Evolution and Human Behavior 39 (2018) 468–469

Contents lists available at ScienceDirect

Evolution and Human Behavior

journal homepage: www.ehbonline.org

Response to "Hormonal Correlates of Pathogen Disgust: Testing the Compensatory Prophylaxis Hypothesis"

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1. Introduction

We commend Jones et al. (2018) for an important, rigorous, and well-powered contribution to investigating compensatory prophylaxis. One of the core predictions in Fleischman and Fessler's (2011) formulation of the compensatory prophylaxis hypothesis (CPH) is that disgust, an emotion associated with avoiding cues of disease, will be amplified when, due to increases in progesterone levels, immunity is reduced. Contrary to this prediction, Jones et al. (2018) find that, both in analyses within subjects (correlating disgust with hormone levels) and between subjects (correlating disgust with hormone levels between women), the pathogen domain of disgust in the Three Domain Disgust Scale (TDDS; Tybur, Lieberman, & Griskevicius, 2009) is not positively associated either with progesterone, or with the ratio of estrogen to progesterone. Below we present three possible explanations for the differences between Jones et al.'s findings and prior results.

2. Possibility 1: the compensatory prophylaxis hypothesis is entirely wrong

"The compensatory behavioral prophylaxis hypothesis holds that evolved psychological mechanisms enhance avoidance of potential contaminants during periods of reproductive immunomodulation so as to decrease the likelihood of infection" (Fleischman & Fessler, 2011, pg 271). The CPH is one of several adaptationist hypotheses that predict motivations, emotions, and behaviors that reduce contact with cues of disease will be upregulated during periods of increased vulnerability to disease (see Ackerman, Hill, & Murray, 2018 for review).

Mediated by progesterone, women show increased susceptibility to infection and a shift in immune response during the luteal phase (Tan, Peeva, & Zandman-Goddard, 2015). Although otherwise puzzling features of physiology are understandable if such patterned changes in immune functioning have indeed selected for second-order adaptations (Amir & Fessler, 2013), nevertheless, it is possible that these changes in immune functioning are too small or not consistent enough to exert selective pressure on mechanisms governing behavior. Potentially compounding a situation of weak selective pressure, due to lactational amenorrhea and pregnancy, ancestral women may not have had enough menstrual cycles over the course of their reproductive lifespans to shape luteal-phase adjustments in behavior.

Although disgust is considered the central emotion of disease avoidance, there is not much evidence that disgust tracks immune vulnerability, or that temporarily elevated infection susceptibility increases disgust (De Barra, Islam, & Curtis, 2014). Although disgust is clearly associated with stimuli that pose a heightened disease risk (Curtis, Aunger, & Rabie, 2004), disgust does not seem to track the prevalence of infectious disease in the environment or the infection susceptibility of the individual. Parasite stress is not associated with disgust sensitivity (Tybur et al., 2016) and, in a Bangladeshi sample, disgust sensitivity does not correlate with self-reported adult health or mother's reports of childhood illness (De Barra et al., 2014). With the caveat that studies of immune vulnerability and activation all employ relatively small sample sizes, there is fairly consistent evidence that disgust stimuli increase immune activation (Schaller, Miller, Gervais, Yager, & Chen, 2010; Stevenson et al., 2012), but scant evidence that immune vulnerability increases disgust sensitivity. For example, patients with increased infection risk because of rheumatoid arthritis showed similar disgust sensitivity to age-matched controls, and immunosuppressive drug use was actually associated with reduced disgust sensitivity (Oaten, Stevenson, & Case, 2017). Childhood infection is not associated with increased disgust sensitivity (De Barra et al., 2014). Ersche et al. (2014) found that cocaine-dependent men showing greater inflammatory markers indicative of infection and who reported more antibiotic use and greater difficulty fighting infection than age-matched controls were more distracted by disgusting images but nonetheless had similar self-reported disgust using Van Overveld, De Jong, and Peters's (2010) disgust propensity scale. Existing evidence is inadequate to determine whether disease vulnerability or recent infection increase disgust sensitivity, although these features appear to influence other measures, such as attention to disgust stimuli (e.g., Miller & Maner, 2011).

3. Possibility 2: measurement issues

A second possibility is that compensatory increases in disease avoidance do occur as a function of progesterone, but are sufficiently subtle as

DOI of original article: https://doi.org/10.1016/j.evolhumbehav.2017.12.004.
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https://doi.org/10.1016/j.evolhumbehav.2018.03.006
1090-5138/

to vary in detectability by method. As Jones et al. (2018) note, Fleischman and Fessler (2011) did not use the TDDS; instead, their positive results derived from self-reported disgust responses to photos, taken from Curtis et al. (2004), depicting disease cues, with similar patterns obtained for self-reported actual disease-avoidance bathroom and grooming behaviors, and contamination-relevant features of subclinical obsessive-compulsive disorder. In contrast, like Jones et al. (2018), Żelaźniewicz, Borkowska, Nowak, and Pawłowski (2016) used a repeated measures design and did not find that women in the luteal phase showed increased pathogen disgust on the TDDS, although they did find elevated responses from women in the luteal phase on the "animal reminder" domain from the older Disgust Scale (Olatunji et al., 2007), which includes body envelope violations. It is thus possible that self-reported disgust for text-only questionnaire items is too blunt an instrument to be deployed during periods of immune vulnerability. It is possible that only certain more sensitive measures of disease avoidance, such as self-reported disgust to graphic visual images containing disease cues, or the latency of response to such images, demonstrate an effect of immune vulnerability. Of relevance in this regard, although results are not conclusive, it appears that image-based measures and response latency show effects of immunocompetence where self-reported disgust sensitivity to text items do not (Ersche et al., 2014; Miller & Maner, 2011). Lastly, there are some indications that sexually active women experience greater immune changes across the cycle than those who are not sexually active (Fleischman & Fessler, 2007; Lorenz, Demas, & Heiman, 2015), suggesting that inconsistent findings across studies may owe not to the instruments used to measure disgust, but to unmeasured differences in sexual activity across samples. There is not published data investigating correlations between the TDDS and image-based ratings. However, a recent paper has shown that a text-based disgust scale and disgust image ratings correlate fairly well, $r = 0.6$ (Haberkamp, Glombiewski, Schmidt, & Barke, 2017). Thus, barring measurement issues, given the greatly elevated power of Jones et al.'s (2018) study relative to prior tests of the CPH, it is likely their null finding accurately reflects the underlying phenomenon.

4. Possibility 3: progesterone is not the driving factor

From the inception of the CPH, progesterone has been theorized to be the driving factor in a postulated underlying proximate mechanism, as it is progesterone that initiates the cascade of immune changes that both allow for maternal tolerance of the conceptus and enhance vulnerability to infection. However, such formulations overlook the fact that, although the risks of maternal infection are greatest during the first trimester of pregnancy – the period that prior work has shown corresponds with increased disgust sensitivity (Fessler, Eng, & Navarrete, 2005) – nonetheless, progesterone levels *increase* throughout pregnancy, and the effects of progesterone and related hormones on immune function is both highly complex and variable over the course of pregnancy (reviewed in Tan et al., 2015). Accordingly, it is possible that prophylactic behaviors in general, and disgust responses in particular, are indeed upregulated in a manner that partially compensates for reproductive immunomodulation, but that progesterone is either not the proximate driver of such changes, or else acts in a complex manner in interaction with other components of the physiological underpinnings of pregnancy.

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