Chapter 5
Collectivism–Individualism, Family Ties, and Philopatry

5.1 Introduction

This chapter's three main topics, which are stated in the chapter title, are empirically analyzed in sequence in relation to the parasite-stress theory of values. All three are examined cross-nationally and across states of the USA. Also collectivism–individualism and philopatry are investigated across indigenous societies in the ethnographic record. Then we deal with the topic of family organization across species in relation to parasite stress. We discuss also reciprocal altruism of people and human-specific cognitive ability in relation to the parasite-stress theory. In this chapter, too, we address the ecological fallacy as well as some misconceptions about the scientific validity of the comparative method.

5.2 Collectivism–Individualism Across Countries

5.2.1 The Origin of the Hypothesis

Individualism and collectivism are fundamental to social scientists' descriptions of culture and cross-cultural differences (Triandis 1995; Hofstede 2001; Heine 2008). Until recently, however, a gap in the large research effort directed at cross-cultural variation in collectivism–individualism was the absence of a theoretical framework that can explain the variation.

With our colleagues Damian Murray and Mark Schaller, we hypothesized that collectivism (in contrast to individualism) functions as a defense against infectious diseases, and thus is more likely to be evoked in cultures that have a greater prevalence of parasites. The logical basis of this hypothesis is evident in all the major defining features of collectivist (versus individualist) value systems tabulated and discussed in the previous chapter (see Tables 4.1 and 4.2). Here we mention only a few of these.

R. Thornhill and C.L. Fincher, *The Parasite-Stress Theory of Values and Sociality: Infectious Disease, History and Human Values Worldwide*,
DOI 10.1007/978-3-319-08040-6_5, © Springer International Publishing Switzerland 2014

First, collectivists, in comparison to individualists, are embedded in their in-group and form durable in-group relations. This provides the health "insurance" of nepotism and other in-group altruism that manages the negative effects of contagion when it occurs in the in-group.

Second, collectivists make strong distinctions between in-groups and out-groups, whereas among individualists the in-group–out-group distinction is weak. Consequently, collectivists are more distrustful and avoidant of contact with out-group people. This xenophobic attitude can serve an effective anticontagion function by reducing exposure to novel infectious diseases that may be harbored in out-groups.

A third distinction between collectivism and individualism lies in their different emphases on conformity versus tolerance for deviance from the norms. Collectivism is characterized by a strong value placed on tradition and conformity, whereas individualism is characterized by a greater tolerance, and even encouragement of, deviation from the traditional status quo (Oishi et al. 1998; Cukur et al. 2004; Murray et al. 2011). Given that many specific customs and norms—such as those pertaining to food preparation (Sherman and Billing 1999) and hygiene—can serve as defenses against pathogen transmission, deviation from the customary status quo may pose a contagion risk to self and others, whereas conformity helps to maintain the integrity and effectiveness of these defenses against infectious disease. In sum, the behaviors arising from collectivist values (compared with the behaviors of individualism) are more likely to provide defenses against infectious diseases.

In contrast, individualistic values promote different important benefits to individuals. For instance, consider the individualistic values of independent self, intellectual autonomy, and openness to new social contacts and new experiences in general discussed in the previous chapter. Both the discovery and the spread of beneficial new ideas and technologies will occur more frequently when individuals are encouraged to think independently, deviate from existing traditions, and engage in interactions with out-group people. In geographical regions characterized by relatively low parasite stress, the benefits of collectivism (in terms of antiparasite defense) will be minimal, compared with the benefits associated with individualism. Under these ecological circumstances, individualistic values are more optimal or socially effective. However, within geographical regions characterized by a greater prevalence of parasites, the benefits of collectivism to individuals also will be greater, and outweigh benefits conferred by individualistic values. Under these circumstances, collectivistic values are likely to have more utility in terms of individual's inclusive fitness.

From this reasoning, it follows that worldwide variation in infectious diseases is expected to correspond to cultural norms toward individualistic versus collectivistic values. Specifically, our colleagues and we hypothesized that countries with low parasite stress will be individualistic, and that countries with high parasite stress will be collectivistic. Prior to our study, it had been shown that equatorial countries are more collectivistic than societies at higher latitudes (Hofstede 2001). This was consistent with our hypothesis because infectious diseases are more prevalent in equatorial regions (e.g., Guernier et al. 2004). Our hypothesis was tested directly first in the paper by Fincher et al. (2008). We discuss that study next.

5.2.2 *Measures of Collectivism–Individualism*

In our cross-national research with colleagues on collectivism–individualism in relationship to parasite stress, reported in Fincher et al. 2008, we used published data from four studies that provided somewhat different, but conceptually overlapping, highly correlated, measures of collectivism–individualism at the country level. Gelfand et al. (2004) reported on the "Global Leadership and Organizational Behavior Effectiveness Research Program" (GLOBE) measures of in-group collectivism across 62 contemporary societies. The GLOBE research program sampled 17,370 middle managers from 951 organizations in three industries (food processing, financial services, and telecommunications services). Samples were gathered during the years 1994–1997. Our analyses were of GLOBE's "in-group collectivism practices" measure. Gelfand et al. (2004) reported that these particular scores showed the greatest convergent validity with other independent measures of collectivism–individualism. Moreover, compared with the alternative measures summarized by Gelfand et al., this particular measure is the one most clearly based on actual behavior.

