Can the common brain parasite, Toxoplasma gondii, influence human culture?

Kevin D Lafferty

Proc. R. Soc. B 2006 273, 2749-2755
doi: 10.1098/rspb.2006.3641
Can the common brain parasite, *Toxoplasma gondii*, influence human culture?

Kevin D. Lafferty*

Western Ecological Research Centre, United States Geological Survey, Marine Science Institute, University of California, Santa Barbara, CA 93106, USA

The latent prevalence of a long-lived and common brain parasite, *Toxoplasma gondii*, explains a statistically significant portion of the variance in aggregate neuroticism among populations, as well as in the ‘neurotic’ cultural dimensions of sex roles and uncertainty avoidance. Spurious or non-causal correlations between aggregate personality and aspects of climate and culture that influence *T. gondii* transmission could also drive these patterns. A link between culture and *T. gondii* hypothetically results from a behavioural manipulation that the parasite uses to increase its transmission to the next host in the life cycle: a cat. While latent toxoplasmosis is usually benign, the parasite’s subtle effect on individual personality appears to alter the aggregate personality at the population level. Drivers of the geographical variation in the prevalence of this parasite include the effects of climate on the persistence of infectious stages in soil, the cultural practices of food preparation and cats as pets. Some variation in culture, therefore, may ultimately be related to how climate affects the distribution of *T. gondii*, though the results only explain a fraction of the variation in two of the four cultural dimensions, suggesting that if *T. gondii* does influence human culture, it is only one among many factors.

**Keywords:** personality; nations; masculinity; neuroticism; uncertainty avoidance

1. INTRODUCTION

Can an infectious disease indirectly alter human culture through its effects on personality? Contemporary human cultures have been described quantitatively using four principal cultural dimensions (individualism, sex roles, uncertainty avoidance and class distinction). These cultural dimensions correspond with the aggregate personalities measured at the national level (Hofstede & McCrae 2004). The traditional view of the link between culture and personality is that cultural dimensions might alter individual personality through environmental conditioning and experience, but there is also support for the hypothesis that aggregate personality might shape cultural dimensions through the collective behaviour of individuals (Hofstede & McCrae 2004). Although the extent of environmental determinism is a contentious issue (Diamond 2005), environmental factors that affect individual personalities could, theoretically, influence culture from the bottom-up.

Infectious disease is one untested but particularly pervasive environmental factor that could affect aggregate personality. In particular, humans with latent infections of the common protozoan parasite *Toxoplasma gondii* appear to experience a variety of long-term personality changes (Webster 2001). For instance, in infected women, intelligence, superego strength (rule-conscious, dutiful, conscientious, conforming, moralistic, staid and rule-bound) and affectothymia (warm, outgoing, attentive to others, kindly, easy-going, participating and likes people) are higher, while infected men have lower intelligence, superego strength and novelty-seeking (low novelty-seeking indicates rigid, loyal, stoic, slow-paced and frugal personalities); both infected men and women have higher levels of guilt-proneness (they tend to be more apprehensive, self-doubting, worried, guilt prone, insecure, worrying and self-blaming; Flegr & Hrdy 1994; Flegr et al. 1996, 2000, 2003).

*Toxoplasma gondii* appears to manipulate human personality as a result of adaptations that normally help complete its complex life cycle from intermediate hosts to the final host, a cat (Webster 2001). The reproductive phase of this protozoan lives in the cells that line the intestine of a feline. Oocysts shed to the soil with the cat’s faeces can, through ingestion, directly infect cats or encyst in the brain and other tissues of a wide range of warm-blooded vertebrates, including humans (Beverley 1976). Tissue cysts in intermediate hosts are long-lived and infective to carnivores (including humans). If the carnivore is not a feline, then *T. gondii* re-encysts—a strategy that can continue up the food chain, though in many cases, particularly for humans, such hosts are often ‘dead ends’ for the parasite. *Toxoplasma gondii* can mature, produce oocysts and complete its life cycle only if the carnivore is a felid. Most infected humans initially suffer from only mild flu-like symptoms (severe pathology can occur in a foetus), whether infected through soil contact or by eating infected meat, but soon the parasites become dormant in the brain and other tissues (though there is often some slow replication and occasional reactivation to the invasive tachyzoite state, particularly in immunocompromised hosts).

