FEAR OF COVID-19: INSIGHTS FROM EVOLUTIONARY BEHAVIORAL SCIENCE

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Abstract

Fear of infectious disease is substantially different from that evoked by other medical conditions. Such a difference depends on psychological and behavioral adaptations shaped by natural selection throughout the evolutionary history of *Homo sapiens*. Selective pressures have favored the evolution of a behavioral immune system that is separate from, and complementary to, the physiological immune system. The two systems interact in a complex way. The psychological mechanisms (i.e. disgust and fear) involved in the behavioral immune system impact also on aspects that pertain to social psychology (i.e. xenophobia, conformism, and authoritarianism). Acknowledging the existence of psychological and behavioral adaptations to avoid infection has important implications for public health programs, including the necessity of fighting stigma and the dubious utility of trauma debriefing for healthcare workers facing the COVID-19 emergency.

Key words: COVID-19, fear of infection, behavioral immune system, stigma, xenophobia, conformism, authoritarianism, trauma debriefing

Introduction

Survey studies conducted in the last few years converge on showing that the most feared diseases by people living in rich countries are cancer, Alzheimer’s disease, heart disease, stroke, and diabetes (Harris Interactive, 2011; Bystad, Grønli, Lilleeggen, & Aslaksen 2016). It is very likely that if a survey were carried out in these days (I am writing this short paper in early spring of 2020), the results would be entirely different, with fear of COVID-19 ranking at the top of the list. The obvious explanation for such a dramatic change in people’s perception of what is the scariest disease points to the role of the media. The COVID-19 has correctly been treated as supremely important, dominating virtually every home page and broadcast. Stories on other diseases have all but disappeared, and some newsrooms have halted altogether coverage of medical conditions associated with the highest death toll (data from the Centers for Disease Control and Prevention [CDC] show that, every year in U.S., most people die from heart disease or cancer). That’s it? Can we conclude that the media and other sources of public information are totally responsible for the way people perceive disease risk and modify their behaviors? I don’t think so. In this paper, I will argue that fear of infectious disease is substantially different from that evoked by other medical conditions, and that such a difference depends on psychological and behavioral adaptations shaped by natural selection throughout the evolutionary history of *Homo sapiens*. Such a difference has major implications for social relationships and public health, as discussed in the final part of the paper.

Psychological defenses against infections: disgust and fear

Throughout their evolutionary history, humans have been exposed to a wide and varying array of pathogens. For those with access, modern medicine radically diminishes exposure to various pathogens. In developed countries, vaccination, better nutrition and improved public health have eliminated or decreased the prevalence of diseases that were common in the past. However, despite medical progress, global selective pressure imposed by pathogens is still very high today, with infectious diseases still representing one of the major causes of mortality (Karlsson, Kwiatkowski, & Sabeti, 2014). From an evolutionary perspective, this means that infectious diseases have exerted some of the strongest selective pressures during human evolution. Selection pressures have caused the evolution of a sophisticated suite of genetic and physiological adaptations that mediate resistance to infectious diseases. In molecular genetics, physiology, and general pathology, the study of the evolution and functioning of the immune system is an expanding research area (Fumagalli & Balloux, 2017). Yet, immunological defense is merely reactive because it is triggered only after the infection has occurred within the body. Schaller (2011) has convincingly demonstrated that selection pressures have reinforced our defenses against infections by causing the evolution of a behavioral immune system that is separate from, and complementary to, the physiological immune system. The behavioral immune system includes a set of proactive mechanisms that—by guiding organisms’ behavior—inhibit contact with...
pathogens in the first place. These mechanisms offer a sort of behavioral prophylaxis against infection. Like the physiological immune system, the behavioral immune system includes both detection and response mechanisms. When an external cue connoting infection risk (e.g. seeing another person with evident manifestations of infectious disease) is detected, it triggers a cascade of emotional, cognitive, and behavioral responses that minimize the infection risk (e.g. through social avoidance of people who appear to pose an infection risk). Disgust and fear are two key emotional responses to infection-connoting cues.

Disgust is a universal emotion that we have in common with many other animal species and that is already present at birth. It is a complex emotional reaction associated with specific facial expressions and mediated by definite neural pathways. What is relevant here is that the stimuli that easily trigger disgust are potential vehicles of infection: feces, fluids that are excreted or secreted from the body, rotten flesh, deteriorated food. From things to people it’s a short step. We are very sensitive to other people’s symptoms of disease, and we react to their sneezes, cough, vomit or bad breath with obvious repulsion. To avoid errors of omission (i.e., not avoiding what should be avoided), natural selection set a low threshold for disgust. Thus, sometimes disgust is triggered by innocuous stimuli because of wrong generalization. For example, congenital malformations may be as much disgusting as symptoms of infection, although they are absolutely harmless because not contagious (Troisi, 2017).

Unlike disgust (which is elicited by objective signs of probable infection), fear of infection is an emotional response that involves cognitive processes and social learning. Before the onset of clinical symptoms, infection is transmissible but invisible (Pappas, Kriaize, Giannakis, & Falagas, 2009). In the last decades, people have been exposed to alarming information on new threats such as AIDS, SARS, MERS, Ebola, avian influenza and now COVID-19. The combination of pervasiveness of pathogen-related concerns expressed in a wide range of media with our innate avoidance of symptoms of infection can lead to the social phenomenon named “geographic panic” (Tomes, 2000). In sum, fear of infection is likely to reflect a biologically predisposed form of learning.

