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Pathogens, Personality and Culture: Disease Prevalence Predicts Worldwide Variability in Sociosexuality, Extraversion, and Openness to Experience

Mark Schaller and Damian R. Murray University of British Columbia

Previous research has documented cross-cultural differences in personality traits, but the origins of those differences remain unknown. We investigate the possibility that these cultural differences can be traced, in part, to regional differences in the prevalence in infectious diseases. Three specific hypotheses are deduced, predicting negative relationships between disease prevalence and (a) unrestricted sociosexuality, (b) extraversion, and (c) openness to experience. These hypotheses were tested empirically with methods that employed epidemiological atlases in conjunction with personality data collected from individuals in dozens of countries worldwide. Results were consistent with all three hypotheses: In regions that have historically suffered from high levels of infectious diseases, people report lower mean levels of sociosexuality, extraversion, and openness. Alternative explanations are addressed, and possible underlying mechanisms are discussed.

Keywords: culture, disease prevalence, extraversion, openness to experience, sociosexuality

People's personalities differ, and some of that individual variability is geographically clumped. But why is that so? How are we to understand the origins of regional differences in personality? A complete response to that question will surely require attention to many different processes operating at different levels of analysis. Here, we focus on one previously unidentified contributor to those differences. We report empirical results indicating that specific kinds of cross-cultural differences in personality result, in part, from regional differences in the prevalence of infectious disease.

Cross-Cultural Differences in Personality

In recent years, several ambitious programs of research have collected data from dozens of countries worldwide as a means of documenting cross-cultural differences on various kinds of personality traits. These investigations assessed personality traits at the individual level of analysis, with standard trait assessment instruments; and on the basis of these data computed mean trait scores at the regional level of analysis.

For example, Schmitt (2005) and his collaborators in the International Sexuality Description Project

Mark Schaller and Damian R. Murray, Department of Psychology, University of British Columbia.

Correspondence may be addressed to Mark Schaller, Department of Psychology, UBC, 2136 West Mall, Vancouver Canada V6T1Z4. Email: schaller@psych.ubc.ca.

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assessed worldwide variability in chronic tendencies toward either a "restricted" or "unrestricted" sociosexual style. (Among other things, more highly unrestricted individuals seek more sexual variety, are more comfortable with casual sexual encounters, and have more sexual partners across their lifetime.) The results document considerable regional variability on this trait. In addition, several international teams of researchers have assessed worldwide variability along the "big five" personality traits of agreeableness, conscientiousness, extraversion, neuroticism, and openness to experience (McCrae, 2002; McCrae, Terracciano, & 78 Members of the Personality Profiles of Cultures Project, 2005; Schmitt, Allik, McCrae, Benet-Martinez, et al., 2007). The results document regional differences along all five fundamental dimensions of personality.

These results, and others like them, are invaluable assets to the scientific study of individual differences and culture. New investigations are addressing the consequences that these cultural differences may have (e.g., on health outcomes and social policy decisions; McCrae & Terracciano, 2008). To date, however, almost no research whatsoever has addressed why these worldwide differences in personality profiles exist in the first place.

Psychology, Ecology, and the Origins of Culture

Many factors may have contributed to cross-cultural differences in personality. Some of those causes may be idiosyncratic to the unique histories of specific populations. While important, analyses of idiosyncratic circumstances don't easily yield conclusions that are predictive on a more global scale. It is toward discovering a more complete (and more broadly predictive) explanation for the origins of cross-cultural differences that it is useful to consider how basic human tendencies, operating in conjunction with varying ecological conditions, can produce cross-cultural differences (Berry, 1979, Cohen, 2001; Gangestad, Haselton, & Buss, 2006; Kenrick, Li, & Butner, 2003; Vandello & Cohen, 1999).

Our conceptual analysis focuses on risks that historically have been posed by infectious diseases, and on specific kinds of psychological responses that may have reduced individuals' risk of infection. This analysis (presented in detail below) considers the costs and benefits of such responses, and the extent to which the cost/benefit ratio may be influenced by the prevalence of infectious diseases in the local ecology. This results in the prediction that specific kinds of cross-cultural differences in personality arose from responses to the different pressures that, in different places, are posed by infectious diseases.

It's worth noting in advance that this predicted causal relationship might be accomplished through a variety of conceptually distinct mechanisms, ranging from genetic selection to cultural transmission. Given the limitations of our empirical investigation, we must remain agnostic about the exact nature of these underlying mechanisms. (Later, in the Discussion section, we speculate about the possible role of different mechanisms.) Our investigation focuses more simply on the previously untested hypothesis that regional variability in the prevalence of infectious diseases has contributed to crosscultural differences in personality.

Vulnerability to Infectious Disease, and Its Psychological Consequences

Infectious diseases have posed a threat for as long as human history has been recorded, and presumably for far longer than that (Wolfe, Dunavan, & Diamond, 2007). Given this persistent threat, it's no surprise that people are equipped with defenses against that threat. One kind of defense exists in the form of highly sophisticated physiological mechanisms (the immune system) that detect and attack disease-causing agents within the body. Other defenses are more psychological in nature, and are reflected in stimulus-response mechanisms through which aversive cognitive and emotional responses

are aroused by the perception of disease-causing parasites and their hosts (Curtis, Aunger, & Rabie, 2004; Schaller & Duncan, 2007). Much recent research indicates that specific kinds of interpersonal prejudices result, in part, from the over-activation of psychological mechanisms that inhibit contact with people who appear to pose a high risk for the transmission of disease (Park, Faulkner, & Schaller, 2003; Park, Schaller, & Crandall, 2007; Schaller & Duncan, 2007). One such line of work has focused on xenophobia and ethnocentrism. Based in part on the assumption that foreign peoples are often unaware of – and thus likely to violate – local customs that serve as barriers against the transmission of disease (e.g., cultural norms pertaining to hygiene and food preparation), it has been hypothesized that disease-avoidant psychological responses contribute to xenophobia and ethnocentrism. This hypothesis has been supported across multiple studies (Faulkner, Schaller, Park, & Duncan, 2004; Navarrete & Fessler, 2006; Navarrete, Fessler, & Eng, 2007).