This scale consists of adding together responses to four questions that comprise a unidimension measuring the "degree to which individuals express pride, loyalty and interdependence in their families" (Gelfand et al. 2004, p. 463). Respondents indicated agreement on a 1–7 scale for each collectivism question. The largest values reflected greater collectivism and the smallest values were the highest individualism (Gelfand et al. 2004). We refer to this scale as *Gelfand In-group Collectivism.*

Hofstede (2001) presented data that was collected from 1967 to 1973 on an IBM employee attitude survey comprising 116,000 questionnaires from 72 countries. From these data, Hofstede created an individualism index, which we refer to as *Hofstede Individualism*. Hofstede (2001, p. 225) describes individualism–collectivism as: "Individualism stands for a society in which the ties between individuals are loose: everyone is expected to look after him/herself and her/his immediate family only. Collectivism stands for a society in which people from birth onwards are integrated into strong, cohesive in-groups, which throughout people's lifetime continue to protect them in exchange for unquestioning loyalty."

Suh et al. (1998) studied the role of following one's emotions (doing what one wants to do) versus that of following cultural norms (doing what one "should" do) for determining life satisfaction or well-being among members of nations. For this study, they developed a new measure of collectivism–individualism that combined Hofstede's values and the rankings of individualism for 41 nations by Triandis (a pioneering researcher in the field of cross-cultural psychology) in 1996. The inter-rater correlation between Hofstede's empirical values and Triandis' ratings for the 26 overlapping nations was 0.78 ($p < 0.001$). Thus, Suh et al. combined Hofstede's empirical data from the 1960s and 1970s and Triandis' more recent rankings to develop a measure of individualism presented on a scale from 1 (most collectivist) to ten (most individualist). We refer to this scale as *Suh Individualism.*

A fourth measure of collectivism–individualism was used in Fincher et al. (2008). Kashima and Kashima (1998) studied the phenomenon of pronoun-drop

across nations (71 countries and 39 languages). Pronoun-drop was defined as the acceptable dropping of first- and second-person pronouns from the dominant language. They demonstrated that pronoun-drop was found primarily in collectivist cultures, while the persistent use of the pronouns was found in individualist cultures. The correlation between first-person pronoun use and *Hofstede Individualism* across cultures was 0.75 ($p < 0.01$). Kashima and Kashima (1998) argued that the dropping of first-person pronouns is predominant in collectivist cultures, because in such cultures the individual is de-emphasized (e.g., the de-emphasis of "I") to such an extent that the personal self is unimportant relative to the in-group or collective (see also Chap. 4). Pronoun-drop, then, provides an independent measure of collectivism–individualism among cultures, and one based on linguistic norms. We recoded their data such that pronoun-drop cultures scored a 1; those that used first-person pronouns were scored a 0. We refer to this scale as *Kashima Collectivism*.

5.2.3 Cross-National Parasite Severity

In part with our colleagues Murray and Schaller, we developed multiple measures of parasite severity across countries that were used in Fincher et al. (2008). By parasite severity, we mean the number of infectious disease cases, not the number of infectious diseases. In Fincher et al. (2008), we used two highly intercorrelated parasite severity measures to study cross-national variation in values. One focuses on historical parasite severity, and the other on contemporary parasite severity.

The measure of *Historical Parasite Severity* estimates the severity of nine pathogens detrimental to human survival and reproductive success (leishmanias, trypanosomes, malaria, schistosomes, filariae, leprosy, dengue, typhus, and tuberculosis) within each of 93 countries worldwide. (Murray and Schaller later updated this measure to include more of the world's countries (Murray and Schaller 2010). Throughout the book, when we analyze *Historical Parasite Severity*, we are referring to our earlier measure based on 93 countries.) The severity of the nine parasites was estimated on the basis of old atlases of infectious diseases and other historical epidemiological information dating back to the early 1900s. The severity estimates (coded on either three- or four-point scales) were standardized (transformed into *z*-scores), and the mean of these nine standardized scores served as the estimate of the historical severity of pathogens in each country (for details, see Fincher et al. 2008). The validity of this measure is shown for example by its very high correlation with an index of similar parasites used by Gangestad and Buss (1993) to assess historical parasite severity within a small sample of countries (see Fincher et al. 2008). Their index was based on one derived independently by Low (1990, 1994) from historical records of parasite severity from disease atlases extending back to the early 1900s.

Our measure of *Contemporary Parasitic Severity* used data we obtained in June–August 2007 from the Global Infectious Disease and Epidemiology Network (GIDEON) (described below). For each country, we calculated a parasite severity

index for a subset of infectious diseases, using a set of parasites similar to those used by others in previous cross-cultural research on parasite severity and human social life (e.g., Low 1990, 1994; Gangestad and Buss 1993; Gangestad et al. 2006; Quinlan 2007). We used the same seven classes of infectious diseases, but expanded the parasites included in the classes to all entries in GIDEON in each class (a total of 22 parasites). We recorded the country-wide disease level of the seven groups of parasites: leishmaniasis, trypanosomes, malaria, schistosomes, filariae, spirochetes, and leprosy. We used GIDEON's three-point scale of parasite severity (3 = endemic, 2 = sporadic, 1 = not endemic) depicted in the geographical distribution maps of the diseases in GIDEON.

