The behavioral immune system has also been found to overgeneralize and react to cues, such as obesity, disability, and foreignness, that do not signal actual disease threat. The purpose of this study is to test if the behavioral immune system overgeneralizes to include homelessness as a marker for disease. Analysis of the difference in salivary cortisol levels between the pre and post saliva samples showed no significant difference in cortisol levels between homeless and non-homeless individuals. The comparison supported the conclusion that cortisol drops due to disgust reactions from disease stimuli and revealed that we do not react to homeless people with disgust.

Keywords: behavioral immune system, cortisol, disease

The Behavioral Immune System: Homelessness as a Disease Marker

The Behavioral Immune System (BIS) is the psychological aspect of the immune system, developed as an evolutionary adaptation (Schaller & Duncan, 2007; Schaller & Park, 2011). The BIS is designed to prevent you from coming into contact with potential sources of infection by reacting to environmental cues and prompting avoidance behaviors (Miller & Maner, 2011; Schaller & Duncan, 2007; Schaller & Park, 2011).

The BIS attempts to keep you from getting infected to begin with by inciting disgust reactions and conscious cognitive strategies to avoid the potential threat (Schaller & Park, 2011). The disgust reaction is what is pertinent to this research. The disgust reaction is the mechanism through which the BIS prompts reactive avoidance. Disgust prompts you to remove yourself from the offending subject or even to gag and remove something dangerous from your body. The BIS is based around being able to identify disease markers, possible environmental cues that could indicate a disease threat, and then causing a disgust reaction so that the threat is avoided. Coughing and skin lesions are examples of disease markers.

The reaction of the BIS to traditional disease cues was tested in a 2010 study by Schaller, Miller, Gervais, Yager, and Chen at the University of British Columbia. This study presented participants with ten minute slideshows depicting people displaying typical symptoms of disease, such as coughing or rashes. They were tested for the presence of interleukin-6 (IL-6), a proinflammatory cytokine associated with an immune system response. This study provided evidence that the mere visual perception of disease is enough to result in a heightened immune system response.

A study done at Hamline University by J. Forrest Olsen in 2016 expanded on Schaller et al.'s experiment. Olsen replicated the slideshows depicting diseased individuals but this time

tested for cortisol levels. He found that cortisol levels decrease after viewing disease markers. The interaction between cortisol and IL-6 level changes after viewing the disease stimuli is explored in the next section. The decrease in cortisol is what this research uses as an indication that an immune response has taken place.

IL-6 and Cortisol

IL-6 is released by macrophages during an immune response (Brydon et al., 2009). IL-6 has been found to activate not only under the presence of infection but can also be activated by psychological stressors (Brydon et al., 2009) (Simmons & Broderick, 2005). Cytokines also help regulate the immune system, making them an integral part to an immune response (Simmons & Broderick, 2005).

Cortisol, on the other hand, is an anti-inflammatory glucocorticoid. Cortisol is produced in the adrenal gland, which is part of the hypothalamic-pituitary-adrenal axis (HPA axis). The HPA axis is an important part of the body's hormonal reaction to stress (Stephens & Wand, 2012). Cortisol can also respond to psychological stressors or in anticipation of future stressors (Ulrich-Lai & Herman, 2009). However, glucocorticoids are also immunosuppressive (Coutinho & Chapman, 2011).

The anti-inflammatory and immunosuppressive properties of cortisol could hinder the ability of IL-6 to regulate an immune response. This points to an interaction between IL-6 and cortisol that explains why concentrations of IL-6 were found to increase in the Schaller et al study and cortisol levels were found to drop in the Olsen study. Cortisol levels may decrease to allow for IL-6 to properly function as a regulator of an immune response.

This relationship is important for this study because cortisol levels are being used as an indicator that an immune response is taking place. If cortisol levels drop the same way as the Olsen study, that indicates that IL-6 is rising, which indicates a BIS response.

BIS Overgeneralization

The BIS's tendency to overgeneralize is in part due to a signal-detection problem, as described in Schaller and Duncan's 2007 chapter in *Evolution and the Social Mind: Evolutionary psychology and social cognition*. The BIS is designed to respond to the perceived presence of disease, which is why it reacts to things such as coughing and skin rashes. However symptoms such as coughing do not always indicate disease, it could just be someone clearing their throat. It is costly to the body if it fails to react to an actual disease but it costs much less to treat all possible symptoms as actual symptoms. The body will err on the side of false positives, rather than risk the threat of false negatives (Schaller & Duncan, 2007). Additionally, the BIS would be unable to avoid false-negatives if it was reacting only to a specific set of criteria. Infections can create a multitude of various symptoms, so the BIS is better served if it is not only over-sensitive but can also overgeneralize (Schaller & Duncan, 2007). However, this does not mean that the over-sensitivity of the BIS is always a good thing. Though you will avoid more false negatives, the multitude of false positives can contribute to prejudice.