One of the key findings from this line of research is that these disease-avoidant psychological responses are variable across persons and contexts. They vary depending on the extent to which individuals are vulnerable (or merely perceive themselves to be vulnerable) to infectious diseases. Individual differences in perceived vulnerability to disease predict aversive responses to subjectively foreign (but not familiar) ethnic groups (Faulkner et al., 2004; Navarrete & Fessler, 2006). Xenophobic responses are also amplified by temporary contextual cues that make the threat of disease temporarily salient (Faulkner et al., 2004), and among people whose normal immunological defenses are temporarily compromised (Navarrete et al., 2007). These findings are explained by a conceptual framework that considers the potential costs and benefits associated with close proximity to foreign peoples. Potential costs reside in the greater risk of disease transmission. But there can also be benefits that attend interactions with foreign peoples too (e.g., the opportunity to establish new relationships and alliances; the exchange of useful technologies and other resources). These costs and benefits may vary across persons or circumstances, and the cost/benefit ratio has an influence on psychological responses to foreign peoples. When the potential costs (disease transmission) are outweighed by the potential benefits (e.g. the rewards associated with novel social interactions) individuals' xenophobic responses are muted. But when the potential costs are relatively greater, xenophobia increases accordingly.

This same framework can be expanded to focus not merely on responses to foreign peoples, but on responses to the broader range of unfamiliar things that individuals potentially encounter in their social and physical environment. To the extent that individuals adopt an incautious attitude toward novel things (e.g., a willingness to become physically intimate with unfamiliar people, a willingness to try unfamiliar foods, a willingness to experiment with unfamiliar practices), they potentially put themselves at greater risk for disease transmission. At the same time, however, an incautious attitude toward unfamiliar people and unfamiliar things may produce benefits as well (new relationships, new discoveries, etc.). Again, the cost/benefit ratio may vary across persons or circumstances. (See Nettle, 2006, 2007, for a broader discussion of context-specific costs and benefits of personality traits). Costs of incautious behavior are likely to be relatively high when the real risk of disease transmission is relatively high. To the extent that psychological tendencies are calibrated, even roughly, to costs and benefits, it follows that when infectious diseases are more prevalent, people may adopt a more cautious and conservative style when interacting with their social and physical environment.

This line of reasoning has implications for cross-cultural differences. There is considerable geographical variability in the prevalence of infectious diseases (Guernier, Hochberg, & Guégan, 2004). Previous research has shown that variability in disease prevalence predicts various kinds of cross-cultural differences – including differences in food preparation, marriage structures, parenting practices, and the attributes that matter most in mate selection (Gangestad et al., 2006; Low, 1990; Quinlan, 2007; Sherman & Billing, 1999). The preceding conceptual analysis suggests that disease prevalence may also help account for cross-cultural differences in personality. Within regions in

which disease prevalence is relatively high, the costs of an incautious disposition may outweigh its benefits. This may result in a general tendency for individuals to be interpersonally cautious and conservative in their responses to unfamiliar things. In contrast, within regions in which disease prevalence is relatively low, the costs of an incautious disposition are likely to be lower, and possibly outweighed by the potential benefits. As a consequence, individuals living within these regions may be more outgoing and open to unfamiliar things.

Predictions for Worldwide Variability Along Specific Personality Traits

How exactly might these cross-cultural differences manifest? What particular kinds of personality traits might reflect these differences? Our analysis suggests at least three specific hypotheses, each informed by the consideration of specific kinds of behaviors that are associated with the risk of disease transmission.

Many diseases may be transmitted from person to person as the result of intimate physical contact of the sort associated with sexual behavior. Sexual contact not only facilitates transmission of diseases explicitly defined by sexual transmission (e.g. syphilis), it also may facilitate transmission of many other pathogens as well. It follows that, in regions in which disease prevalence is high, people may be dispositionally cautious in the domain of sexual behavior. Thus, at a regional level of analysis, disease prevalence is expected to be associated with a more restricted (as opposed to unrestricted) sociosexual style.

Social contact need not be intensely intimate to increase the risk of disease transmission. As the epidemiological practice of quarantine indicates, many kinds of ordinary social interactions have the potential to expose individuals to disease-causing pathogens. The risk of disease transmission increases with the number of different people with whom one has social contact. Thus, a dispositional tendency toward extraversion may be associated with an enhanced risk of disease transmission (Hamrick, Cohen, & Rodriguez, 2002; Nettle, 2005), and this may be the case particularly under ecological circumstances in which diseases are highly prevalent. At a regional level of analysis, then, higher levels of disease prevalence are expected to be associated with lower levels of extraversion.

While many diseases are transmitted through direct interpersonal contact, many are transmitted indirectly, through a variety of means (e.g., contaminated water supplies, inexpertly-prepared foods). Many institutionalized norms and collective practices serve as barriers against these forms of transmission (e.g., normative guidelines that serve to keep human waste away from sources of drinking water; cultural standards for the appropriate spicing of meats). To the extent that individuals violate these conventions (e.g., fail to follow local prescriptions for food preparation) or expose themselves to novel aspects of the environment (e.g., drink from an unfamiliar water source), they may expose themselves and others to infectious diseases. This is especially the case in regions in which the prevalence of disease is high. This has an implication for personality traits pertaining to curiosity, experimentation and willingness to deviate from the status quo – the sorts of traits signaled by openness to experience. At a regional level of analysis, higher levels of disease prevalence are expected to be associated with lower levels of openness.