The validity of our *Contemporary Parasite Severity* index is shown by two analyses (Fincher et al. 2008). First, it is highly correlated with an index mentioned earlier of similar parasites used by Gangestad and Buss (1993) to assess historical parasite severity within a sample of countries. Second, our index also shows a high correlation with a separate measure of contemporary parasite severity known as DALY (Disability Adjusted Life Years). DALY is a measure of morbidity and mortality across the globe from many sources, as reported by the World Health Organization (WHO). We used the DALY for infectious diseases per 100,000 population, as reported by WHO for the year 2002 (WHO 2004). This DALY combines into one measure the time lived with disability and the time lost due to premature mortality; one DALY equals one lost year of healthy life and the associated burden of infectious disease. It is a measurement of the gap between current health status and an ideal situation where everyone lives into old age free of infectious disease and disability from contagious agents (www.who.int). For the 192 countries for which we had data on both this DALY corrected for population size (ln transformed due to skew) and our contemporary parasite-severity index, the correlation is high: $r = 0.74$, $p < 0.0001$. Hence, our contemporary parasite severity index correlates highly with historical and contemporary parasite stress as measured by others. *Infectious Disease DALY* is our label throughout the book for the DALY for infectious diseases corrected for population size and log transformed.

5.3 Findings

The results supported the hypothesis that, across countries, pathogen severity will correlate negatively with measures of individualism and positively with measures of collectivism (see Table 5.1). Across two measures of pathogen severity, and the four measures of individualism–collectivism, the results are consistent with the hypothesis. As presented in Table 5.1, *Historical Parasite Severity* was an especially strong predictor of both individualism and collectivism (absolute magnitude of the r's ranged from 0.63 to 0.73; all p's < 0.001). *Contemporary Parasite Severity* showed the identical pattern of results, although the magnitude of the correlations was somewhat less strong.

Table 5.1 Pearson zero-order correlations between two measures of parasite severity, and four measures of individualism–collectivism

	Parasite-severity measure	
Values measure	Historical	Contemporary
Hofstede Individualism	–0.69 (68)	–0.59 (68)
Suh Individualism	–0.71 (58)	–0.58 (58)
Gelfand Collectivism	0.73 (52)	0.56 (57)
Kashima Collectivism	0.63 (70)	0.44 (70)

All p's < 0.001; the number of countries in each analysis is in parentheses (results were originally reported in Fincher et al. 2008; reprinted with permission)

To address potential alternative causal explanations, in Fincher et al. (2008), we assessed the relation between *Historical Parasite Severity* and collectivism–individualism while statistically controlling for other variables that earlier published research mentioned as possible causes of collectivism–individualism. Four additional variables were assessed: Gross Domestic Product (GDP) per capita (a measure of wealth of the average person in a country), Gini (the wealth disparity among the people of a country), population density, and residual life expectancy. High values of GDP per capita and of Gini mean high average wealth per person and high wealth disparity among people, respectively. Residual life expectancy means the deviations from the general statistically fitted regression line when life expectancy of both sexes combined is regressed on *Historical Parasite Severity*. This residual is the variation in life expectancy not accounted for by *Historical Parasite Severity*. Of the four additional variables, only GDP per capita and Gini were correlated reliably with collectivism–individualism. Residual life expectancy correlated significantly with only one of the four collectivism–individualism measures, and population density failed to correlate significantly with any of the four measures. By contrast, GDP per capita and Gini were substantially and significantly correlated with all four individualism–collectivism measures (all p's < 0.05). Consequently, we conducted four multiple regression analyses, in which *Historical Parasite Severity*, GDP per capita and Gini were entered simultaneously as predictors of each of the four individualism–collectivism measures. An identical pattern of results emerged across all four analyses. There were no unique effects of Gini (all p's > 0.05). By contrast, GDP per capita did exert unique predictive effects (all p's < 0.05); greater GDP per capita was associated with greater individualism and less collectivism. Of primary interest, parasite severity also uniquely predicted all four measures of individualism–collectivism (all p's < 0.05). Thus, while other variables (like economic development) also may predict cultural differences in collectivism–individualism, these other variables cannot account for the predictive effects of pathogen severity (also see Chap. 11 on economics and parasite adversity).

Furthermore, Fincher et al. (2008) reported that the pattern of results above relating collectivism–individualism to parasite stress was replicated when cultural regions (rather than countries) were treated in analysis. Regardless of whether the world is divided up according to Murdock's (1949) six world regions, or Gupta and

Hanges' (2004) ten cultural regions of the world, composite scores on pathogen severity correlated negatively with composite scores on individualism, and positively with composite scores on collectivism. These correlations were very strong in some cases. For example, when Murdock's six world regions were used in analysis, the correlation between *Historical Parasite Severity* and the Gelfand et al. collectivism measure was 0.93 ($p = 0.004$, $n = 6$); and when Gupta and Hanges' (2004) ten cultural regions were used in analysis, the correlation was 0.80 ($p = 0.003$, $n = 10$). Hence, the variation in the two key variables of parasite stress and collectivism within world or cultural regions is consistent with the same pattern worldwide. (Murdock's world regions, a typical division of the world by anthropologists into cultural regions, are described more fully later in the book.)