Parasites are under selection to increase the chance that final hosts eat intermediate hosts (Lafferty 1999). As a result, many parasites alter the behaviour of intermediate hosts to increase predation risk (Moore 2002). For example, *T. gondii* appears to manipulate rodent behaviour in sophisticated ways that would increase transmission to domestic cats (Webster 2001). Rodents infected with *T. gondii* are more active (Hay et al. 1983, 1984), first to enter traps (Webster et al. 1994), and less fearful of cats and

---

*lafferty@lifesci.ucsb.edu
their associated smells (Berdoy et al. 2000). Mice infected with T. gondii have elevated levels of dopamine (Stibbs 1985), a neurotransmitter known to alter novelty-seeking (Benjamin et al. 1996; Ebstein et al. 1996) and neuroticism (Lee et al. 2005). One postulated mechanism for the effects of infection on personality in humans is the local immune response in the brain that is required to keep T. gondii dormant. This response alters cytokine levels, which then influence the level of neuromodulators (Novotna et al. 2005) such as increasing dopamine (Skalova et al. 2005). Both anti-toxoplasma drugs and dopamine antagonists normalize the behaviour of infected rats, indicating a neurological mechanism for how T. gondii alters behaviour (Webster et al. 2006). Currently, cats rarely eat humans, so there should be little selective advantage for T. gondii to specifically manipulate human behaviour. Still, T. gondii cysts infecting a human have nothing to lose, evolutionarily speaking, in trying manipulative strategies adaptive in other intermediate host species.

The seroprevalence (percentage of people with antibodies to latent infections) of T. gondii varies geographically nearly from 0 to 100% (Tenter et al. 2000), suggesting that T. gondii could lead to variation in aggregate personality among populations (Lafferty 2005). In other words, the average personality of a population might be shifted if a higher proportion of individuals are infected with T. gondii. The ability to detect statistically an effect of T. gondii on aggregate personalities of populations will depend on how strongly the parasite affects individual personality, the extent of variation in prevalence among populations and the consistency of the personality change between men and women. This begs the question: could aspects of human culture result from a parasite selected to predispose its host to predation by cats (Lafferty 2005)? Here, I report associations between the prevalence of T. gondii infection and aggregate personality that could explain some of the variation in cultural dimensions among human populations.

2. MATERIAL AND METHODS

Toxoplasma gondii seroprevalence increases strongly with age in most of the human populations and can vary by gender (Jones et al. 2001). Because testing is most often conducted on women to determine immunity during pregnancy, data on women of childbearing age make up a large fraction of available studies. Comparing seroprevalence only among this subgroup helped control for gender differences and narrowed the age range of subjects. It is important to acknowledge that using data from women could lead to type II error in countries (like Japan) with strong gender differences in seroprevalence. Studies were grouped by nations to be comparable with measures of aggregate personality and cultural dimension. When more than one study was available from a nation, I used the average. Within-nation variation in prevalence was low compared with among-nation variation in prevalence.

Subjects tested for T. gondii seroprevalence in countries with older ages of pregnancy have had a longer exposure period, on average, before testing. One way to correct this is to adjust prevalence to a standard age (I standardized to an age of 22 years because personality assessments were taken from college-aged respondents). The standardized prevalence (as a proportion) of a nation at an age of 22 years was \( P_{22} = 1 - (1 - P) \frac{22}{\text{age}} \) (derivation available on request), where \( P \) is the reported prevalence (as a proportion) and age was the average age of pregnancy (from the Population Division of the Department of Economic and Social Affairs of the United Nations Secretariat) or, when available, the age provided by the study.