Large subjective physical differences between people is one such category that the BIS may overgeneralize as possible disease cues, such as obesity (Park, Schaller, & Crandall, 2007) or foreignness (Schaller & Duncan, 2007). Foreignness is a well-studied subjective difference that has been found to elicit disgust and avoidance reactions that are indicative of BIS activation. Ethnocentric attitudes and preference were found to increase as perceived vulnerability to disease

increased (Navarrete & Fessler, 2005). Additionally, pregnant women in their first trimester were also found to be increasingly ethnocentric (Navarrete, Fessler, & Eng, 2006). The first trimester of pregnancy is a particularly dangerous time for both the mother and the fetus as they are more susceptible to infection during this time than any other time during the pregnancy, so it follows that the mother would show increased disease avoidance behavior.

Perceived Vulnerability to Disease Scale

The Perceived Vulnerability to Disease Scale (PVD scale) is a 15-item self report questionnaire to gauge how vulnerable a participant believes themselves to be to contracting disease. It measures two subscales: the participant's beliefs on their susceptibility to disease (Perceived Infectability) and their emotional discomfort in situations that denote a particularly high risk for disease transmission (Germ Aversion). The questionnaire was tested for internal consistency (Cronbach's $\alpha = .82$) and construct validity (Duncan, Schaller, & Park, 2009). The items were found to have acceptable validity and the complete list of correlations can be found in the aforementioned study.

The PVD scale has been used in multiple studies to gauge the relationship between perceived disease vulnerability and certain prejudicial attitudes. Positive correlations have been found between PVD scores and anti-fat attitudes (Park et al., 2007), xenophobia (Faulkner et al., 2004), negative attitudes towards physical disability (Park et al., 2003), and ageism (Duncan & Schaller, 2009). Our use of the PVD scale is to continue this type of investigation and see if high PVD scores correlate with cortisol responses or negative attitudes to homeless people, another marginalized group, as determined by responses to the post-experiment questionnaire.

Hypotheses

Cortisol levels will have a significant drop between pre and post saliva samples for the homeless stimuli and no drop between pre and post saliva samples for the non-homeless stimuli.

High PVD Scale scores will correspond to the largest drops in cortisol.

Methods

Participants

Participants ($N = 30$) were recruited from Hamline University undergraduate students in exchange for extra credit for the experiment. The experiment was presented as a study on hormone changes due to visual stimuli. Participants' responses to the questionnaire were kept confidential to encourage honesty and accuracy in the responses. Participants were treated in accordance with APA ethical guidelines and completed an informed consent form before both the questionnaire and the experiment.

Procedure

Participants ($N = 119$) completed a questionnaire three weeks prior to the start of the experiment (Appendix A). The questionnaire consisted of the Perceived Vulnerability to Disease scale and four questions regarding prior involvement with groups often overgeneralized by the BIS. The questionnaire was distributed to participating professors to administer to their students. This was done to avoid participants linking the questionnaire to the experiment via recognition of the experimenter. 30 experiment participants were found within these 119 questionnaire participants.

Before participants arrived, the experimenter set up the proper slideshow and the timer and relaxation video on the computer. The storage tubes and wrapped oral swabs were also set

out to the side of the desk, the tubes labelled with the slideshow label and pre- or post- slideshow designation.

The participant was met in the common area of the psychology labs. They were reminded to get a drink of water and rinse their mouth at a nearby water fountain if they had not already. Afterwards, they were escorted into the lab. Before proceeding, participants were instructed to carefully read the consent form and sign if they wished to continue with the experiment. They were also reminded that their participation was voluntary and they could withdraw at any time without penalty.

The participants were then asked exclusionary questions to determine if they were fit to continue. They were also asked to turn off all electronic devices and remove them from their person and place them out of sight. The experimenter requested they not discuss the experiment or its purpose with anyone until the results were posted.