In sum, across multiple measures, we found that worldwide variation in parasite severity substantially predicted societal values of individualism–collectivism. Within ecological regions characterized by higher severity of infectious diseases, human cultures are characterized by greater collectivism. The size of this effect was substantial and remained significant even when controlling statistically for potential confounding variables. The effect also remained strong when broader cultural regions (rather than individual countries) were used in analysis. These findings are consistent with the hypothesis that individualism confers benefits upon individuals, but the behaviors that define individualism also enhance the likelihood of pathogen transmission, and thus are maladaptive under ecological conditions in which pathogens are highly prevalent. In contrast, the behaviors of collectivism function in antipathogen defense, and thus are adaptive under conditions of high pathogen prevalence.

These findings help to explain additional variables that were known prior to Fincher et al. (2008) to be correlated with individualism–collectivism. A positive correlation between individualism and latitude has frequently been noted, but never explained (Cohen 2001; Hofstede 2001). Our results imply that this correlation is substantially accounted for by parasite adversity: the meteorological and ecological conditions associated with lower latitudes provide the ideal circumstances for the proliferation of parasites (Guernier et al. 2004), which, in turn, evoke collectivist cultural values as a defense against the high parasite adversity.

Multiple researchers have observed a strong, positive correlation between GDP per capita and individualism and have suggested ways through which economic affluence might lead to individualism (Triandis 1995; Hofstede 2001). Our results indicate that the sizeable correlation between economic productivity and individualism results, in part, from each of these two variables sharing variance with parasite severity. Even the apparently unique effect of GDP per capita may indirectly reflect some causal role of pathogens, given that infectious diseases are potent inhibitors of economic development (Chap. 11). Thus, the traditional literature on collectivism–individualism over-estimates economic influences, while underestimating the causal influences of parasites, a topic we return to later in the book when we treat fully parasite stress and the wealth of nations (Chap. 11).

5.4 Nonzoonosis and Collectivism–Individualism Cross-Nationally

One limitation of the study reported in Fincher et al. (2008) is that it employed relatively crude measures of pathogen severity. One measure estimated overall parasite severity (number of cases of disease) on the basis of data pertaining to a diverse set of nine human infectious diseases represented in epidemiological atlases that refer back to the early 1900s. A second measure estimated overall parasite severity (number of disease cases) on the basis of data pertaining to a diverse set of 22 human infectious diseases, obtained in 2007 from an online database of contemporary human infectious diseases (GIDEON). Statistical analyses attest to the reliability and validity of these measures (e.g., Fincher et al. 2008; Thornhill et al. 2009; Murray and Schaller 2010), but these measures are indicators only of overall parasite adversity.

Most importantly, these measures fail to discriminate between conceptually distinct categories of human parasites defined by different modes of transmission to humans. Parasitologists and epidemiologists classify human diseases into three distinct categories based on their modes of transmission: zoonotic, multihost, and human-specific (Smith et al. 2007). Zoonotic parasites develop and reproduce entirely in nonhuman hosts (livestock and wildlife) and can infect humans as well, but are not transmitted directly from human to human. Multihost parasites can use both nonhuman and human hosts to complete their life cycle and may be transmitted directly from human to human as well as to humans through between-species transmission. Human-specific parasites are transmitted only from human to human (although ancestrally they often had a zoonotic transmission origin; see Pearce-Duvet 2006).

These categorical distinctions are important in the parasite-stress theory of values. The cross-national differences in collectivism–individualism discussed earlier are predicted by a parasite-stress theory of *human sociality* that especially emphasizes the potential infection risks associated with interactions with other humans. Hence, according to the parasite-stress theory of human sociality, worldwide differences in the domains of human values are unlikely to correlate substantially with the presence of zoonotic parasites (which cannot be transmitted from human to human), but should correlate strongly with the presence of nonzoonotic parasites (which have the capacity for human-to-human transmission). Empirical evidence consistent with this prediction would provide unique and novel support for the parasite-stress theory of human sociality. Thornhill, Fincher, Murray et al. (2010) reported such evidence, which is summarized below. (See the original publication for further details about methodology and results.)