The three preceding hypotheses emerge clearly from our conceptual framework. In addition, there may be reasons to suspect that disease prevalence might also predict cross-cultural variability along other fundamental personality traits. Both conscientiousness and neuroticism are defined, at least in part, by behavioral prudence versus recklessness. (One measure of the "big five" personality traits explicitly includes "deliberation" as a facet of conscientiousness and "impulsiveness" as a facet of neuroticism; Costa & McCrae, 1992) Both conscientiousness and neuroticism are, in fact, correlated with health outcomes (e.g., Bogg & Roberts, 2004; Booth-Kewley & Vickers, 1994; Goodwin & Friedman, 2006). It's worth noting, however, that many of these correlations pertain to health-related

variables that are largely irrelevant to disease transmission (e.g., alcohol use; tobacco use; accident-proneness). There's also another reason to doubt any straightforward functional relationship between disease prevalence and regional differences in conscientiousness or neuroticism: Both conscientiousness and neuroticism are partially defined by facets (e.g., "competence" and "depression," respectively) that are causally influenced by actual illness – which (contrary to the implication of our analysis) implies that greater disease prevalence may be associated with lower (rather than higher) conscientiousness and higher (rather than lower) neuroticism. Nevertheless, in addition to testing the primary hypotheses (pertaining to sociosexuality, extraversion, and openness), we also explored the possibility that disease prevalence might also predict cross-cultural variability along the remaining "big five" personality traits (agreeableness, conscientiousness, neuroticism).

Overview of Present Investigation

The results reported below provide the first empirical tests of the conceptual hypotheses identified above. For all analyses, geopolitical regions (nations or territories) served as the unit of analysis. We developed an index indicating the historical prevalence of infectious diseases in dozens of geopolitical regions worldwide. Regional scores on personality traits were obtained from other researchers' published articles on cultural variability in personality (McCrae, 2002; McCrae et al., 2005; Schmitt, 2005; Schmitt et al., 2007). Statistical analyses tested the extent to which disease prevalence correlated, as hypothesized, with regional differences in sociosexuality, extraversion, and openness to experience. (In addition, we also examined relations between disease prevalence and regional variability on agreeableness, conscientiousness, and neuroticism).

The empirical methods are necessarily correlational, but the conceptual hypotheses are clearly causal. Given the causal chronology implied by these hypotheses, we ensured that our index of disease prevalence was based on measurements assessed many years prior to the measurement of personality traits. To rule out alternative causal explanations for the results, we conducted additional analyses that statistically controlled for potential confounding variables.

Methods

Assessment of Regional Differences in the Prevalence of Infectious Disease

As part of a larger project, we developed a numerical index that estimates the relative prevalence of infectious diseases in each of the 71 geopolitical regions from which relevant personality trait data were available. In most cases, these regions were nations; in a few cases (Hong Kong, Puerto Rico) they were territories or culturally distinct regions within a nation. (The sample included nations/territories from each of the six major world regions identified by Murdock [1949]. The number of specific geopolitical regions within each of these major world regions were as follows: Western Eurasia = 37, Eastern Eurasia = 9, Insular Pacific = 6, Africa = 10, North America = 4, South America = 5.) In order to estimate the historical prevalence of infectious diseases in each region, we employed procedures modeled after those used in previous cross-cultural inquiries (e.g., Gangstad & Buss, 1993; Low, 1990). For each region, the prevalence of nine different kinds of disease-causing agents was estimated on the basis of old medical atlases and other epidemiological sources. These diseases were: leishmania, schistosoma, trypanosoma, malaria, filaria, leprosy, dengue, typhus, and tuberculosis.

For six of these diseases (leishmania, schistosoma, trypanosoma, malaria, filariae, and leprosy), prevalence was estimated on the basis of epidemiological maps included in Rodenwaldt and Jusatz' (1952-1961) *World-atlas of epidemic diseases*. A 4-point coding scheme was employed: 0 =

completely absent or never reported; 1 = rarely reported; 2 = sporadically or moderately present; 3 = present at severe levels, or epidemic levels at least once. For dengue and typhus, prevalence was estimated on the basis of information included in Simmons et al.'s (1944) Global Epidemiology. This source provided epidemiological maps and verbal descriptions that allowed for the following 4-point coding scheme: 0 = absent or never reported; 1 = rare or present only in isolated areas; 2 = present, but not at severe levels; 3 = prevalent at high levels." Finally, prevalence of tuberculosis was based on a map in the National Geographic Society's *Atlas of the World* (2005), which provided, for each region, the rate of incidence per 100,000 people. Prevalence of tuberculosis was coded according to a 3-point scheme: 1 = 3-49 or no data; 2 = 50-99; 3 = >100. (In a few rare cases, information about a specific disease in a specific region was not evident in the sources indicated above. In these cases, we sought additional historical information from the website of the World Health Organization. For the most part, this additional information indicated that the disease was either very rare or entirely absent from the region omitted in the epidemiological atlases).

The nine numerical prevalence estimates were summed to create an overall index indicating the historical prevalence of disease-causing pathogens in each region (Cronbach's alpha = .84). Values on this overall index ranged from 1 to 24 (M = 11.92, SD = 6.46).