Participants were instructed to wear sound-deadening headphones and watch a ten-minute slideshow. The experimenter stayed out of the participants' field of view for the duration of the slideshow. Afterwards, the participant donated a second saliva sample and was debriefed.

To participate in the experiment, several conditions were required to be met, of which the participants were informed prior to signing up. First, they had to be 18 years of age or older and could not have epilepsy. Second, they could not be sick or have been sick within two weeks prior to the experiment. Third, they could not consume alcohol within three days prior to the experiment. Fourth, they could not eat, use tobacco, or chew gum within two hours of the experiment. Fifth, they had to rinse their mouths with water prior to the experiment.

The experimenter carried a hard copy of the experimental protocol and read questions and directions directly from it. This was done to avoid possible confounds by deviation from the protocol.

Participants provided a saliva sample before and after viewing a ten minute slideshow depicting homeless or non-homeless individuals. The stimuli were cropped to 6" x 4". Random presentation order was assigned via Latin Square. All slideshows were made using Microsoft PowerPoint software. The slideshow design was adapted from the visual disease cues experiment done by Hamline University's J. Forrest Olsen in 2016. Slideshows averaged ten minutes long (\pm 3 seconds).

Participants were randomly assigned to one of 4 groups. A control group ($N = 4$) using non-homeless stimuli at 4 second exposure, or experimental groups using homeless stimuli consisting of 10 ms ($N = 8$), 400 ms ($N = 8$), and 4 second ($N = 8$) exposure groups.

All slideshows included 50 stimuli throughout the ten minute slideshow, regardless of exposure group. To meet the required ten minute length, slideshows also included neutral gray slides and blank transition slides. Blank slides were standardized at 4 seconds each. Gray slides were presented the same amount of time as the stimuli in each exposure group. Experimental condition exposure groups viewed images of homeless individuals (figure 1.1). Control condition exposure groups viewed images of non-homeless individuals (figure 1.2). Gray slide placement was calculated to appear in the most regular intervals possible that timing would allow.

Figure 1.1



Figure 1.2



Exclusion criteria. All participants were screened after the consent form was signed. If a participant was not eighteen years of age or older, had a visual impairment with no corrective lenses or procedure, or had epilepsy, the experiment ended there and they were excused. If a participant had not followed pre-experiment requirements, such as not eating for two hours prior, had been sick in the last two weeks, consumed alcohol in the prior three days, or had visual impairment but did not have their corrective lenses with them, they were rescheduled for a later date.

Salivary Cortisol Assays

Saliva samples were collected using Salimetrics LLC oral swabs and storage tubes. Samples were stored immediately after donation in a Criterion™ refrigerator-freezer unit kept in the corner behind the participant.

Saliva samples were sent to Salimetrics for salivary cortisol testing using their Salivary Cortisol ELISA Assays and tested in replicate.

Results

The questionnaire yielded no results.

Four participants were excluded from analysis due to lack of cortisol detection in the lab analysis or due to being outliers.

Cortisol levels decreased 4.91% (95% CI [3.1% , 6.7 %]) after viewing face stimuli ($F(1,22) = 31, p < .001, \eta_p^2 = .59$). Exposure condition and interaction were non-significant ($F(3,22) = .2$ and $F(3, 22) = 2.17$, respectively). There were no differences between homeless and happy (non-homeless) faces ($t(24) = .06$). Figure 2 shows the variation in cortisol levels per exposure as well as the control group. Perceived Vulnerability to Disease Scale scores were also found to be non-significant when compared to the saliva samples.

An independent samples t-test between healthy faces (the data for the homeless and non-homeless conditions) and sick faces (Olsen, 2016) revealed a significant difference in changes of cortisol levels ($p = .027, t = -2.282, n = 54$). Figure 3 displays the average percent decrease in cortisol between happy faces, homeless faces, and sick faces. Figure 4 combines happy and homeless faces into healthy faces and compares that with sick faces.

Conclusion and Discussion

The 4.91% drop in cortisol across both homeless and happy conditions demonstrates that no difference was seen between the homeless and happy faces, as far as the BIS is concerned. These results compared with the sick faces results from the 2016 Olsen study display a striking difference in cortisol levels. Where the healthy faces (happy and homeless) garnered about a 5% drop in cortisol, the sick faces garnered a 15% drop. This indicates that the homeless faces were not treated as disease cues, as the sick faces were, but were treated the same as the happy faces which lacked any disease markers. Since the healthy faces did not meet the level of cortisol drop found for sick faces, levels of IL-6 likely did not increase, so a BIS reaction likely did not take place.