5.4.1 Methods

For each of 227 countries, we computed three indices of human *Disease Richness* (number of kinds of human parasitic diseases) based on the presence or absence of every human infectious disease cataloged in the GIDEON database. GIDEON is a

frequently updated, subscription-based online database of human infectious diseases available to the medical community and researchers. GIDEON data have been used extensively in research on the global distribution of infectious diseases (e.g., Guernier et al. 2004; Smith et al. 2007; Thornhill et al. 2009; Fumagalli et al. 2011). Our indices were generated from data obtained from GIDEON in 2008. We classified each human infectious disease as either zoonotic, multihost, or human-specific, according to Smith et al.'s (2007) classification scheme, with updates based on more recent epidemiological information in GIDEON and in other sources (see Thornhill et al. 2010 for details of updating). This classification has 154 diseases as zoonotic (e.g., rabies, hantavirus), 40 diseases as multihost (e.g., leishmaniasis, Chagas disease, Dengue fever), and 117 diseases as human-specific (e.g., measles, cholera, filariasis). For each country, we computed separately the sums of all zoonotic diseases, multihost diseases, and human-specific diseases that GIDEON listed as having a presence within that country. These three sums represented three distinct indices of *Disease Richness*. Across all countries combined, the mean parasite richness scores were as follows: zoonotic: mean (M) ± standard deviation = 53.92 ± 10.40 (range = 38–87); multihost: M ± SD = 23.59 ± 2.81 (range = 20–32); human-specific: M ± SD = 102.33 ± 2.96 (range = 98–110). In Thornhill et al. (2010) we did not publish the data for the numbers of the three disease categories per country; these are given in this chapter's Appendix 1, and the methods used in distinguishing the three disease categories are described in Thornhill et al. (2010).

The three parasite indices do not distinguish between certain aspects of disease transmission—e.g., vector-borne versus those requiring direct contact—nor need they. Whether a disease transmitted between people is carried through the air by way of a mosquito or by expelled mucus droplets is not relevant to our main hypothesis about differences between nonzoonotic and zoonotic infectious diseases and values. Similarly, the taxon of the disease—e.g., fungi, viral, helminth ("worm"), etc.—is not relevant to this hypothesis. An argument could be made for the importance of investigating the impact of differences among infectious disease virulence in understanding the evolution of values, but that research has not been done yet.

Note that these measures of parasite *richness* are only indirect measures of the *severity* (the number of infectious-disease cases) that parasites impose on people. Nevertheless, when we did the research using the richness measures, there was evidence that human parasite richness covaries substantially with human parasite severity (Fincher et al. 2008; Fincher and Thornhill 2008a, b); consequently, these measures of parasite richness were used to test hypotheses derived from the parasite-stress theory of human sociality. After we present the results from our study of richness of the types of human infectious diseases, we then discuss new research on severity of the types in relation to collectivism–individualism. The results are similar across the two infectious-disease measures.

Table 5.2 Pearson zero-order correlations and *p*-values between each measure of parasite richness and each measure of collectivism–individualism

Values measure	Parasite richness measure						
	Human-specific	*p*	Multihost	*p*	Zoonotic	*p*	n
Hofstede Individualism	−0.60	<0.001	−0.70	<0.001	−0.17	>0.10	67
Suh Individualism	−0.58	<0.001	−0.61	<0.001	−0.20	>0.10	57
Gelfand Collectivism	0.51	<0.001	0.51	<0.001	0.27	0.04	57
Kashima Collectivism	0.35	0.003	0.45	<0.001	0.19	>0.10	70

n = the number of countries in each analysis (results were originally reported in Thornhill et al. 2010)

5.4.2 Findings

The findings of the study based on *Disease Richness* are presented in Table 5.2. Each of the two individualism measures correlated substantially negatively with both human-specific and multihost parasite richness; in contrast, they correlated only weakly with zoonotic parasite richness. Analogously, each of the two collectivism measures showed moderate to strong positive correlations with both human-specific and multihost parasite richness, and weaker correlations with zoonotic parasite richness.

Follow-up regression analyses included all three parasite-richness indices as predictors of the values. The results revealed that the predictive effects associated with human-specific parasite richness remained significant with *Suh Individualism* ($p < 0.001$), and marginally significant on two other measures (*Hofstede Individualism and Gelfand In-group Collectivism*; p's = 0.09 and 0.12, respectively). The predictive effects of multihost parasite richness remained significant on three of the measures (both individualism measures, as well as the *Kashima Collectivism*; all p's < 0.001) and marginally significant on the additional measure (the *Gelfand In-group Collectivism*; $p = 0.10$). In contrast, the modest relations with zoonotic parasite richness actually *reversed* in sign when controlling for shared variance with the other parasite-richness measures. For the two individualism measures, the reversal in sign actually resulted in significant *positive* relations with zoonotic parasite richness (both p's < 0.002), in direct contrast with the significant *negative* relations with human-specific and multihost parasite richness.

Additional regression analyses that included the zoonotic index and the nonzoonotic composite index (multihost and human-specific combined) as predictors revealed a clear distinction: nonzoonotic parasite richness was a unique negative predictor of individualism (both p's < 0.001), and a unique positive predictor of collectivism (all p's < 0.001); zoonotic parasite richness had no consistent unique effect, and any such effect at all (on the two individualism measures) was exactly opposite to that indicated by the correlations in Table 5.2.

In sum, although the Fincher et al. (2008) study (discussed earlier in this chapter) provided a substantial body of evidence linking the prevalence of human infectious diseases to the human value dimension of collectivism–individualism, that study

was limited by the fact that (a) the previous indices of human parasite adversity represented only a small fraction of the many infectious diseases that affect human health, and (b) these indices failed to distinguish between different human disease categories defined by their mode of transmission to humans. To address these limitations, Thornhill et al. (2010) used data bearing on more than 300 different human infectious diseases, computed separate indices assessing the prevalence of three functionally distinct categories of these diseases (human-specific, multihost, zoonotic), and examined the extent to which each index uniquely predicted cross-national differences in societal values. The results were convincing.