This index is based on the prevalence of just a small sample of the many disease-causing pathogens that exist on the planet, and coding procedures were necessarily crude. We must therefore be attentive to the utility of this index as an indicator of the underlying construct. We conducted three preliminary analyses to assess the construct validity and reliability of our disease prevalence index. One analysis was informed by research showing that the diversity and prevalence of infectious diseases is fundamentally a product of climate, and is much greater in the tropics than in regions further from the Earth's equator (Epstein, 1999; Guernier, Hochberg, & Guégan, 2004). It follows that our disease prevalence index should be inversely correlated (and strongly) with absolute latitude. It is, r = -.80. (The absolute latitude for each region was computed based on the latitude of that region's most populous city.) A second analysis examined the correlation between our disease prevalence index and a measure reported by Gangestad and Buss (1993), who used a similar procedure to estimate the summed prevalence of 7 disease-causing pathogens in each of 29 countries. The correlation between their index and ours was high, r = .89. A third analysis examined the correlation between our disease prevalence index (which was based primarily on historical evidence) and an analogous index computed from a sampling of contemporary epidemiological data (the Global Infectious Diseases and Epidemiology Online Network; for details see Fincher, Thornhill, Murray, & Schaller, in press). These two indices were also substantially correlated, $r = .77.^{1}$

These results suggest that, although crude, our disease prevalence index does provide a useful measure of the relative prevalence of disease-causing pathogens in different countries. And so it provides a tool for testing the hypothesized inverse relations between the prevalence of disease and cultural tendencies toward incautious interpersonal behavior.

Assessment of Regional Differences in Unrestricted Sexual Style

Individual differences in the tendency toward an unrestricted approach to sexual relationships can be measured with the Sociosexual Orientation Inventory (SOI; Simpson & Gangestad, 1991). Low

¹

¹ Given this high correlation, it follows that the index assessing contemporary pathogen prevalence should also predict cross-cultural differences in sociosexuality, extraversion, and openness to experience. It does; the patterns of correlation are identical to those we report in the Results section below. It's worth noting, however, that these predictive effects were consistently weaker than those obtained with our index computed from historical (rather than contemporary) disease prevalence data. This has useful inferential implications, to which we return in the Discussion.

SOI scores indicate a restricted approach to sexuality, whereas higher SOI scores indicate a more unrestricted approach (including greater chronic interest in new sexual partners, and greater comfort with casual sexual encounters).

Schmitt (2005) conducted a cross-cultural investigation of restricted and unrestricted sociosexuality, assessing the SOI scores of 14,059 individuals living in 48 different geopolitical regions. Schmitt (2005) reports mean SOI scores (standardized) in each of the 48 regions. These mean SOI scores are reported separately for men and women. We employed these means as our region-level measure of unrestricted sexual style.

Assessment of Regional Differences in the "Big Five" Personality Traits

Three different cross-cultural investigations provide region-level measures of the "big five" personality traits (extraversion, openness, agreeableness, conscientiousness, and neuroticism).

McCrae (2002) summarized results from several dozen independent investigations that used the NEO-PI-R questionnaire (Costa & McCrae, 1992) to assess the self-reported personality traits in 33 different geopolitical regions. Separately, McCrae et al. (2005) used the NEO-PI-R questionnaire and an observer-report methodology to assess the personality traits of 11,985 individuals living in 50 different geopolitical regions. In a third investigation, Schmitt et al. (2007) used a different measure of the big five personality traits (the BFI questionnaire; John & Srivastava, 1999) to assess self-reported personality traits from 17,837 individuals in 56 different regions. Each of these 3 sources provides standardized region-level mean scores on each of the big five personality traits.²

Because any single indicator of region-level trait scores may be less than ideally reliable, we also created two sets of composite scores as well. A two-sample composite score for each of the big five traits was computed as a mean of the scores reported in the two largest cross-cultural samples (McCrae et al., 2005; Schmitt, 2007), for which there were 38 regions in common. We also computed a 3-sample composite score for each trait: The mean of the scores reported across all three of the independent samples (for which there were 23 regions in common).

For our analyses, we examined the extent to which disease prevalence predicted the McCrae (2002) trait scores, the McCrae et al. (2005) trait scores, the Schmitt et al. (2007) trait scores, the 2-sample composite trait scores, and the 3-sample composite trait scores.

Exploratory Investigation of NEO-PI-R Facet Scores

One measure of the big five personality traits (the NEO-PI-R) also produces values on 30 more specific trait-like "facets" (each of the big five traits is comprised by 6 facets). McCrae (2002) reports region-level facet scores based on the self-report data obtained from 33 geopolitical regions. McCrae and Terraciano (2008) report region-level facet scores based on observer-report data obtained from 50 geopolitical regions. Following up on our analyses on the big five traits, we also explored the extent to which disease prevalence predicted cross-cultural variability along each of the 30 facet scores derived from t these two investigations.

² The big five trait scores were relatively independent at the regional level of analysis. Correlations between extraversion and openness were .16, .00, and .27 in the McCrae (2002), McCrae et al. (2005), and Schmitt et al. (2007) samples, respectively. Variability on these traits did overlap somewhat with sociosexuality. Both extraversion and openness tended to correlate positively with region-level SOI scores. (For extraversion: across the three independent cross-cultural samples of big five traits, *r*'s with male SOI ranged from .19 to .62, and *r*'s with female SOI ranged from .32 to .56. For openness: across the three samples, *r*'s with male SOI ranged from .00 to .30, and *r*'s with female SOI ranged from .24 to .62).

Table 1 Correlation coefficients indicating the extent to which regional variation in disease prevalence predicts cross-cultural variation on each of the Big 5 personality traits.

