Mechanisms by Which Parasites Influence Cultures, and Why They Matter

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At least four conceptually distinct mechanisms may mediate relations between parasite stress and cultural outcomes: Genetic evolution, developmental plasticity, neurocognitive flexibility, and cultural transmission. These mechanisms may operate independently, or in conjunction with one another. Rigorous research on specific mediating mechanisms is required to more completely articulate implications of parasite stress on human psychology and human culture.

[Commentary on Fincher & Thornhill]

Increasingly, the question is not *whether* parasite prevalence has cultural consequences, but *why*: Exactly what mediating mechanisms account for relations between the parasite load in the local ecology and the traits, values, and social norms observed within the human population occupying that ecology?

As currently articulated, the parasite stress model predicts outcomes observed at a societal level (e.g., religiosity, strong family ties, collectivistic values, authoritarian governments), but does not specify the exact mechanisms through which those outcomes emerge. Societal outcomes don't just happen. They are emergent products of individuals' actions and interactions, which are in turn products of individuals' cognitions, emotions, and behavioral dispositions. These, in turn, are products of the developmental process through which genetic material builds bodies. Multiple levels of analysis are implicated, along with multiple conceptually distinct mechanisms that may be influenced by parasite stress. One mechanism is *genetic evolution*: Heritable traits that reduce contact with parasites (and the alleles associated with those traits) may become more common in populations occupying ecologies characterized by high levels of parasite stress. A second mechanism is *developmental plasticity*: The phenotypic consequences of genetic information depend on whether and how the genes are expressed during development; genes for traits that reduce contact with parasites may be expressed more readily in ecologies characterized by high levels of parasite stress. A third mechanism is *neurocognitive flexibility*: Human nervous systems are adaptively designed to detect threat-connoting cues in the immediate perceptual context, and to respond flexibly depending on the presence of these cues; responses that reduce contact with parasites may occur more frequently, and more strongly, when perceptual processes register a greater prevalence of cues connoting infection. A fourth mechanism is *cultural transmission*: Societal outcomes depend in part upon the exact nature of interpersonal influence that occurs when individuals interact and communicate with one another;

influence on behavioral tendencies that limit contact with parasites may occur more readily when people perceive greater threat of infection.

There is evidence that implicates each of these mechanisms as a plausible route through which parasite stress may lead to cultural differences (Schaller & Murray, 2011). And, although conceptually independent, these mechanisms may also influence each other. (Neurocognitive flexibility has implications for cultural transmission; cultural transmission reshapes the social ecology and so has implications for genetic evolution; and so forth.) But just because they have potential implications for one another, this does not mean they are all equally implicated as mediating mechanisms in the causal link between parasite stress and cultural outcomes. Nor is it logically necessary for each cultural outcome to result equally from each of the plausible mediating mechanisms. Highly heritable individual-level traits (such as religiosity; Waller, Kojetin, Bouchard, Lykken, & Tellegen, 1990) may be more strongly influenced by genetic mechanisms, whereas societal outcomes such as democratization and the strength of family ties may be more strongly influenced by cultural transmission. Each specific mechanism must be considered, and tested, as a possible mechanistic explanation for each specific cultural outcome predicted by the parasite stress model. By doing so can we address additional questions about the effects of parasites on human culture.

One question pertains to the time lag between ecological and cultural change. Parasite ecologies can change quickly, especially when people apply technological interventions (e.g., vaccination programs) toward the purpose of eliminating infectious diseases. Research linking parasite-stress to cultural outcomes offers the intriguing implication that these interventions may also have unintended consequences on a broad range of cultural outcomes (secularization, democratization, the reduction of xenophobia, the disintegration of family ties, etc). If so, how quickly might this happen? To the extent that an effect is mediated by population-level changes in gene frequencies, it may take a very long time indeed. To the extent it is mediated by developmental plasticity, it may take just a single generation. To the extent it is mediated by neurocognitive flexibility, it may take virtually no time at all. And to the extent it is mediated by cultural transmission, the implications for cultural change are more complicated. Interpersonal communication processes allow for rapid diffusion of novel behavioral norms. However, the psychology of interpersonal influence often emphasizes conformity and resistance to change instead; these conformity pressures tend to be especially strong under conditions of high parasite stress (Murray, Trudeau, & Schaller, 2011).

A second question pertains to a broader set of possible psychological and societal outcomes. Any single mediating mechanism may not only to produce outcomes that are predicted by the parasite stress model, but also additional outcomes that aren't. Genes that promote individual-level traits linked to the reduction of infection (e.g., religiosity, xenophobia) probably do so because of their phenotypic expression within specific neurotransmitter systems (e.g., the serotonin transmitter system; Chaio & Blizinsky, 2010). Each neurotransmitter system has wide-ranging implications for additional traits too, many of which have no obvious bearing on infection-reduction at all. These consequences cannot be predicted by the parasite stress model alone; meaningful explanation of these additional consequences requires explicit inquiry into the specific mediating mechanism. Analogously, religious practices that reduce infection risk are neither practiced nor preached in isolation from other practices. For example, in many societies, Islamic law requires

that women wear veils and heavy clothing, which reduces incidence of mosquito-borne infections (Russell, 1952); but this particular practice is bundled into a broader set of religious rituals, many of which are infection-irrelevant. Cultural transmission mechanisms tend to operate on these bundles, rather than on single practices, with the result that many infection-irrelevant norms may also be linked to parasite-stress. Again, any real understanding of these cultural outcomes requires careful articulation of the specific mechanisms that produces them.

Bottom line: It is only by considering mediating mechanisms more explicitly that we can more completely predict, explain, and appreciate the implications of parasite stress on people, and on the cultures that people create.

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