The 5% drop in cortisol for healthy faces may be due to the soothing effect seeing other people can have. Humans are social by nature, so it is possible that this very small drop in cortisol is due to a social effect.

The lack of high enough cortisol drops in the healthy faces, combined with the lack of significant results from the questionnaire, indicates that even people who are especially concerned about illness fail to experience a BIS response to homelessness. This also indicates that the BIS has some other criteria for what is generalized to, which will need to be expanded in further research.

It is important to note that the overgeneralization of the BIS is not reacting to potential disease cues as if they are threats that should involve fear. The BIS is reacting with disgust for the sake of avoidance. Additionally, it is suggested that disgust utilizes the parasympathetic nervous system, which is supported by our data. If the BIS used the sympathetic nervous system as part of the disgust mechanism then the data would likely have shown increases in cortisol, not decreases. Also, the BIS activation does not preclude the possibility of other more complex

emotional responses to various stimuli, such as the homeless. Complex emotional reactions surrounding the homeless are likely typical, but it is the potential disgust reaction that is key to this study.

The behavioral aspect of disgust is also the only aspect of a complex basic emotion that is being considered here. The behavioral component of avoidance in disgust and links to hormone changes is what is supported by the previously discussed research and could be utilized in this study. Disgust still involves an array of cognitions and also simply the experience of the emotion itself, none of which is explored here.

Though the order of inferences in this study goes from cortisol drops to IL-6 increases to BIS activation and disgust, the actual order of events may be different. This study had to follow this order to draw the conclusion, but this order does not need to be followed in real life to remain true. The order could start with a disgust reaction, which then leads to IL-6 and cortisol changes, all of which taken together is the BIS activation. This study is simply not equipped to measure when disgust and hormone changes happen in what order. The inferences are still valid, regardless of the true overlap or order of events.

Since this study indicates that no BIS activation occurred from the homeless, it points to a more social explanation of why the homeless are treated with prejudice. The complex emotions surrounding the homeless are also an important component to why we avoid the homeless so regularly. Perhaps we avoid them because we feel ashamed or are uncomfortable. Perhaps we avoid them because of preconceived notions about what kind of people the homeless are. This is an area in which further study would be useful, to identify exactly what it is about our reactions to the homeless that make us treat them the way we do. This study simply shows that it is not due to an overgeneralized disease response.

Figure 2.

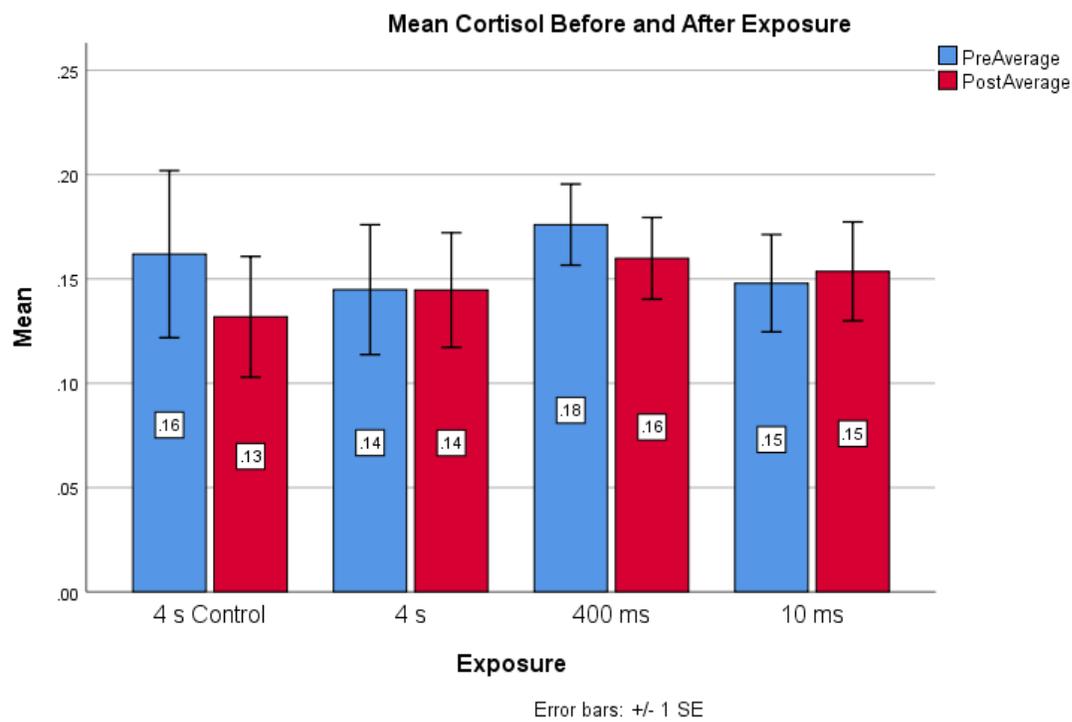


Figure 3.

