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Original Article

Compensatory up-regulation of behavioral disease avoidance in immuno-compromised people with rheumatoid arthritis

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ABSTRACT

Disgust and disease-related cues can activate the immune system. Here, we test whether immuno-suppression is associated with an up-regulation of cognitions and behaviors that assist in disease avoidance. People with rheumatoid arthritis (RA), who have a heightened risk of infection-related morbidity and mortality, were compared to age, gender and demographically matched healthy controls on a range of disease avoidance tasks. People with RA scored higher on reports of behavior likely to control infection, were more accurate in spotting individuals who were sick, and showed disease-specific ethnocentrism, ascribing a greater risk of contracting disease to non-Caucasians, although having no overall propensity for greater racism on the Modern Racism Scale. Contrary to predictions, disgust sensitivity (DS) did not differ between groups, however among people with RA, DS was found to be *lower* in those taking drugs that can increase infection risk. While more explicit disease avoidance behaviors are clearly up-regulated in people with RA, changes in DS may have a different and perhaps more biological casual basis.

1. Introduction

Infectious disease is both a current threat to human health, accounting for 40% of deaths in low-income countries in 2010 (WHO, 2011), and also an ancient one, providing selective pressure for the evolution of complex immune systems (Hirano, Das, Guo, & Cooper, 2011). One way that humans and animals have sought to minimize the costs associated with infection, is to try and avoid getting sick in the first place (Curtis, Aunger, & Rabie, 2004; Curtis & Biran, 2001; Schaller, 2011). In humans, affective (e.g., disgust), cognitive (e.g., germ theory) and behavioral (e.g., hygiene) processes all contribute to what can be termed a disease avoidance system. There has been some interest in determining whether the disease avoidance system can affect the immune system, preparing it for the possibility of a pathogen attack. Exposure to disgusting images (Stevenson, Hodgson, Oaten, Barouei, & Case, 2011; Stevenson et al., 2012) or to disease related images (e.g., people sneezing; Schaller, Miller, Gervais, Yager, & Chen, 2010) results in innate immune activation. However, less is known about the reverse pathway, namely whether alterations in immune function are associated with changes to the disease avoidance system. This MS focuses on this latter question.

Evidence that alterations in immune function can impact the disease avoidance system comes from several different sources. One is from studying mothers during pregnancy, where various forms of immune suppression occur so as to aid tolerance towards the fetus (e.g., Luppi, 2003). Pregnant mothers show ingestive selectivity, elevated disgust, increased preference for healthy faces, and elevated ethnocentrism — all of which arguably compensate for immune suppression (Fessler, Eng, & Navarrete, 2005; Navarrete, Fessler, & Eng, 2007). Three more recent and complimentary studies have been reported using different populations. First, Stevenson, Case, and Oaten (2009) found that the more frequently a person experienced common infectious diseases, the higher their reported degree of contamination sensitivity. Second, Huang, Shidlovskaya, Ackerman, and Bargh (2011) found that *previous* flu immunization – weeks or months ago – reduced ethnocentrism, an effect they found to be driven by beliefs about disease vulnerability, rather than by immune activation. Third, Miller and Maner (2011) found that recent illness (within 1 week), which can produce mild-immune suppression, automatically increased attention to disease salient cues independent of beliefs about disease vulnerability, suggesting a possible biological cause.

These various findings suggest that alterations in immune function should lead to compensatory changes in one or more aspects of disease avoidant behavior. Before turning to our current test of this hypothesis it is important to note that not all examinations of this idea have been supportive (and see Currie & Mace, 2012, for broader concerns about

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the link between pathogens and behavior). de Barra, Islam, and Curtis (2014), found that greater childhood exposure to pathogens was not associated with increased disgust sensitivity as an adult, and also that disgust sensitivity as an adult was not associated with recent infection history. While de Barra et al. (2014) suggest several plausible reasons for their null result, the idea that alterations in immune function might lead to compensatory changes in behavior is not universally supported.

In the current study, we tested this idea in a novel way by examining a group of people who have rheumatoid arthritis (RA). RA is a label that very likely includes several different autoimmune diseases, all characterized by systemic and chronic inflammation, especially affecting the joints (Lee & Weinblatt, 2001; Picerno et al., 2015). It is one of the most common autoimmune diagnoses (0.25–0.5% prevalence) typically manifesting in the 4th or 5th decade, and occurring more frequently in women than in men (Gibofsky, 2014). RA is believed to result from a complex interplay between genetic susceptibility and environmental triggers, with prior infectious disease, smoking, poor diet and pollutants all possible factors (Toben, Youinou, & Saraux, 2010). One well-identified consequence of developing RA is that it significantly increases the likelihood of contracting both opportunistic and common infections (e.g., Doran, Crowson, Pond, O'Fallon, & Gabriel, 2002). Not only is the risk of infection increased, but so too is the risk of dying from one. Several studies have now determined that death from infection is more common among people with RA than among matched controls (e.g., Mutru, Laakso, Isomaki, & Koota, 1985; Reill, Cosh, Maddison, Rasker, & Silman, 1990). These excess deaths result most commonly from respiratory and urinary tract infections (Koivuniemi, Leirisalo-Repo, Suomalainen, Piirainen, & Paimela, 2006).