It is difficult to predict the effect of T. gondii infection on personality at the population level because men and women often respond in opposite directions to infection. Guilt-proneness (using Cattell’s 16-factor model) is the only response that is consistent in both men and women, increasing in infected individuals (Flegcr et al. 2000). To make predictions about guilt-proneness at the population level, however, first required converting from Cattell’s 16-factor model to the five-factor NEO-PI-R model. Because guilt-proneness corresponds to the associated five-factor NEO-PI-R measure of neuroticism (John 1990; Griffin & Bartholomew 1994; McCrae & Costa 1996; Digman 1997), I predicted that populations with...
A high prevalence of *T. gondii* would have higher aggregate neuroticism. Aspects of human culture associated with neuroticism are male control, materialism, rules and structure (Hofstede & McCrae 2004), leading to the additional prediction that *T. gondii* could increase the cultural dimensions of ‘masculine’ sex roles and uncertainty avoidance. Although each cultural dimension associates with aggregate neuroticism, sex roles and uncertainty avoidance were not significantly associated with each other in this study.

To test these predictions, I used published data on cultural dimension (Hofstede 2001) and aggregate personality (McCrae & Terracciano 2005) collected in those countries for which there were also published data on *T. gondii* seroprevalence from women of childbearing age (table 1). In addition, I used an independent assessment of aggregate neuroticism by Lester (2000), which is not significantly correlated with neuroticism as measured by the five-factor NEO-PI-R model ($R \approx 0.04$).

The hypothesis that disease affects population-level variation in culture could only be investigated indirectly through correlation methods. General linear models were used to determine the extent to which the national seroprevalence of latent *T. gondii* (the independent variable) predicted the dependent variables. Because some researchers have argued that culture-level correlations need to control for economics (Hofstede 2001; Leung & Bond 2004), I incorporated *per capita* gross domestic product (GDP) as a covariate in the analyses (this covariate was removed if non-significant). For the two tests of aggregate neuroticism, significance was evaluated with a Bonferroni correction so that the adjusted critical alpha was 0.025. The same method was followed for the two associations with cultural dimension. All tests

Table 1. Age-adjusted prevalence, aggregate neuroticism (N), the cultural dimensions of uncertainty avoidance (U) and masculinity (M) among nations. Original references for most of the *T. gondii* data used are derived from Tenter et al. (2000). Prevalence was age-adjusted to 22 years. Key to references: 1, Tenter et al. (2000); 2, Koskiniemi et al. (1992); 3, Széna et al. (1997); 4, Konishi et al. (2000); 5, Flynn (1979); 6, Franklin et al. (1993); 7, Win et al. (1997); 8, Vlasveld et al. (2001); 9, Morris & Groxson (2004); 10, Cantella et al. (1974); 11, Malgorzata et al. (2001); 12, Batet et al. (2004); 13, Évengard et al. (2001); 14, Jacquier et al. (1995); 15, Jones et al. (2001); 16, Bobic et al. (1998); 17, Lester (2000); 18, McCrae & Terracciano (2005); 19, Hofstede (2001).