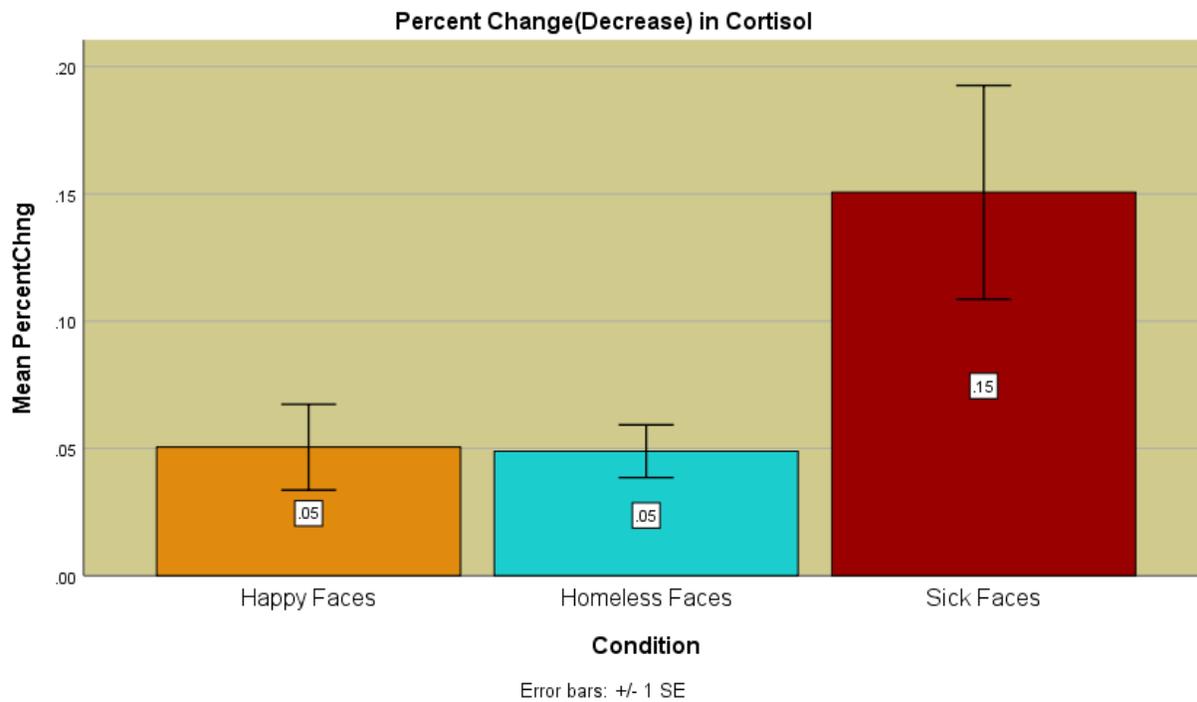
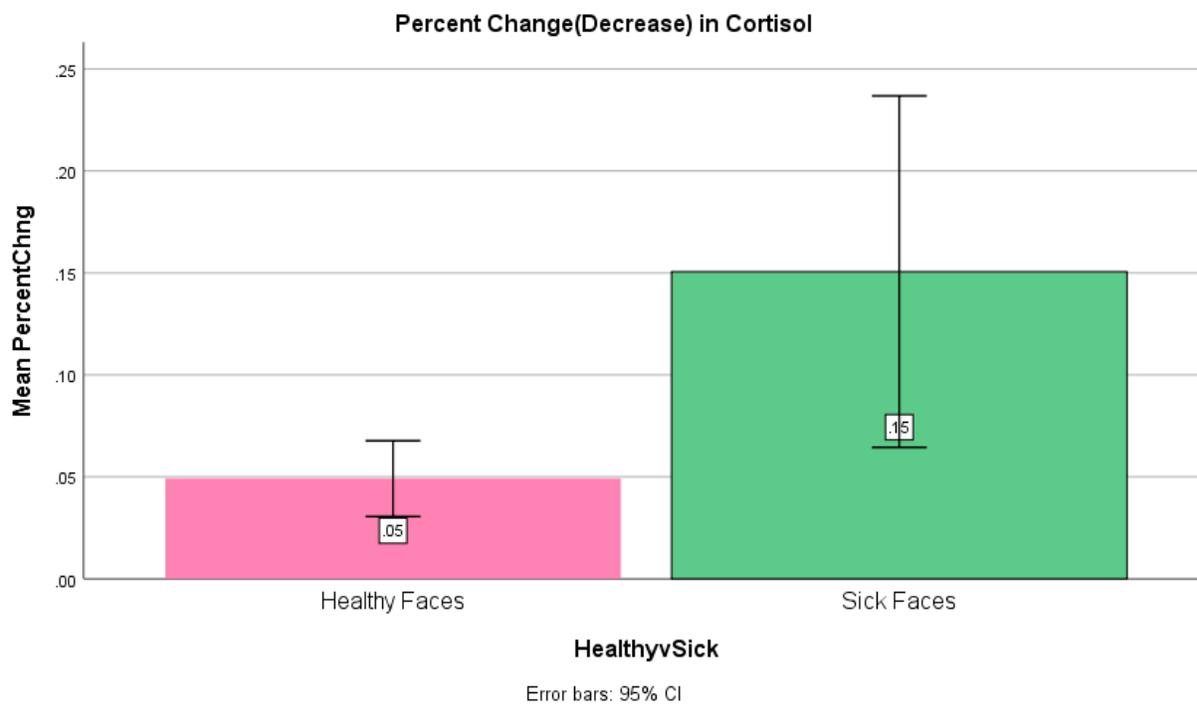