Both human-specific and multihost parasite richness predicted uniquely cross-national differences in collectivist–individualist values. Zoonotic parasite richness contributed little, if at all, to previously documented cross-national relationships between parasite prevalence and these values. Thus, worldwide variation in the values predicted by parasite prevalence appears to be attributable almost entirely to the prevalence of nonzoonotic diseases. The richness of human-specific parasites and the richness of multihost parasites were approximately equally predictive of collectivistic–individualistic values. These findings conform to the functional logic of the parasite-stress model, because collectivism–individualism has consequences for a broad range of behaviors, including behaviors with implications for interpersonal contact (e.g., approach versus avoidance of unfamiliar peoples), as well as behaviors with implications for interspecies transmission of pathogens (e.g., violation versus conformity to cultural norms pertaining to hygiene).

The preceding analyses found that relationships linking parasite prevalence to collectivism–individualism are attributable primarily to the prevalence of nonzoonotic parasites (human-specific and multihost parasites). Compared to the effects of nonzoonotic parasite richness, any effects associated with zoonotic parasite richness were negligible. Before conclusively ruling out an important contribution of zoonotic parasites to these worldwide values, it is critical to consider an alternative explanation, based on differential measurement error. It is possible that epidemiologists and health agencies are especially attentive to diseases that are transmitted from human to human, whereas the presence of human zoonotic parasites may be relatively poorly recorded. If so, then simply for reasons of differential measurement error, zoonotic parasite richness would be expected to correlate less strongly than nonzoonotics with any outcome variable. The plausibility of this alternative explanation is undermined by evidence that many zoonotic diseases are monitored by the Centers for Disease Control and Prevention and other relevant agencies worldwide as zoonotics are thought to be an important source of emerging human infectious diseases (Greger 2007; Jones et al. 2008). Some zoonotics, however, may escape surveillance by these agencies (e.g., Maudlin, Eisler and Welburn 2009). One way to address this alternative explanation empirically is to show that the zoonotic parasite-richness index is measured with sufficient fidelity to predict additional outcome variables to which it is conceptually related—such as the presence of livestock within a country. Many zoonotic diseases are contracted from livestock, and so we should observe an especially strong relationship between livestock and zoonotic parasite richness—but only if the index of zoonotic parasite richness is measured with a high degree of validity.

For 205 countries, we computed the total number of avian and mammalian livestock over the period from 2000 to 2004 (data obtained from the Global Livestock Atlas of the World Agricultural Information Center; http://www.fao.org/index_en.htm). To correct for skew, this value was log-transformed prior to analyses. Correlations with the three parasite richness indices were as follows: human-specific, $r=0.31$; multihost, $r=0.44$; zoonotic, $r=0.78$ (all p's <0.001). In a follow-up regression analysis with all three parasite-richness indices simultaneously entered as predictors, only zoonotic parasite richness remained significantly, positively related to the total number of livestock ($p<0.001$). These results reveal that the zoonotic parasite-richness index is measured with sufficient accuracy to be a uniquely powerful predictor in domains of theoretical relevance. Differential measurement error, therefore, is unlikely to account for the fact that nonzoonotic parasite richness predicted cross-national variability in collectivism–individualism to a much greater extent than did zoonotic disease richness.

5.4.3 Severity of Disease Types

Subsequent to the cross-national research on collectivism–individualism published in Thornhill et al. (2010) (just described) based on parasite-richness measures, we tallied the parasite severity (number of cases) for all the diseases in GIDEON in 2009 separated by zoonotic and nonzoonotic categories, and then obtained severity indices for each category for each of the countries of the world; our methods are described fully in Fincher and Thornhill (2012) and the parasite severity data are published as electronic supplements to Fincher and Thornhill (2012). The above results for parasite richness in relation to collectivism–individualism were repeated with the new parasite severity measures. All analyses showed that nonzoonotic severity related more strongly to collectivism–individualism than did zoonotic severity. For example, with *Suh Individualism*, nonzoonotics, $r=-0.62$, $p<0.0001$, and zoonotics, $r=-0.23$, $p=0.09$; $n=57$ for both. Also we found that the measures of parasite richness were correlated nearly perfectly with measures of parasite severity. For example, nonzoonotic severity and nonzoonotic richness showed an $r=0.96$ across 222 countries, and zoonotic severity and zoonotic richness correlated $r=0.98$ across the same countries.

5.5 Scientific Strengths of the Research

We emphasize that the research studies reported earlier on collectivism–individualism in relation to parasite richness and severity across countries have two major scientific strengths. First, they offer general and synthetic comparative perspectives on the important value dimension collectivism–individualism that may explain it in terms of parasite stress as both proximate and ultimate causation. Another strength

of the studies pertains to the data sets involved. Data collection was not biased in favor of the hypothesis that parasite adversity causes values. Unconscious biases can enter at the data-collection stage in scientific studies to bias results toward a hypothesis held by researchers, but this cannot be the case here because the data sets on values and parasite adversity were assembled by researchers who were unaware of the parasite-stress theory of values.