<table>
<thead>
<tr>
<th>Country</th>
<th>Prevalence (%)</th>
<th>N&lt;sup&gt;17&lt;/sup&gt;</th>
<th>N&lt;sup&gt;18&lt;/sup&gt;</th>
<th>U&lt;sup&gt;19&lt;/sup&gt;</th>
<th>M&lt;sup&gt;19&lt;/sup&gt;</th>
<th>per capita GDP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Argentina</td>
<td>52.7&lt;sup&gt;1&lt;/sup&gt;</td>
<td>—</td>
<td>51.3</td>
<td>86</td>
<td>56</td>
<td>3.8</td>
</tr>
<tr>
<td>Australia</td>
<td>28.0&lt;sup&gt;1&lt;/sup&gt;</td>
<td>—</td>
<td>48.6</td>
<td>51</td>
<td>61</td>
<td>19.7</td>
</tr>
<tr>
<td>Austria</td>
<td>36.0&lt;sup&gt;1&lt;/sup&gt;</td>
<td>1.26</td>
<td>48.3</td>
<td>70</td>
<td>79</td>
<td>23.5</td>
</tr>
<tr>
<td>Belgium</td>
<td>46.8&lt;sup&gt;1&lt;/sup&gt;</td>
<td>0.95</td>
<td>49.6</td>
<td>50</td>
<td>54</td>
<td>22.6</td>
</tr>
<tr>
<td>Brazil</td>
<td>66.9&lt;sup&gt;1&lt;/sup&gt;</td>
<td>—</td>
<td>53.7</td>
<td>76</td>
<td>49</td>
<td>2.8</td>
</tr>
<tr>
<td>China</td>
<td>24.3&lt;sup&gt;1&lt;/sup&gt;</td>
<td>—</td>
<td>53.1</td>
<td>40</td>
<td>66</td>
<td>1.0</td>
</tr>
<tr>
<td>Colombia</td>
<td>54.2&lt;sup&gt;1&lt;/sup&gt;</td>
<td>—</td>
<td>50</td>
<td>64</td>
<td>1.8</td>
<td></td>
</tr>
<tr>
<td>Croatia</td>
<td>37.4&lt;sup&gt;1&lt;/sup&gt;</td>
<td>—</td>
<td>49.3</td>
<td>—</td>
<td>4.6</td>
<td></td>
</tr>
<tr>
<td>Czech Rep.</td>
<td>26.6&lt;sup&gt;1&lt;/sup&gt;</td>
<td>—</td>
<td>51.4</td>
<td>74</td>
<td>57</td>
<td>5.6</td>
</tr>
<tr>
<td>Denmark</td>
<td>22.0&lt;sup&gt;1&lt;/sup&gt;</td>
<td>1.22</td>
<td>50.3</td>
<td>23</td>
<td>16</td>
<td>29.9</td>
</tr>
<tr>
<td>Egypt</td>
<td>24.3&lt;sup&gt;1&lt;/sup&gt;</td>
<td>1.86</td>
<td>—</td>
<td>—</td>
<td>1.4</td>
<td></td>
</tr>
<tr>
<td>Finland</td>
<td>15.2&lt;sup&gt;1&lt;/sup&gt;</td>
<td>0.09</td>
<td>—</td>
<td>59</td>
<td>26</td>
<td>24.1</td>
</tr>
<tr>
<td>France</td>
<td>45.0&lt;sup&gt;1&lt;/sup&gt;</td>
<td>1.79</td>
<td>52.7</td>
<td>86</td>
<td>43</td>
<td>22.8</td>
</tr>
<tr>
<td>Germany</td>
<td>42.7&lt;sup&gt;1&lt;/sup&gt;</td>
<td>0.59</td>
<td>48.1</td>
<td>65</td>
<td>66</td>
<td>21.8</td>
</tr>
<tr>
<td>Greece</td>
<td>26.9&lt;sup&gt;1&lt;/sup&gt;</td>
<td>0.43</td>
<td>—</td>
<td>112</td>
<td>57</td>
<td>11.5</td>
</tr>
<tr>
<td>Hungary</td>
<td>58.9&lt;sup&gt;3&lt;/sup&gt;</td>
<td>2.3</td>
<td>53.8</td>
<td>82</td>
<td>88</td>
<td>5.6</td>
</tr>
<tr>
<td>Indonesia</td>
<td>46.2&lt;sup&gt;1&lt;/sup&gt;</td>
<td>—</td>
<td>50</td>
<td>48</td>
<td>46</td>
<td>0.7</td>
</tr>
<tr>
<td>Ireland</td>
<td>25.0&lt;sup&gt;7&lt;/sup&gt;</td>
<td>—</td>
<td>50.1</td>
<td>35</td>
<td>—</td>
<td>24.4</td>
</tr>
<tr>
<td>Israel</td>