Figure 4.



Appendix A

Please **circle the number** indicating the degree to which you agree or disagree with the following statements, “1” corresponding to strongly disagree and “7” corresponding to strongly agree.

1. *It really bothers me when people sneeze without covering their mouths.*

Strongly disagree	Disagree	Somewhat disagree	Neutral	Somewhat agree	Agree	Strongly agree
1	2	3	4	5	6	7

2. *If an illness is 'going around', I will get it.*

Strongly disagree	Disagree	Somewhat disagree	Neutral	Somewhat agree	Agree	Strongly agree
1	2	3	4	5	6	7

3. *I am comfortable sharing a water bottle with a friend.*

Strongly disagree	Disagree	Somewhat disagree	Neutral	Somewhat agree	Agree	Strongly agree
1	2	3	4	5	6	7

4. *My past experiences make me believe I am not likely to get sick even when my friends are sick.*

Strongly disagree	Disagree	Somewhat disagree	Neutral	Somewhat agree	Agree	Strongly agree
1	2	3	4	5	6	7

5. *I have a history of susceptibility to infectious diseases.*

Strongly disagree	Disagree	Somewhat disagree	Neutral	Somewhat agree	Agree	Strongly agree
1	2	3	4	5	6	7

6. *I prefer to wash my hands pretty soon after shaking someone's hand.*

Strongly disagree	Disagree	Somewhat disagree	Neutral	Somewhat agree	Agree	Strongly agree
1	2	3	4	5	6	7

7. *In general, I am very susceptible to colds, flu, and other infectious diseases.*

Strongly disagree	Disagree	Somewhat disagree	Neutral	Somewhat agree	Agree	Strongly agree
1	2	3	4	5	6	7

8. *I dislike wearing used clothes because you don't know what the past person who wore it was like.*

Strongly disagree	Disagree	Somewhat disagree	Neutral	Somewhat agree	Agree	Strongly agree
1	2	3	4	5	6	7

9. *I am more likely than the people around me to catch an infectious disease.*

Strongly disagree	Disagree	Somewhat disagree	Neutral	Somewhat agree	Agree	Strongly agree
1	2	3	4	5	6	7

10. *My hands do not feel dirty after touching money.*

Strongly disagree	Disagree	Somewhat disagree	Neutral	Somewhat agree	Agree	Strongly agree
1	2	3	4	5	6	7

11. *I am unlikely to catch a cold, flu, or other illness, even if it is going around.*

Strongly disagree	Disagree	Somewhat disagree	Neutral	Somewhat agree	Agree	Strongly agree
1	2	3	4	5	6	7