Samples From which Region-Level Big 5 Trait Scores Were Estimated

.01 (.961)

	1	C			
Trait	McCrae 2002 (N = 33)	McCrae et al. 2005 (N = 50)	Schmitt et al. 2007 (N = 56)	2-Sample Composite (N = 38)	3-Sample Composite (N = 23)
Extraversion	67 (.000)	50 (.001)	26 (.052)	51 (.001)	59 (.003)
Openness	45 (.008)	34 (.016)	24 (.079)	44 (.006)	59 (.003)
Conscientiousness	.44 (.011)	08 (.594)	.23 (.085)	.02 (.891)	.14 (.516)
Agreeableness	.20 (.257)	44 (.001)	.27 (.039)	18 (.270)	.14 (.534)

Note: Within each sample, tabled coefficients indicate zero-order correlations with disease prevalence. (2-tailed p-values are in parentheses, following each correlation coefficient.)

.15 (.309)

Results

Primary Analyses

Neuroticism

.24 (.173)

Does Disease Prevalence Predict Regional Differences in Sociosexual Style? Our conceptual analysis suggests that an unrestricted sociosexual style is more costly in geographical regions that havehistorically had a higher prevalence of infectious diseases; and as a consequence, sociosexual tendencies in those regions are likely to be relatively restricted. This analysis leads to the prediction of a negative correlation between disease prevalence and mean SOI scores. Results generally support the hypothesis. Disease prevalence was strongly negatively correlated with female SOI scores, r = -.62 (p < .001). The correlation with male SOI scores was also negative, but weaker, r = -.27 (p = .066).

Does Disease Prevalence Predict Regional Differences in Extraversion? Our conceptual analysis suggests that extraversion is a potentially more risky trait in regions characterized by higher prevalence of infectious diseases. This leads to the prediction of a negative correlation between disease prevalence and mean extraversion scores. Table 1 reports correlations (and associated *p*-values) for each of the five measures of extraversion (the three independent measures, plus the two composite measures). Across the five measures, the correlations were all negative and, with one exception, substantially so (*r*'s ranged from -.26 to -.67).

Does Disease Prevalence Predict Regional Differences in Openness to Experience? Our conceptual analysis also predicts a negative correlation between disease prevalence and openness. Results support this hypothesis as well (r's ranged from -.24 to -.59; see Table 1).

Were There Any Effects on the Other Big Five Traits? In contrast to the predictive effects of disease prevalence on extraversion and openness, there were no consistent relations between disease prevalence and the other personality traits (see Table 1). Disease prevalence correlated positively with two of the independent measures of conscientiousness (one of these positive r's was statistically significant), but negatively with the third measure; correlations with the two composite measures of conscientiousness were both close to zero. Disease prevalence also produced an inconsistent pattern of correlations across the five measures of agreeableness; the two largest of these correlations (both of which were statistically significant) were in opposite directions. As for neuroticism: All five measures

were positively correlated with disease prevalence, but none of these correlations was sufficiently substantial to even approach conventional levels of statistical significance.

Ancillary Analyses on the NEO-PI-R Facet Scores

We also computed correlations between disease prevalence and region-level values on each of the six facets associated with each of the big five personality traits. These correlations were computed for the region-level facet scores reported by McCrae (2002), as well as for those reported by McCrae and Tarraciano (2008). Given the ancillary (and, to some extent, exploratory) nature of these analyses, interpretational caution is advised. Therefore, we identify only those facet scores that showed at least a moderate-sized correlation with disease prevalence (specifically: r = +/-.20 or greater), and did so consistently across both samples.

Four of the six extraversion facets met this standard: warmth, gregariousness, activity, and positive emotions all showed moderate to large correlations with disease prevalence, and did so across both samples (r's ranged from -.21 to -.70; median r = -.46; median p = .007).

Similarly, four of the six openness facets met the standard: *fantasy*, *feelings*, *ideas*, and *values* all showed moderate to large correlations with disease prevalence, across both samples (r's ranged from - .20 to -.73; median r = -.58; median p = .001).

Only two of the conscientiousness facets met the standard, and the correlations were in opposite directions: Disease prevalence correlated negatively with *competence* (r's were -.30 and -.49, both p's < .09), and correlated positively with *deliberation* (r's were .36 and .63, both p's < .01). Similarly, only two of the agreeableness facets met the standard: Disease prevalence correlated negatively with *altruism* and *modesty* (r's ranged from -.29 to -.59; median r = -.37; median p = .029). Finally, four of the neuroticism facets met the standard, but (as with the conscientiousness facets) the direction of these correlations was inconsistent: Disease prevalence correlated positively with *depression*, *vulnerability*, and *self-consciousness* (r's ranged from .34 to .45; median r = .40; median p = .010), and correlated negatively with *impulsiveness* (r's were -.36 and -.59, both p's < .01).

Additional Analyses Addressing Possible Alternative Causal Explanations

The preceding analyses offer support for the three primary hypotheses linking regional variation in disease prevalence to cross-cultural variation in sociosexuality, extraversion, and openness to experience. (The ancillary analyses on the NEO-PI-R facet scores suggest that there may also be relations between disease prevalence and other, more narrowly defined traits as well.) We now return to these primary hypotheses, to address possible alternative explanations for the observed correlations between disease prevalence and region-level values on SOI, extraversion, and openness. We conducted a series of multiple regression analyses to test whether the hypothesized relations hold even when statistically controlling for other variables.