5.6 A Caution About Interpreting Correlational Results

At this point in this book, it is important to emphasize that sometimes the scientific significance of the patterns of results presented in this chapter is misunderstood. People sometimes stumble intellectually when shown these kinds of results, and make statements such as, "[t]he researchers have some correlations only and correlation can not prove causation," or "[t]o be convincing, they must conduct experiments." Or, as one researcher put this, "[o]nly experiments can truly test theory." (Stearns 1976, p. 42)

The lines of thinking behind these statements, which are articulated often in the form of criticisms of comparative research findings like those presented earlier, are scientifically erroneous. They fail to understand the concept of correlation in scientific research. More specifically, they fail to recognize that each method of applying the scientific method—lab experiments, field experiments, observation, and the comparative method—has strengths and weaknesses, uses correlational data, and can demonstrate causation. Our discussion below of common misunderstandings and controversy about comparative analyses is based on the more extensive treatment in Thornhill and Fincher (2013).

First, note that *all* scientific findings are correlational—that is, they are *co-relationships* between dependent and independent variables. Alexander (1978) pointed this out in response to creationist critics' claims that evolutionary biology is fatally flawed because its core evidence provided by Darwin is based on correlations obtained by the comparative method. (Also see Thornhill and Palmer (2000) for a related response to certain secularist critics of evolutionary theory as applied to human behavior.) So, to say that correlational data do not count or are less convincing than data generated from experimentation is to misunderstand fundamentally scientific methodology and evidence; the findings from experiments are just as correlational as those from statistical analysis called correlational or regression analysis. In all cases, the scientific value of a finding—its ability to address causation—depends solely upon the control of confounders, not the type of method itself. This is true for studies conducted at individual or group levels.

In discussing scientific methodology with students and colleagues, we have observed that the equivalence of all scientific findings as correlated variables can be understood easily by using the simple example of an imaginary botanical greenhouse experiment to test the hypothesis, say, that potassium causes the growth of hibiscus plants. The researcher grows hibiscus under three different levels of potassium

supplementation to the soil. The same basic soil is used in each of three treatments of ten plants per treatment, and all plants have the same watering schedule and genetic background. The researcher's hypothesis will be supported if the data show that the average hibiscus growth across treatments corresponds positively to the amount of experimentally added potassium; that is, by data of a positive correlation between the level of potassium and hibiscus growth. In this case, the actual analysis used to see if the correlation is present is not a statistical correlation analysis per se, but the test is indeed for a corelationship between variables. Note that if the experimenter uses only two treatments and tests the hypothesis with a *t*-test for a difference in average growth between treatments, the procedure is still directed at establishing whether the predicted corelationship exists between the two variables, potassium level and plant growth. Statistics, whether *t*-statistics, correlation coefficients, or other statistics are interchangeable because they are all for determining if there is a correlation between variables. Note, too, that this experiment is based on comparative data, comparing plant growth under different levels of a nutrient. All scientific results are fundamentally both correlational and comparative.

Say the researcher finds the positive correlation predicted by the hypothesis and submits a paper describing the research to a scientific journal or presents the work at a scientific conference. Critics would point to the potential confounds unaddressed; they cannot accept the results as proof of causation, because another factor (potential confound) may be responsible for the correlation of the two variables. For example, it may be that a fungal parasite of hibiscus was present in part of the greenhouse, but not in other parts. Presumably, hibiscus has hundreds of types of parasites, so there is much room for potential confounds, just in the domain of parasites alone. Or perhaps there was a slight, but significant lighting or temperature or humidity variation across parts of the greenhouse, and so on. This example illustrates the general rule in interpreting scientific results, all of which are correlational: the confidence one can have in a study's finding depends entirely upon the ability of the procedure used to control potential confounds. Hence, this confidence is independent entirely of what kind of test method is conducted (experimentation, observations, or a study employing the comparative method).

The comparative method we have used in this chapter to produce findings uses statistical controls, which are routine, straightforward, and scientifically respected analytical procedures for controlling potential confounds. Recently, Minkov (2011, p. 35) criticized our research with colleagues on the cross-national relationship between parasite stress and collectivism–individualism (Fincher et al. 2008) by saying that, although the patterns found in the research are very strong ones, even strong correlations may not identify causation. Of course, this is a correct point. As discussed earlier in this chapter, it is the reason we used control procedures in our cross-national analysis, which allowed us to conclude that parasite stress significantly and positively predicts collectivism when various potential confounds are controlled (Fincher et al. 2008, and see earlier). And this is why we also draw on many lines of evidence to evaluate hypotheses. For example, our predictions about collectivism and parasite stress at the cultural level are bolstered by findings of studies of individuals and their xenophobic attitudes in relation to pathogen sensitivity (Faulkner et al. 2004).