12. *It does not make me anxious to be around sick people.*

Strongly disagree	Disagree	Somewhat disagree	Neutral	Somewhat agree	Agree	Strongly agree
1	2	3	4	5	6	7

13. *My immune system protects me from most illnesses that other people get.*

Strongly disagree	Disagree	Somewhat disagree	Neutral	Somewhat agree	Agree	Strongly agree
1	2	3	4	5	6	7

Please respond to the following questions by **circling Yes or No**:

	Circle Yes or No	
Have you ever worked with or affiliated with someone who is elderly?	Yes	No
Have you ever worked with or affiliated with someone who is homeless?	Yes	No
Have you ever worked with or affiliated with someone who is chronically ill?	Yes	No
Have you ever worked with or affiliated with someone who is a foreign immigrant?	Yes	No

References

Brydon, L., Walker, C., Wawrzyniak, A., Whitehead, D., Okamura, H., Yajima, J., ... & Steptoe, A. (2009). Synergistic effects of psychological and immune stressors on inflammatory cytokine and sickness responses in humans. *Brain, behavior, and immunity*, 23(2), 217-224.

Coutinho, A. E., & Chapman, K. E. (2011). The anti-inflammatory and immunosuppressive effects of glucocorticoids, recent developments and mechanistic insights. *Molecular and Cellular Endocrinology*, 335(1), 2-13. doi:10.1016/j.mce.2010.04.005

Duncan, L. A., Schaller, M., & Park, J. H. (2009). Perceived vulnerability to disease: Development and validation of a 15-item self-report instrument. *Personality and Individual Differences*, 47(6), 541-546. doi:10.1016/j.paid.2009.05.001

<https://digitalcommons.hamline.edu/dhp/47>

Miller, S. L., & Maner, J. K. (2011). Sick Body, Vigilant Mind. *Psychological Science*, 22(12), 1467-1471. doi:10.1177/0956797611420166

Navarrete, C. D., & Fessler, D. M. (2006). Disease avoidance and ethnocentrism: The effects of disease vulnerability and disgust sensitivity on intergroup attitudes. *Evolution and Human Behavior*, 27(4), 270-282.

Navarrete, C. D., Fessler, D. M., & Eng, S. J. (2007). Elevated ethnocentrism in the first trimester of pregnancy. *Evolution and Human Behavior*, 28(1), 60-65.
doi:10.1016/j.evolhumbehav.2006.06.002

Olsen, J. Forrest, "Human Vision Inspires Cortisol and Immune Behaviors" (2016). Departmental Honors Projects. 47.

Park, J. H., Schaller, M., & Crandall, C. S. (2006). Psychological disease-avoidance mechanisms and stigmatization of fat people. Unpublished manuscript, University of Groningen, The Netherlands.

Schaller, M., & Duncan, L. A. (2007). The behavioral immune system: Its evolution and social psychological implications. In J. P. Forgas, M. G. Haselton, & W. von Hippel (Eds.), *Sydney symposium of social psychology. Evolution and the social mind: Evolutionary psychology and social cognition* (pp. 293-307). New York, NY, US: Routledge/Taylor & Francis Group.

Schaller, M., & Park, J. H. (2011). The Behavioral Immune System (and Why It Matters). *Current Directions in Psychological Science*, 20(2), 99-103. doi:10.1177/0963721411402596

Schaller, M., Miller, G. E., Gervais, W. M., Yager, S., & Chen, E. (2010). Mere Visual Perception of Other People's Disease Symptoms Facilitates a More Aggressive Immune Response. *Psychological Science*, 21(5), 649-652. doi:10.1177/0956797610368064

Simmons, D. A., & Broderick, P. A. (2005). Cytokines, stressors, and clinical depression: Augmented adaptation responses underlie depression pathogenesis. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, 29(5), 793-807.

doi:10.1016/j.pnpbp.2005.03.009

Stephens, M. A., Mccaul, M. E., & Wand, G. S. (2014). The Potential Role of Glucocorticoids and the HPA Axis in Alcohol Dependence. *Neurobiology of Alcohol Dependence*, 429-450. doi:10.1016/b978-0-12-405941-2.00021-3

Ulrich-Lai, Y. M., & Herman, J. P. (2009). Neural regulation of endocrine and autonomic stress responses. *Nature Reviews Neuroscience*, 10(6), 397-409.