For these analyses (as in the primary analyses reported above), statistical power was constrained by the number of regions on which the outcome variable was measured in prior investigations. Given the well-documented inferential costs associated with low statistical power (e.g., Schmidt, 1996), and the practical impossibility of enhancing power by increasing the sample size, we employed another principled strategy to boost statistical power and reduce the likelihood of statistical inference errors: We used one-tailed tests when testing the null hypothesis associated with the predicted effects of disease prevalence. (Two-tailed tests were used in all analyses reported above). This strategy is justified by the fact that (a) there are clear conceptual hypotheses, (b) the conceptual hypotheses are clearly directional (i.e., specifying *negative* correlations with SOI, extraversion, and openness), and (c) these hypothesized relations were borne out by the zero-order correlations reported above. (Therefore,

if any of the analyses below yielded reversals of the predicted negative relations, those effects – no matter how large in magnitude – would be considered uninterpretable.) Thus, in the following analyses, one-tailed p-values are reported in conjunction with observed negative relations between disease prevalence and the three primary outcome variables (SOI, extraversion, openness).

Can The Effects Be Explained by Differences in Life Expectancy? Schmitt (2005) reported negative correlations between SOI scores and the mean life expectancy in these different regions. He offered an explanation that focused on ecological hardships and implications for the adoption of short-term versus long-term mating strategies – an explanation that has nothing to do with the risk of disease transmission, per se. Of course, life expectancy is powerfully influenced by the prevalence of infectious diseases. Does disease prevalence predict SOI independent of whatever other hardships might factor into life expectancy? To answer this question, we conducted a pair of regression analyses (one on male SOI scores and the other on female SOI scores) in which both disease prevalence and life expectancy were entered as predictors. (Life expectancy values for both men and women were those reported by the World Health Organization, for the year 2004.) On male SOI scores, neither predictor variable exerted a significant unique effect (betas = .13 and -.16 for life expectancy and disease prevalence). On female SOI scores, the unique effect of life expectancy was negligible (beta =-.07), but the unique effect of disease prevalence remained strong (beta = -.65, p < .001). Thus, whatever predictive effect life expectancy may have on SOI, this effect is not independent of disease prevalence. In contrast – at least for female SOI scores – there is a uniquely powerful predictive effect specific to the prevalence of disease-causing pathogens.

We also examined whether life expectancy might account for the apparent effects of disease prevalence on extraversion and openness. In 10 regression analyses, we entered life expectancy and disease prevalence simultaneously as predictors of each measure of extraversion and each measure of openness. For only one of these 10 analyses was there any evidence of a unique effect of life expectancy. In contrast, even when controlling for life expectancy, disease prevalence continued to exert a unique effect on both extraversion and openness. (E.g., on the 2-sample composite estimate of extraversion, beta = -.42, p = .010; and on the 2-sample composite estimate of openness, beta = -.34, p = .033).

Can The Effects Be Explained by Differences in Economic Development? Economic development is commonly assumed to have an impact on cultural values (e.g., Hofstede, 2001), and arguments might be developed also about causal connections between economic development and personality traits. It is impossible to fully disentangle any such speculations from the consequences of infectious diseases. (The prevalence of infectious diseases is a powerful inhibitor of economic development; Sachs & Malaney, 2002). Nevertheless, the predictive effects of disease prevalence on personality would be more compelling if they remained even after controlling for per capita gross domestic product (GDP; specific values for each country were those reported by the World Bank, for the year 2004). To control for GDP, both GDP and disease prevalence were entered as predictor variables in a series of regression analyses with measures of SOI, extraversion, and openness as criterion variables.

For SOI, the results were unequivocal. When controlling for disease prevalence, GDP was a unique predictor of neither female nor male SOI. But, even when controlling for GDP, disease prevalence uniquely predicted both male SOI scores (beta = -.38, p = .014) and female SOI scores (beta = -.56, p < .001).

For extraversion, there was no consistent unique effect of GDP across the 5 measures. In contrast, even when controlling for GDP, the significant negative effect of disease prevalence persisted across all measures of extraversion. (E.g., on the 2-sample composite measure, beta = -.60, p < .001).

Similar results emerged for openness. GDP showed no consistent unique effect across the five measures of openness. In contrast, even when controlling for GDP, disease prevalence continued to uniquely predict 4 of the five measure of openness. (The one exception occurred on the McCrae et al.,

2005 measure; for the other 4 measures, p < .05. E.g., on the 2-sample composite measure of openness, beta = -.43, p = .013).

Can The Effects Be Explained by Differences in Individualism / Collectivism? Perhaps no dimension of cross-cultural difference has attracted more attention than individualism / collectivism. Individualistic values are less apparent among peoples living in tropical regions (Hofstede, 2001; Kashima & Kashima, 2003). And because disease prevalence is much higher in the tropics, individualism and disease prevalence are negatively correlated. Moreover, worldwide differences in individualism are correlated with worldwide differences along some personality traits, including extraversion (Hofstede & McCrae, 2004). Does individualism / collectivism provide a viable alternative explanation for the apparent effects of disease prevalence on personality? For two reasons, the answer appears to be no. One reason is conceptual. Although values and personality traits may be correlated, it is by no means certain that cultural differences in individualism/collectivism are a cause, rather than a consequence of differences in personality (Hofstede & McCrae, 2004). Moreover, there is evidence that cross-cultural differences in individualism / collectivism are themselves a consequence of regional differences in disease prevalence (Fincher, Thornhill, Murray, & Schaller, in press).