Not only does RA itself increase susceptibility to infection, a number of treatments used to ameliorate disease progression also result in immuno-suppression and possibly additional infection risk. Several different unrelated classes of medicine are used to suppress the joint-damaging effects of RA and these are known collectively as disease modifying anti-rheumatic drugs (DMARDs). It has been suspected for many years that several DMARDS may increase infection risk (McLean-Tooke, Aldridge, Waugh, Spickett, & Kay, 2009), but more recent treatments that specifically target the immune system directly (the so-called 'biologicals') seem to carry the clearest additional risk (Singh et al., 2015).

RA then is a disease that both in and of itself, and by virtue of its treatment, results in some degree of immune-impairment, with increased risk of infection and death. Consequently, we might expect that people with RA would experience up-regulation of the disease avoidance system, so as to compensate for the reduced effectiveness of their biological immune system. To test this hypothesis, we recruited two groups of participants, one with RA and another matched on age, gender and demographic characteristics, so we could determine whether disease avoidance was altered by the presence of RA.

We asked participants to report how disgust sensitive they were using a behaviorally validated measure of disgust sensitivity (Haidt, McCauley, & Rozin, 1994; Rozin, Haidt, McCauley, Dunlop, & Ashmore, 1999), employing the revised factor structure to avoid some of its more well known psychometric shortcomings (Olatunji et al., 2007). We also asked participants to reflect on their explicit disease-related cognitions and behaviors using the Perceived Vulnerability to Disease scale (Duncan, Schaller, & Park, 2009). Two behavioral tasks were also included. The first asked participants to view Caucasian, Chinese, South Asian and African faces, judging in each case the likelihood of contracting an infectious disease from that person. The second asked participants to view (this being covertly timed) a set of healthy, birth-marked and sick faces. Participants were later given a surprise memory test and asked to recall how many sick-looking people they had viewed earlier in the experiment. We expected that RA participants would: (1) score higher on disgust sensitivity and perceived vulnerability to disease; (2) would judge non-Caucasian participants to be a greater disease threat (participants here mainly being Caucasian) than Caucasians; and (3) would be more accurate in recalling how many sick people they saw, by presumably attending to these disease-relevant stimuli more closely during the exposure period.

Needless to say having any chronic disease may have major effects on cognition and behavior that relate to feeling fatigued, irritable, in pain, anxious, stressed and depressed. These could in turn generate differences between groups on the type of variables identified above (e.g., stress and depression affect immunity; Maier & Watkins, 1998). Moreover, it is possible that pre-existing differences in attitudes, education and socio-economic status could result in more prejudicial attitudes in one group relative to another. For these reasons we attempted to match both groups on age, gender and demographic profile, as well as collecting information pertinent to racism in general (Modern Racism Scale — Australian version; McConahay, 1986), health and functioning (SF12; Ware, Kosinski, & Keller, 1996), depression, anxiety and stress (DASS; Lovibond & Lovibond, 1995) and medical history. All of these factors were then taken into account when comparing the RA group to controls.

2. Method

2.1. Participants

Inclusion criteria for the RA group were being aged over 17, having received a medical diagnosis of RA, and living in Australia or New Zealand (necessary for the ethnocentric-related tasks and so we could offer links from our survey to local support sites if needed). To recruit people with RA, posts were placed on Australian/New Zealand based Internet sites frequented by people with this condition. Two hundred and twenty-two people started the survey, with 81 failing to complete more than the demographic and medical information. Nineteen participants were excluded for either not being residents of Australian/New Zealand or reporting a diagnosis of something other than RA (most being osteoarthritis). This left 122 participants in the RA group available for analysis.

Healthy control participants were recruited from the friends and family of RA group participants, via word and mouth, and from the Internet, targeting those with a similar demographic profile to the RA group (i.e., predominantly female, middle aged and older, Caucasian, Australian/New-Zealand born, generally well educated — see Table 1 and Results for demographic details). One hundred and thirty-seven people started the survey with 55 failing to complete more than the demographic and medical questions. Ten controls were excluded, one

Table 1
Demographic characteristics of the sample.

Variable	Healthy control group (n = 70)	Rheumatoid Arthritis group (n = 122)
Age in years, mean (SD)	45.8 (17.2)	45.2 (12.6)
Gender (column % women)	78.6	83.9
Education (column %)		
Postgraduate	14.3	8.2
Undergraduate	64.3	67.2
Secondary school	18.6	20.5
Primary school	2.9	4.1
Ethnicity (column %)		
Born in Australia/New Zealand	71.4	74.6
Caucasian	91.4	96.7
Relationship status (column %)		
Single	38.6	28.7
Married/de-facto	58.6	61.5
Other	2.9	9.8
Work status (column %)		
Full-time	47.1	45.1
Part-time	17.1	24.6
Not in paid employment	35.7	30.3

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