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Frequency and recency of infection and their relationship with disgust and contamination sensitivity

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Abstract

Both disgust and contamination sensitivity likely evolved to protect us from infectious disease. Paradoxically, disgust may be reduced by frequent exposure to disgust-inducing cues — cues most likely to occur in disease-rich environments. In this study, we examined whether more frequent or recent illness might act to reverse this process. To test this, we surveyed 616 adults, obtaining illness frequency and recency data, disgust and contamination sensitivity, and a variety of control measures. Heightened contamination sensitivity was associated with more frequent infectious illness, but not with recency of infection. We also found that participants who had heightened contamination sensitivity and who were also more disgust sensitive had significantly fewer recent infections. These findings suggest that frequent illness may upregulate contamination sensitivity potentially counteracting the effects of exposure on disgust. More importantly, these data provide the first direct evidence of a protective effect of contamination and disgust, against infectious disease.

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1. Introduction

It has been suggested that the emotion of disgust evolved to protect us from contracting infectious disease (e.g., Curtis & Biran, 2001; Davey, 1994; Fessler & Navarrete, 2003). This perspective on disgust as a disease avoidance mechanism is based upon several observations, most notably the concordance between stimuli that evoke disgust and those that connote the presence of pathogens, and by the apparent cross-cultural universality of this concordance (e.g., Curtis & Biran, 2001). Disgust may function in this role by generating intense negative affect towards potentially disease-bearing sources, thereby reducing the risk of contact and infection. A related but distinct psychological entity, which is also involved in disease avoidance, is contamination. A sense of being contaminated may be accompanied by emotions other than disgust, notably fear (Rachman, 2004). Feeling contaminated can invoke particular types of behavior such as avoidance and hand washing (Rachman, 2004). These two

constructs, disgust, with its intense negative affect, and contamination, with its associated action tendencies, both contribute to disease avoidance in humans.

Whilst there may be good grounds for a functional connection between disease avoidance and disgust, there is a significant and hitherto unacknowledged problem. Exposure to disgust elicitors results in a reduction of self-reported disgust, and this effect has been observed in several studies. In an exploratory survey participants who had cared for a sick person and who changed soiled bedding, wounds, etc., reported reduced disgust sensitivity for these and similar cues (Stevenson, unpublished data). Relatedly, we have observed a strong correlation between scores on a questionnaire designed to assess participants' exposure history to disgust cues and their responses on the Disgust Sensitivity Scale (Case, Stevenson, & Oaten, In preparation). Greater reported exposure equated to less reported disgust.

Behavioral data also suggests the same conclusion. Case, Repacholi, and Stevenson (2006) found that mothers' were significantly less disgusted by the smell of their own babies' soiled (feces) nappies, than by a stranger, an effect that appeared to result from differential exposure history. Rozin (2008) has recently identified a similar phenomenon in

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medical students, by examining their self-reported disgust sensitivity before and after they had spent several months dissecting a cadaver. He found a significant reduction in disgust responses to death and body envelope violations, but no significant change in any other type of disgust. That exposure leads to diminution of disgust for similar cues is problematic for a disease avoidance account. This is because environments that are rich in pathogens are likely to be those that contain many disgust-evoking cues, both from exposure to the symptoms of illness and from the environmental conditions that foster ill-health (e.g., poor sanitation). If frequent exposure to such cues results in reduced disgust, and thus less avoidance, the functional value of disgust as a disease-avoidance mechanism is weakened under the very conditions where it is needed most.

In an environment where disease, and thus disgust cues, is most prevalent, contracting infection will be common. Being ill could act to up-regulate a person's propensity to feel contaminated as well as the behaviors that this state results in. Contamination is defined here as "an intense and persisting feeling of having been polluted or infected or endangered as a result of contact, direct or indirect, with a person/place/object that is perceived to be soiled, impure, infectious or harmful" (Rachman, 2004; p. 1229). Although disgust may provide an important signal in the definition above as to what is "perceived to be soiled, impure, infectious or harmful," Rachman (2004) has argued that it is a person's contamination sensitivity that dictates both avoidance behavior and attempts to remove the contaminant.

A further issue here concerns the nature of the infection. At a basic level, contamination sensitivity could be upregulated in response to infection recency (i.e., more recent infections over more distant ones) or by infection frequency (i.e., the infection burden on an individual in the past). Both frequency and recency are likely to be related and, moreover, other infection-related variables might also be important (e.g., severity, presence or absence of particular symptoms, etc.). Whatever the details, and these issues have not been explored before, our first aim was to establish whether more recent or more frequent infections are positively associated with contamination sensitivity. Put bluntly, is getting sick with an infectious disease associated with greater contamination sensitivity, after controlling for other potentially confounding variables (e.g., age, gender, etc. — see Method for details)? Such a relationship would provide one possible solution to the disgust-exposure paradox outlined above, as well as illustrate for the first time a link between psychological variables involved in disease avoidance and reported ill-health.

The arguments presented above naturally lead to a further prediction. If a capacity for disgust and contamination arose to protect us from infectious disease, can evidence be obtained to demonstrate this relationship now? In the context of this study, would being more disgust sensitive, and/or more contamination sensitive, be associated with lower rates of infectious illness? Two points bear upon this hypothesis.

First, any such relationship might be obscured by the predicted enhancing effect of infectious illness on contamination sensitivity. Thus detecting any protective effect would have to take into account any illness-related factor that was positively associated with contamination sensitivity. Second, and as described in the first paragraph, disgust and contamination sensitivity probably act in concert. As a result, it may be that the interaction (or moderating effect) between them is more predictive of reduced rates of infectious disease than either variable alone.

2. Method

2.1. Participants

Six hundred and sixteen participants were recruited from the Macquarie University community. Of these 616 (some with missing values for various variables), 214 were male and 394 female. Eight participants did not report their gender. Age ranged from 16 to 64 years, with a median of 20 years.

2.2. Procedure

Participants completed biographical data and several questionnaires in a single sitting. These included the Disgust Sensitivity Scale (Haidt, McCauley, & Rozin, 1994), with a higher score indicating greater disgust sensitivity. This scale is unique in being the only one validated against actual behavioral reactions to disgust elicitors (Rozin, Haidt, McCauley, Dunlop, & Ashmore, 1999). In calculating the overall score, we utilised only the subset of items suggested by Olatunji et al. (2007) in their recent refinement of this scale (alpha=.84). Participants also completed the contamination sensitivity scale of the Padua Inventory (Burns, Keortge, Formea, & Sternberger, 1996; subscale alpha=.85). This is a subset of items that form part of a larger questionnaire used to assess individual differences in obsessive-compulsive disorder symptoms. Higher scores reflect greater contamination sensitivity.

In addition to these questionnaires, participants also completed a number of control measures. First, the Whiteley Index, a validated hypochondria scale (Hiller, Rief, & Fichter, 2002; Speckens, Spinhoven, Sloekers, Bolk, & van Hemert, 1996; higher scores=greater hypochondria; alpha=.76), was included as high scorers may report more illnesses as a consequence of a more liberal criterion for what constitutes 'illness'. Second, the short form of the Crowne-Marlowe Social Desirability Scale (Reynolds, 1982; higher scores=greater social desirability; alpha=.76) was included to control for any effects of social desirability on reporting of disgust and health-related variables. Third, the DASS-21 (Depression, Anxiety and Stress Scale; Lovibond & Lovibond, 1995; higher scores equate to greater depression, stress and anxiety; alpha=.88) was included to assess general mental health, as greater levels of reported stress, anxiety and

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