In addition to that conceptual argument, there is a separate reason – entirely empirical – to discount individual / collectivism as an alternative explanation for the effects of disease prevalence: With only one exception, these effects persist even when statistically controlling for individualism / collectivism. The one exception occurred on male SOI scores: When disease prevalence and individualism scores (obtained from Hofstede's website: http://www.geert-hofstede.com) were simultaneously entered as predictor variables in a regression equation, the unique effect of disease prevalence on male SOI was negligible. But for every other outcome variable, the effects of disease prevalence held strong. When controlling for individualism, disease prevalence continued to predict female SOI scores (beta = -.41, p = .009). When controlling for individualism, disease prevalence continued to predict both extraversion and openness. (E.g., on the 2-sample composite measure of extraversion, beta = -.37, p = .044; and on the 2-sample composite measure of openness, beta = -.43, p = .028).

It's also worth noting that individualism had no unique predictive effects on any measure of extraversion or openness. Thus, the previously documented relationship between individualism and extraversion may be spurious, resulting from shared variability with disease prevalence.

Can The Effects Be Explained by Other Variables that Correlate with Climate? Many scholars have speculated about processes through which different cultural values might arise from the differential roles of agriculture, foraging, and herding in the local economies (Berry, 1979, Cohen, 2001; Triandis, 1994; Vandello & Cohen, 1999). Many of these variables are (like disease prevalence) products of local climactic conditions and, because of this, absolute latitude and mean annual temperature have been used as an approximate indicators of those additional ecological variables (e.g., Cohen, 1998; Kashima & Kashima, 2003). Our results might be more compelling if the predictive effect of disease prevalence remained even after controlling for absolute latitude and mean annual temperature. (For each region, we computed the absolute value of the latitude of the region's most populous city. Mean temperature for each region's most populous city was computed from monthly mean temperature data reported by the World Meteorological Organization.) The results are reassuring:

On none of the outcome variables did absolute latitude exert a reliable predictive effect when controlling for disease prevalence. In contrast, the effects of disease prevalence persisted, with one exception, even when controlling for absolute latitude. (The only exception was in the prediction of male SOI scores, beta = -.14, p = .269). Even when controlling for absolute latitude, disease prevalence continued to predict female SOI scores (beta = -.54, p = .003). Even when controlling for absolute latitude, disease prevalence continued to consistently predict both extraversion and openness. (E.g., on the 2-sample composite measure of extraversion, beta = -.44, p = .028; and on the 2-sample composite measure of openness, beta = -.34, p = .077).

The same pattern of results emerged when controlling for mean annual temperature. Temperature exerted no unique predictive effects on any of the outcome variables. In contrast, when controlling for temperature, disease prevalence still predicted female SOI scores (beta = -.50, p = .003), but not male SOI scores (beta = -.07, p = .364). In addition, even when controlling for temperature, disease prevalence continued to predict both extraversion and openness. (E.g., on the 2-sample composite measure of extraversion, beta = -.58, p = .008, and on the 2-sample composite measure of openness, beta = -.39, p = .057).

Discussion

These results provide support for all three hypotheses. At a regional level of analysis, a historically higher level of disease prevalence predicted a more restricted sociosexual style, particularly among women. Greater disease prevalence also predicted lower scores on extraversion, and on openness to experience. These results (along with ancillary results indicating additional correlations with deliberation and impulsiveness) are consistent with the more general speculation that personality styles may vary as a functional response to the presence of pathogens in the local ecology: Under circumstances in which pathogen transmission has been more likely, people exhibit a more careful and cautious dispositional style

The empirical methods were, of course, correlational. Thus, while the results are consistent with the causal hypotheses articulated at the outset, we cannot disregard the possibility that these correlations may have emerged as a consequence of some alternative causal process. Plausible alternative explanations are, however, not readily apparent. A reverse causal explanation is inconsistent with the fact that personality differences were predicted more strongly by a measure of historical pathogen prevalence than by an alternative measure computed from contemporary epidemiological data (see Footnote 1). Moreover, the observed correlations are exactly opposite of what would be expected if disease prevalence was a consequence, rather than a cause, of incautious personality traits. In addition, we conducted ancillary analyses that statistically controlled for a variety of potential confounding variables. With only one exception (regional variability in male sociosexual style), the hypothesized effects persisted even when controlling for these additional variables. It is noteworthy that the predictive effects of disease prevalence trumped those of other variables despite the multicollinearity problems associated with these regression analyses. It is also notable that these effects emerged despite the fact that disease prevalence was almost certainly measured with less precision than several of these additional variables (e.g., GDP, latitude).

It's also worth noting that these predicted effects occurred despite the fact that there must surely be some "noise" in the personality trait scores employed in our analyses. Substantial methodological difficulties attend any attempt to measure personality traits across cultures (e.g., translation issues, reference group effects, etc.), most of which are likely to introduce nonsystematic measurement error into any computation of region-level mean trait values. In fact, some recent research suggests caution when interpreting cross-cultural measures of conscientiousness in particular (Heine, Buchtel, & Norenzayan, in press). The lack of a consistent correlation between disease prevalence and conscientiousness may reflect this measurement issue. More generally, the correlations reported above may actually *under*estimate the real relation between disease prevalence and worldwide variability in personality.

Thus, as with any investigation of this sort, we must be aware of the inferential limitations associated with the empirical methods employed. Still, there are several interesting implications of these results that merit further elaboration and discussion.

Why, for instance, is disease prevalence such a strong predictor of worldwide variation in female sociosexual style, but a relatively weak predictor of male sociosexual style? One sensible explanation

(which is consistent with the overall conceptual framework within which our hypotheses were deduced) considers sex differences in the trade-offs between the costs and benefits of an unrestricted sociosexual style (Schmitt, 2005). Because of differential reproductive investment, the fitness benefits of unrestricted sociosexuality are greater among men than among women. Among men, these benefits may exceed the costs of unrestricted sociosexuality even at relatively high levels of disease prevalence. Among women, however, any fitness benefits associated with unrestricted sociosexuality may be more readily overwhelmed by costs as disease prevalence increases. The upshot is a weak (or perhaps even nonexistent) effect of disease prevalence on male sociosexual style, and a substantial effect on female sociosexual style.