<td>16.7&lt;sup&gt;7&lt;/sup&gt;</td>
<td>1.3</td>
<td>81</td>
<td>47</td>
<td>16.4</td>
<td></td>
</tr>
<tr>
<td>Italy</td>
<td>32.6&lt;sup&gt;1&lt;/sup&gt;</td>
<td>0.52</td>
<td>52.6</td>
<td>75</td>
<td>70</td>
<td>18.7</td>
</tr>
<tr>
<td>Jamaica</td>
<td>52.1&lt;sup&gt;1&lt;/sup&gt;</td>
<td>—</td>
<td>13</td>
<td>68</td>
<td>2.7</td>
<td></td>
</tr>
<tr>
<td>Japan</td>
<td>12.3&lt;sup&gt;7&lt;/sup&gt;</td>
<td>0.45</td>
<td>50.7</td>
<td>92</td>
<td>95</td>
<td>33.5</td>
</tr>
<tr>
<td>The Netherlands</td>
<td>24.5&lt;sup&gt;7&lt;/sup&gt;</td>
<td>0.39</td>
<td>48.6</td>
<td>53</td>
<td>14</td>
<td>22.9</td>
</tr>
<tr>
<td>New Zealand</td>
<td>26.9&lt;sup&gt;7&lt;/sup&gt;</td>
<td>0.11</td>
<td>49</td>
<td>58</td>
<td>13.8</td>
<td></td>
</tr>
<tr>
<td>Norway</td>
<td>8.6&lt;sup&gt;7&lt;/sup&gt;</td>
<td>0.16</td>
<td>47.4</td>
<td>50</td>
<td>8</td>
<td>42.2</td>
</tr>
<tr>
<td>Peru</td>
<td>32.5&lt;sup&gt;10&lt;/sup&gt;</td>
<td>—</td>
<td>48.5</td>
<td>87</td>
<td>42</td>
<td>2.1</td>
</tr>
<tr>
<td>Poland</td>
<td>46.5&lt;sup&gt;11,11&lt;/sup&gt;</td>
<td>—</td>
<td>50.7</td>
<td>93</td>
<td>64</td>
<td>4.7</td>
</tr>
<tr>
<td>South Korea</td>
<td>4.3&lt;sup&gt;7&lt;/sup&gt;</td>
<td>0.43</td>
<td>48.4</td>
<td>85</td>
<td>85</td>
<td>6.8</td>
</tr>
<tr>
<td>Slovenia</td>
<td>30.9&lt;sup&gt;9&lt;/sup&gt;</td>
<td>—</td>
<td>50.6</td>
<td>—</td>
<td>—</td>
<td>10.4</td>
</tr>
<tr>
<td>Spain</td>
<td>22.7&lt;sup&gt;1,12&lt;/sup&gt;</td>
<td>0.24</td>
<td>49.7</td>
<td>86</td>
<td>42</td>
<td>15.1</td>
</tr>
<tr>
<td>Sweden</td>
<td>12.5&lt;sup&gt;1,13&lt;/sup&gt;</td>
<td>0.35</td>
<td>46.3</td>
<td>29</td>
<td>5</td>
<td>26.5</td>
</tr>
<tr>
<td>Switzerland</td>
<td>36.7&lt;sup&gt;1,14&lt;/sup&gt;</td>
<td>1.48</td>
<td>47.3</td>
<td>58</td>
<td>70</td>
<td>34.6</td>
</tr>
<tr>
<td>Thailand</td>
<td>11.2&lt;sup&gt;1&lt;/sup&gt;</td>
<td>—</td>
<td>48.9</td>
<td>—</td>
<td>—</td>
<td>2.0</td>
</tr>
<tr>
<td>Turkey</td>
<td>46.8&lt;sup&gt;1&lt;/sup&gt;</td>
<td>1.72</td>
<td>51.4</td>
<td>85</td>
<td>45</td>
<td>2.7</td>
</tr>
<tr>
<td>UK</td>
<td>6.0&lt;sup&gt;6,15&lt;/sup&gt;</td>
<td>0.8</td>
<td>50.1</td>
<td>35</td>
<td>66</td>
<td>26.7</td>
</tr>
<tr>
<td>USA</td>
<td>12.3&lt;sup&gt;1&lt;/sup&gt;</td>
<td>0.38</td>
<td>48.1</td>
<td>46</td>
<td>62</td>
<td>36.6</td>
</tr>
<tr>
<td>Venezuela</td>
<td>48.8&lt;sup&gt;1&lt;/sup&gt;</td>
<td>0.05</td>
<td>76</td>
<td>73</td>
<td>4.0</td>
<td></td>
</tr>
<tr>
<td>Yugoslavia</td>
<td>66.8&lt;sup&gt;16&lt;/sup&gt;</td>
<td>—</td>
<td>51.1</td>
<td>—</td>
<td>—</td>
<td>1.2</td>
</tr>
</tbody>
</table>

