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## Commentary

## Look what the cat dragged in: do parasites contribute to human cultural diversity?

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If human culture emerges from the modal personality of a population, can global variation in parasitism that affects personality lead to cultural diversity among nations? The answer could help explain why people seem to vary so much from one land to another. Thomas et al. (2005) review how parasites manipulate behaviour, including human behaviour. To quote them, "The rabies virus lives in the brain, affording the virus ample opportunity to directly affect host behaviour. Rabid animals do show changes in behaviour, including increased aggression and biting." Rabies affects a wide range of mammals and the aggressive biting associated with furious rabies appears to increase transmission. The personality transformation of infected humans can be horrifying, transforming loved ones into thrashing, baying beasts. Not coincidentally, in Europe, past periods of rabies outbreaks correspond to increases in werewolf trials. Although rabies can have a dramatic effect, the present rarity of human rabies cases and the availability of a vaccine, means that the behavioural effects of rabies are primarily an illustrative curiosity.

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Other parasites are much more common in human populations. Some may have the ability to enact subtle, but long-term, changes in behaviour – to the extent of changing personality. The most notable example is Toxoplasma gondii. This parasite is primarily of concern because it can cause pathology under some circumstances. Obstetricians warn expectant mothers that this parasite can pass through the womb and blaze through an immunologically immature fetus (resulting in blindness, permanent central nervous system damage or death). Toxoplasmosis is also a significant mortality source of the immune suppressed. Nevertheless, clinical cases are rare compared with latent toxoplasmosis that occurs, on average, in 40% of adults (average estimates vary). Although the medical profession treats these latent cases as asymptomatic and clinically unimportant (Webster, 2001), accumulating evidence indicates that the infected 40% behave in subtle, but statistically different ways from the uninfected 60%. Seroprevalence varies greatly among nations, meaning that variation in T. gondii can lead to variation in the collective (modal) personality among nations.

To explain how *T. gondii* affects human personality, it helps to describe how it gets from host to host.

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In its final host, the cat, the T. gondii parasite lives inside a cell lining the gut. An infected cat sheds numerous oocysts that contaminate the soil or kitty litter. Cats can accidentally ingest oocysts and infect themselves. But given cats' notoriously good hygiene, it is unclear whether direct transmission happens frequently. Toxoplasma exploits an alternative, indirect mode of transmission because oocvsts can also infect other mammals and birds. In the non-feline host, the oocyst releases sporozoites that enter cells, divide rapidly (tachyzooites), and ultimately invade muscle and brain tissue where division slows or stops (bradyzooites). The dividing phase can lead to flu-like symptoms (fever, lymph node swelling), but soon becomes dormant because the host immune system limits division (and provides lifetime immunity to new infections). Infections can readily transfer to any carnivore through ingestion of tissue-dwelling cysts in raw flesh. After ingestion, the intracellular tachyzooite and bradyzooite phases reestablish in muscles and the brain. However, if the carnivore is a cat, T. gondii comes full circle by invading the intestinal lining and producing oocysts.

As Thomas et al. (2005) point out, this sort of trophically transmitted parasite should be under selection to alter the behaviour of the intermediate host to increase the chance of predation by a final host. Studies with rats infected with T. gondii provide compelling evidence for manipulation (Webster, 2001). Rats normally view the world as a dangerous place. They avoid the light and stay clear of new things. The scent of cats sends them scampering. In contrast, infected rats are more active, less fearful (Hay et al., 1983, 1984), and first to enter traps (Webster et al., 1994). T. gondii also specifically reduces a rat's innate fear of cats and their associated smells; infected rats actually prefer the scent of cat urine over the scent of rabbit urine (Berdoy et al., 2000). The behavioural changes of infected rats seem all about increasing the risk of predation by cats; T. gondii does not alter irrelevant behaviours related to social status or mating success (Berdoy et al., 1995). Although the mechanism of behaviour modification is not known, mice infected with T. gondii have elevated levels of dopamine (Stibbs, 1985), a neurotransmitter known to alter behaviour.

The tricks that *T. gondii* uses on its rat host stand little chance of working on humans. Still, there is no cost for the parasite to try. Moreover, the efforts are

bound to do something because the rat and human brain are similar enough that the same neurotransmitters act to alter behaviour. However, the effects of *T. gondii* in humans differ qualitatively from the effects on rats. In particular, novelty-seeking behaviour decreases with *T. gondii* in men while it increases in rats. In addition, the vastly larger mass of the human brain may be more difficult to manipulate.