There is a substantial difference between fear of infection and fear of degenerative diseases that rank at the top of the list in ordinary times. Cancer, Alzheimer’s disease, heart disease, stroke, and diabetes are evolutionary novelties because their etiology and pathogenesis are largely dependent on risk factors and life habits that are typical of modern environments (e.g., extended longevity, high calories diet, sedentary lifestyle, obesity, smoking, drinking alcohol, pollution, etc.). Our ancestors living in the natural environment were not exposed to those risk factors and, therefore, they had an infinitesimal likelihood of getting cancer or developing senile dementia. Yet, they had a very high likelihood of dying from an infection. The fact that degenerative diseases were not a serious threat to human health and survival throughout the evolutionary history of Homo sapiens is clearly demonstrated by the difficulty of implementing successful prevention programs. People know that smoking increases the risk of lung cancer or that obesity is associated with stroke and heart attack. Yet, people continue to smoke and eat unhealthy food because the neural circuits that mediate fear response in the human brain are not predisposed to detect cigarettes or hamburgers as dangerous stimuli. Fear of degenerative diseases is largely cognitive and prompted by cultural inputs. By contrast, fear of infection is deeply rooted in our emotional brain.

Social effects of evolved mechanisms to avoid infection

Because of the complexity of cognitive functioning and social organization in our own species, the psychological mechanisms involved in the behavioral immune system impact on aspects that, apparently, are not linked with infection avoidance. These aspects pertain to social psychology and include xenophobia, conformism, and authoritarianism (Schaller, 2019).

There is evidence that, when the threat of pathogen infection is salient, people are likely to be hypersensitive to inferential cues that discriminate between familiar and foreign persons. When those cues are detected, they trigger aversive and discriminative reactions (Schaller, Murray, & Bangerter, 2015). The combination of two evolutionary factors (i.e. the social ecology of ancestral humans and the smoke detector principle) can explain the link between the behavioral immune system and xenophobia. For most of their evolutionary history, humans lived in widely dispersed, nomadic, small populations made up of individuals with a high degree of familiarity. Such a kind of social ecology minimized the risk of getting infectious diseases. By contrast, contact with foreign people who were host of exotic pathogens could be especially virulent for a local population. There are many historical examples of imported introduction of novel and deadly diseases to groups that had little resistance to them (e.g. Maya, Toltec, Quechua, and many other peoples in Mesoamerica died in great numbers from measles wherever they encountered Spanish invaders). Obviously, the identification of any foreign person as a potential source of contagion is often an erroneous over-generalization but, from an adaptive perspective, false-negative errors (an infectious foreign is erroneously perceived as healthy) are costlier than false-positive errors (a healthy foreign is erroneously perceived to be infectious). This is the logic that guides many adaptive defenses according to the smoke detector principle (i.e. evolved systems that regulate protective responses often give rise to false alarms and apparently excessive responses).

Xenophobia elicited by fear of infection is well illustrated by the tendency in public health history to associate new infectious diseases with foreign nationals and foreign countries. For example, despite not originating in Spain, the 1918 influenza pandemic is commonly known as the “Spanish flu” and, on these days, President Donald Trump is calling the coronavirus the “Chinese virus”. Ethnic discrimination flourishes during infective outbreaks. In the wake of cholera and typhus outbreaks in 1892, New York City officials selectively quarantined Jewish immigrants, whereas Italians arriving on the same boat were detained for only a brief time (Hoppe, 2018). Interestingly, the target of xenophobic attacks changes depending on the ethnic background of those who perceive the infection hazard: “Since the outbreak of the coronavirus (and the disease it causes, COVID-19) began, reports of racism toward East Asian communities have grown apace. More recently, this has expanded beyond East Asian populations: Thailand’s public-health minister yesterday appeared to lash out at white foreigners who he said were dirty and spreading the virus in the country, adding that people should be more afraid of Westerners than Asians.” (https://www.thelatin.com/international/archive/2020/03/coronavirus-covid19-
Implications for public health strategies

Acknowledging the existence of psychological and behavioral adaptations to avoid infection invites some reflections that may be relevant to public health strategies. Prevention and treatment of infectious diseases can benefit from a greater awareness of the interactions between the two immune systems, the physical and the behavioral. For example, there is evidence that, when social distancing results in social isolation, the functionality of the physical immune system is reduced (Hawkley & Cacioppo, 2010). By contrast, the activity of the physical immune system is enhanced by visual exposure to symptoms of infectious disease in others (Schaller, Miller, Gervais, Yager, & Chen 2010).

The psychological responses activated by the behavioral immune system evolved to afford adaptive benefits in a socio-ecological environment that was substantially different from modern environments. In small bands made up of individuals with a high degree of familiarity, fear of immigrants and social conformity were effective means to reduce the risk of contagion. In the “global village” where we live, xenophobia, intolerance of dissent, and preference for authoritarianism are useless reactions that can impact negatively on social organization. This suggests that fighting against stigma should be a standard component of coronavirus campaigns, like the CDC is doing in these days (https://www.cdc.gov/coronavirus/2019-ncov/daily-life-coping/share-facts.html).

Finally, the decision to implement crisis interventions for small group of healthcare workers who are facing the COVID-19 emergency should be carefully evaluated in the light of findings showing even an increase in post-traumatic stress disorder (PTSD) symptoms after trauma debriefing compared with control treatments (Locher, Koechlin, Gaab, & Gerger, 2019). In debriefing interventions, the trauma experience is discussed with a focus on distinguishing between facts, cognitions, and emotions. Such a distinction is difficult to do when dealing with an ancestral reaction like fear of infection. In addition, treating fear of infection as an emotional response worthy of professional intervention may trigger the undesirable effects of pathologizing a normal condition. The information regarding potentially occurring negative reactions after trauma experience may increase the expectation of the occurrence of negative reactions, which may in turn induce the development of such negative reactions (van Emmerik, Kamphuis, Hulsbosch & Emmelkamp, 2002).
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