Toxoplasma and culture  K. D. Lafferty 2751

were considered as one-tailed predictions. Assumptions of the general linear model were not violated by the distribution of the residuals.

3. RESULTS
As predicted by the relationship between individual personality and seropositivity, aggregate neuroticism, as measured by Lester (2000), increased significantly with T. gondii prevalence (figure 1; \( n = 25, R^2 = 0.38, p = 0.001 \), GDP \( p = 0.010 \); \( R^2 \) between aggregate neuroticism and prevalence after controlling for GDP=0.28). Aggregate neuroticism, as measured independently by the five-factor NEO-PI-R model, also increased significantly with prevalence (\( n = 31, R^2 = 0.30, p = 0.001 \)). Three countries were significant outliers (points outside the 95% confidence boundaries for individual predictions) for one measure of neuroticism: Hungary (Lester) and China (NEO-PI-R) had higher neuroticism than expected, while Turkey (NEO-PI-R) had a lower neuroticism than expected. Prevalence was positively, but weakly and not significantly, associated with the cultural dimensions of uncertainty avoidance (\( n = 32, R^2 = 0.07, p = 0.061 \)) and ‘masculine’ sex roles (\( n = 32, R^2 = 0.06, p = 0.099 \)). Two dramatic outliers were South Korea and Japan, which had lower prevalence than expected. When only Western nations were analysed (i.e. excluding China, South Korea, Japan, Turkey and Indonesia), uncertainty avoidance (\( n = 27, R^2 = 0.15, p = 0.017 \)) and masculine sex roles (\( n = 27, R^2 = 0.27, p = 0.003 \)) increased significantly with the prevalence of T. gondii (figures 2 and 3). There were two significant outliers for uncertainty avoidance among Western nations: Jamaica had less uncertainty avoidance than expected and Greece had more uncertainty avoidance than expected.

4. DISCUSSION
The associations between prevalence and cultural dimensions are consistent with the prediction that T. gondii can influence human culture. Just as individuals infected with T. gondii score themselves higher in the neurotic factor guilt-proneness, nations with high T. gondii prevalence had a higher aggregate neuroticism score. In addition, Western nations with high T. gondii prevalence were higher in the ‘neurotic’ cultural dimensions of masculine sex roles and uncertainty avoidance. These results were predicted by
a logical scaling-up from individuals to aggregate personalities to cultural dimensions.

These results are thought-provoking, but several caveats are important to consider when interpreting the data. Since this study is correlative, it is impossible to confirm causation (e.g. the analysis cannot reject the alternative interpretation that aggregate personality or cultural dimension alters risk of exposure), or to exclude the possibility of spurious results (i.e. non-causal correlations between aggregate personality and aspects of climate and culture that influence T. gondii transmission). In addition, the results only explain a fraction of the variation in two of the four cultural dimensions, suggesting that if T. gondii does influence human culture, it is only one among many factors. Studies on the effects of genetics and environment have found strong effects of both on individual personality (Eysenck 1990), and history is often thought to exert a strong influence on culture (Hofstede & McCrae 2004).