A second issue of importance pertains to the extent to which the effects documented here reflect processes operating at the individual or cultural level of analysis. Personality is typically defined at the individual level of analysis. When there exist dispositional differences between individuals from the same cultural population, it's clear that those differences reflect something specific to the individuals (and not, for example, differences in cultural norms). But when there exist mean differences between sets of individuals sampled from different cultural populations, the interpretation is more complicated. Those mean differences may indeed reflect differences in processes operating strictly at the individual level of analyses (i.e., the sorts of processes typically studied by personality psychologists). Those mean differences might also reflect value differences that operate at a more societal level of analysis – differences, for example, in the extent to which local cultural norms prescribe or proscribe specific kinds of behavioral tendencies. While our results are consistent with an interpretation phrased in the language of individual-level personality processes ("in places with historically high levels of infectious diseases, individuals have less extraverted personalities"), they are also consistent with an interpretation phrased in a way that emphasizes societal norms and values instead ("in places with historically high levels of infectious diseases, extraversion is less culturally valued").

Within the context of the present investigation, this distinction is of relatively little concern. (Regardless of whether our results are phrased in the language of individual-level personality processes, or in the language of cultural values, these results represent the first empirical support for a novel perspective on the origins of cross-cultural differences in behavioral dispositions.) The distinction begins to matter more, however, when we consider the underlying mechanisms through which disease prevalence might exert a causal influence on sociosexuality, extraversion, and openness to experience. Several different kinds of underlying mechanisms might be postulated.

One possible mechanism is through natural selection, in which different genes are selectively favored under different ecological circumstances. It is well established that extraversion and openness are partially heritable (e.g., Jang, Livesley, & Vemon, 1996); presumably, sociosexuality is as well. Indeed, recent research has begun to identify specific genetic markers associated with some of these personality traits (e.g., Savitz & Ramesar, 2004; Stein, Schork, & Gelernter, 2004). It's also clear that infectious diseases can pose a powerful selective force on gene frequencies within different human populations (Williamson et al., 2007). It is possible, therefore, that in regions characterized by a persistently high level of disease prevalence, there has been a natural selection process favoring alleles that are probabilistically associated with lower levels of extraversion, openness, and/or sociosexuality.

Our results might also be explained by a rather different kind of genetic mechanism. Genes associated with specific personality traits may be differentially expressed depending upon the prevalence of infectious diseases within the local ecology. Considerable evidence within the literatures on genetics, developmental biology, and behavioral ecology reveal that many phenotypic differences between individual organisms result not from the presence or absence of specific genes within the genome, but rather from the differential expression of common genes (for accessible overviews of some of this work, see Carroll, 2005 and Ridley, 2003). A gene's expression often depends upon input from the immediate environment (e.g., Godwin, Luckenbach, & Borski, 2003). Given that personality

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traits may confer either costs or benefits, and that these costs and benefits vary under different circumstances (Nettle, 2007), it's plausible that some of the genetic substrates for these traits are sensitive to informational inputs from the immediate environment – including input signaling the presence of infectious diseases. Such input might take a variety of forms, including neurochemical changes (such as those associated with a disgust response) that occur when potential sources of disease transmission are perceived, or perhaps even direct input from the immune system itself (e.g., the activation of an immune response may suppress the expression of specific genes associated with extraversion, openness, and/or unrestricted sociosexuality). Thus, regional differences in personality traits may constitute a form of "evoked culture" – wherein cultural differences reflect the phenotypic plasticity of the human genome, and emerge through mechanisms in which universal human capacities are differentially evoked under different ecological circumstances (Gangestad et al., 2006; Schaller, 2006; Tooby & Cosmides, 1992).

It's also possible, of course, that disease prevalence may lead to regional differences in personality through mechanisms that have nothing directly to do with genes at all. Just as genomes evolve in response to ecological pressures, so too do the norms and values and behavioral prescriptions that help to define human cultures. People are adept at detecting dangers in their immediate environments – including the potential dangers posed by infectious diseases (Schaller & Duncan, 2007). They are also inclined to communicate to others about these dangers (Conway & Schaller, 2007; Heath, Bell, & Sternberg, 2001). Through acts of communication, cultural norms emerge and persist over time (Latané, 1996; Schaller, Conway, & Tanchuk, 2002). It's hardly far-fetched to suppose that, in regions characterized by persistently high levels of disease, cultural learning and cultural communication processes conspired to sculpt cultural norms that prescribe behavioral means of minimizing the risk of disease transmission. In these regions (compared to regions with a lower disease prevalence), cultural values may advise against unrestricted sociosexuality, extraversion, and openness to experience. Individuals' behavioral dispositions – manifest as personality traits – may reflect these different cultural values.

These are three very different kinds of mechanisms through which worldwide variability in disease prevalence may lead to cross-cultural differences in personality. These mechanisms are conceptually independent, and are certainly not mutually exclusive. Even if there are differences in gene frequencies in different human populations, the expression of those genes may still be contingent upon input from immediate ecological circumstances. And even if there are disease-based genetic mechanisms contributing to these regional differences in personality, there may still be cultural mechanisms contributing as well. Our results cannot reveal the exact mechanism (or, more likely, mechanisms) through which disease prevalence might sculpt cross-cultural differences. That is a task for future research. And a considerable task it is: Each kind of mechanism summarized above has unique empirical implications that may require extensive programs of research to fully explore. Perhaps the greatest value offered by our results is to reveal that these programs of research are worth pursuing.

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