I am usually confident that I can easily do things that most people would consider dangerous (such as driving an automobile fast on a wet or icy road) states a true-false question on the Cloninger's Temperament and Character Inventory personality test. A parasite could affect your answer. At least that is what recent studies about the link between personality and parasitic infection indicate (Flegr et al., 2003). Human temperament, a key and heritable factor of personality, is associated with varying levels of the same neurotransmitters that occur in rat brains (Cloninger et al., 1993). People with latent toxoplasmosis score themselves differently in self administered personality profiles than do uninfected people (Flegr and Hrdy, 1994; Flegr et al., 1996, 2000). Infected men seem more dogmatic, less trusting of others, less respectful of rules, more orderly and more jealous. Unlike infected rats, infected men are more wary and less impulsive (Flegr et al., 2003). In stark contrast, infected women are more likely to score themselves as warm-hearted, easy-going, conscientious, persistent, insecure and moralistic (Flegr et al., 2000). Infected women are also more likely to have many friends and romantic partners and like to shop for clothes. Both men and women are more prone to guilt when infected. Personality changes are greater in individuals that have had infections for a long time, helping to discredit the hypothesis that personality determines exposure risk. Perhaps the most dangerous thing about latent T. gondii is that it slightly reduces the ability to focus on a task (Havlicek et al., 2001). This may explain why infected people are nearly three times more likely to crash their cars (Flegr et al., 2002).

Modern pharmaceuticals provide a range of popular therapies that can alter personalities to the psychological benefit of patients. Selective serotonin reuptake inhibitors (e.g., Zoloft, Prozac, Paxil, and Luvox) are now widely used to treat depression and anxiety. Now imagine that you discover you are seropositive for *Toxoplasma*. Would you, given the chance, cure yourself to get your innate personality back? If you answered

yes, unfortunately, no complete cure is available. How about if you were not infected? Could a dose of toxoplasmosis improve your life? Some women might find it tempting. Though it seems unlikely we will be seeing a market for toxoplasmosis therapy, it is hard to conclude that uninfected women have more fit personalities than do infected women.

Many factors influence whether you get toxoplasmosis. The first has to do with the abundance of oocysts around you. Cysts live longer in humid, low altitude regions, especially at mid-latitudes with infrequent freezing and thawing (Dubey and Beattie, 1988; Dubey, 1974; Walton et al., 1966). Homes with or near cats (Kean et al., 1969) or lots of other people (Jones et al., 2001) have more oocytes around them. Working with the soil (Jones et al., 2001) and poor hygiene increases an individual's exposure to oocysts while a taste for rare or undercooked meat increases an individual's exposure to pseudocysts (Baril et al., 1999). The probability of contact accumulates with age and is higher for men than for women (Jones et al., 2001). It is easy to see how climate, geography, economic standing, and culinary traditions can interact to alter the prevalence of T. gondii. It is also obvious that these risk factors vary greatly from place to place.

Differences in risk cause prevalences of toxoplasmosis to vary widely among populations. For example, in the United States, the prevalence of *Toxoplasma* is higher in the Northeast (29%) than in the south (23%) and Midwest (21%) and lowest in the arid West (18%) (Jones et al., 2001). Not surprisingly, there is even more variation among nations. This ranges from 81% in France (where raw beef – steak tartare – is a traditional dish) and 79% in Brazil (where climatic conditions are favorable for oocyst survival), to 7% in Japan (Nguyen and Bigaignon, 1994). It seems possible that differences in personality between my wife and I could partly result from her having grown up in humid, toxoplasmosis rich Brazil and me being raised in arid, toxoplasmosis poor California.

Ethnic and linguistic groups often exhibit culture (ideas, beliefs, customs, institutions, tools, art, etc.) distinct from neighboring groups (Terpstra and David, 1985). Variation in the four principal dimensions of culture (individualism, sex roles, risk taking and class distinction) corresponds with variation in modal personality traits among cultures (Hofstede and McCrae, 2004). Although there is tremendous intra-national

variation in personality, this linkage often leads to national stereotypes of personalities (the formal Japanese, macho Italian, precise German, informal American, etc.) that emerge from the concept of national culture (Inkeles and Levinson, 1969). The traditional view in the field of anthropology explains the association between personality and culture by arguing that culture shapes the modal personality of a population through myriad top-down effects of environmental conditioning and experience. However, it is difficult to distinguish between the cause and effect relationships of modal personality and national culture. It seems comparably plausible that the modal personality of a populace can help shape culture from the bottom up.

If T. gondii can alter individual personality, and modal personality can shape culture, then T. gondii may be one of the factors that shape culture. Similarly, variation in climate, diet and other risks of exposure lead to variation in risk. This could contribute, in part, to the diversity of culture. Perhaps parasites and their effect on behaviour have a bigger impact on our human world that presently imagined. Although enough evidence exists to conclude that *Toxoplasma* affects personality, it is unclear how much of the variation in personality it can explain, and whether these effects can have a detectable effect on cultural variation among populations. If Toxoplasma does affect human culture, can we conclude that Toxoplasma is culturally deleterious? Without it might the result be a France a little less French or a Brazil a little less Brazilian?

Thomas et al. (2005) point out that parasites can have pervasive and powerful effects on the behaviour of their hosts. Particularly for trophically transmitted parasites, these behavioural changes appear advantageous for the parasite. The authors note that humans too are hosts for manipulative parasites. The speculation that this may have important implications for human culture is testable and provides direction for where we should go from here.

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