Furthermore, while using nations as a unit of replication is convenient and expedient, most nations are sufficiently ethnically diverse to be multimodal in personality and have within-country variation in prevalence. To the extent that these limitations of the data reduce statistical power, the effects of T. gondii on culture could actually be greater than suggested. It also suggests that studies of large diverse nations could be used as independent tests of this hypothesis.

What leads to variation in exposure? Differences in exposure risk cause wide variation in T. gondii prevalence among human populations. In particular, climate can alter exposure risk. Oocysts live longer in humid, low altitude regions, especially at mid-latitudes with infrequent freezing and thawing (Walton et al. 1966; Dubey 1974; Dubey & Beattie 1988). The resulting geographical variation in the risk of T. gondii in humans could indirectly lead to geographical variation in cultural dimensions, potentially explaining the observation of a greater differentiation of sex roles in warm countries (Hofstede 2001).

Climate is not the only factor that influences T. gondii. Human behaviour can also alter exposure. Dwelling among cats (Kean et al. 1969) or high human densities (Jones et al. 2001) increases exposure to oocysts. People working with soil and experiencing poor hygiene are more likely to ingest these oocysts (Jones et al. 2001). Finally, a cuisine including rare or undercooked meat increases an individual’s exposure to tissue cysts (Baril et al. 1999). To put this in perspective, 38% of meat products available for sale in the UK test PCR positive for T. gondii tissue cysts, some of which are probably viable (Aspinall et al. 2002).

How are populations with a high prevalence of T. gondii different? Individuals in populations that are ‘masculine’ in the sex-role cultural dimension reinforce traditional gender work roles, gender differentiation, and have a higher focus on ego, ambition, money, material possessions, self-achievement and work than on relationships, people, social support and quality of life (Hofstede 2001). Individuals in populations that rate high in the cultural dimension of uncertainty avoidance feel threatened by uncertain or unknown situations, leading to a rule-oriented society geared to reduce uncertainty (Hofstede 2001). These cultural effects were only apparent in Western populations, suggesting that if T. gondii does affect cultural dimension, then it does not drive differences seen among the major cultural groups (e.g. Western, African, Asian). Tests among Western nations were possible owing to adequate sample sizes. Further data would be needed to address this question in African and Asian nations, but this underscores the possibility that effects of T. gondii might not be uniform among cultures.

To what extent might T. gondii drive human culture? Many factors affect personality and culture, and we should not expect any one factor (such as T. gondii) to explain an overwhelming amount of the variance among populations. Furthermore, focusing on the link between infection and neuroticism leaves many aspects of personality and culture unexplored. Still, T. gondii affects other aspects of individual personality than neuroticism. However, because the associations between personality and T. gondii differ between men and women, it is more challenging to predict what aggregate personality or cultural differences might result at the population level from these effects. In addition to the associations with guilt-proneness assessed here, infected women are more intelligent, rule-conscious, dutiful, conscientious, conforming, moralistic, staid, rule-bound, warm, outgoing, attentive to others, kindly, easy-going and participating, while infected men are less intelligent and more reflective, rigid, loyal, stoic, slow-tempered, frugal, reactive emotionally, changeable, affected by feelings, emotionally less stable and easily upset. These associations suggest that the effect of T. gondii on culture could be broader than postulated here.

This work was conducted as part of the Analysing Pattern and Process in Human Cultural Diversity Working Group supported by the National Centre for Ecological Analysis and Synthesis, the University of California and the University of California, Santa Barbara. I am indebted to Jonah Haas for his dedicated efforts on this project as a graduate assistant. Jeff McCrae and Jaroslav Flegr provided useful comments on the manuscript and its relationship to their work. Statistician Julie Yee reviewed the analysis. Tom Suchanek, Jenny Shaw, Ryan Hechinger, Mike Behrens and Armand Kuris provided valuable comments on a draft of the manuscript. This manuscript has benefited from support received from the National Science Foundation through the NIH/NSF Ecology of Infectious Disease Program (DEB-0224565). This is a work of the U.S. Government and is not subject to copyright protection in the United States. Foreign copyrights may apply.

REFERENCES