The comparative method also employs the control procedure of randomization. This is used commonly also, for example, in field experimentation in biological research to randomize the locations of treatment replicates in order to control site effects. The randomization procedure in such field experimentation attempts to control unknown potential confounds by making any influence these confounds may have independent of the treatments used. In comparative studies, the kind and diversity of comparison groups allow randomization of the influence of many potential confounders on an effect of interest (Alexander 1978). The parasite-stress theory of values predicts a positive correlation between the degree of collectivism and the degree of parasite stress. In our cross-national analyses, parasite stress corresponds to the "treatment" variable. Consider nations with high parasite stress: They vary in many ways such as language, religion, cooking and clothing styles, diet, subsistence type, social and other ecological features, and so on. The same can be said for nations with low parasite stress. Indeed, at any point across the treatment variable there is much variation in many cultural traits. Thus, across the comparisons of values systems in different countries, many potential confounds are randomized with regard to the treatment variable of degree of parasite adversity.

Now suppose the hibiscus researcher repeats the work, but with careful attention to the criticisms of the first study such that numerous potential confounds are controlled: lighting, temperature, humidity, and the use of fungicide and other pesticides are equal across all treatments, and also the locations of the plants within the greenhouse are randomized in regard to potassium treatment. Again, the correlation is found and the results from the study are presented to scientists. Critics could agree that there is now good evidence that potassium causes hibiscus growth; that is, there is evidence of a correlation—as predicted by theory—between potassium and hibiscus growth unconfounded by other factors so far examined. This confidence that the results show causation stems from the new procedures used to control for confounders, which always constitutes credibility of causation, regardless of the method of testing (experiment or otherwise).

The confidence these critics have in the study, however, has important limits: the research was conducted in a greenhouse, a very unnatural environment. Critics would reasonably still want to see results from nature showing the same positive correlation that was found in the "laboratory" setting. Say the researcher obtains data that show that, within a uniform geographical region, high-potassium-soil locations contain hibiscus plants that grow faster than those in low-potassium-soil locations, which provides evidence that the lab results have meaning in nature. Relevant here, also, would be a field experiment in which potassium levels are manipulated by the researcher in a more natural ecological setting than the lab. It is nature, after all, that science is charged with discovering; unnatural or laboratory results are supportive of a hypothesis about nature only when they are shown to address the natural environment. We emphasize that our studies using the comparative method inherently contain the naturalness of the groups compared, which, as in our analyses earlier, are people living in their cultural ecology.

All the standard methods of testing hypotheses are valid scientific procedures and each can address causal conjectures (hypotheses). Each method has advantages

and disadvantages, and no method is superior to all others. Experiments typically involve manipulation of presumed causes, which can yield manipulation anomalies that render the results useless or misleading. The strengths and weaknesses of each of the methods of testing are discussed further in Thornhill (1984). Given that no single method is superior to another, confidence in a hypothesis is increased by the use of multiple methods of testing a prediction of a hypothesis: lab experiment, field experiment, comparative method, and field observations without any manipulations of variable(s). As we show in this book, the parasite-stress theory of values is supported by the full range of scientific-testing methods: experimental, comparative, and observational in both contemporary and ethnographic societies.

We emphasize again that the ability of findings to address causation (to test any hypothesis) depends only on the quality of controls for confounding variables. There is no qualification or exception to this basic feature of the scientific method. Thus, the specific method of testing is always, in itself, irrelevant. When thorough controls are in place, correlation documents causation.

In each of the methods of scientific testing, researchers must decide which variables to control, whether by manipulation or statistically. Because there are always many possible confounds to be considered, in practice, scientists choose appropriate controls based on the hypothesis under investigation; yet sometimes, in order to get their paper published, they must control for the favorite candidate confounds of particular reviewers of the submitted papers; this applies equally across methods.

Critics' demands for controls, however, can lead to an analysis that commits the "partialling fallacy" (Gordon 1968). This fallacy is the use of control variables that are not based on a specified theoretical model. It is called the partialling fallacy in reference to the statistical procedure of multiple regression and partialling (controlling) many different variables that are claimed by critics to be confounds according to their intuitions only. Yet the partialling fallacy must be considered in any application of the scientific method, regardless of whether it tests with experimentation, observation, or comparative analysis. This fallacy is committed widely and is criticized appropriately because it is not a test of a causal conjecture (scientific hypothesis). The only thing it tests is the purely statistical notion that an observed result can survive the addition of any conceivable control variable(s). In proper hypothesis testing, a control is included in a study only when a specified theoretical context demands it. Without this basic understanding of hypothesis testing, the partialling fallacy may be committed, and if so, results are useless for testing causal ideas (hypotheses).

In Chap. 2, we mentioned the great importance of the scientific method—it is *the* method for understanding causes of natural things. Some scholars have pointed out that Francis Bacon, one of the method's founders, was adamant about the requirement of "experimentation" to test ideas and thereby learn the truth of our universe. This is certainly true, but to Bacon "experiment" meant simply searching for data that a hypothesis says must exist (for further discussion, see Wilson 1998). The word "experiment" now has a different meaning in science in that it is typically applied when a method of testing a hypothesis manipulates a presumed cause. The erroneous opinion that only this form of experiment provides accurate scientific

results has led to inappropriate conclusions and research directions in biology. Mayr (1982) discusses some well-documented cases of this in the history of biology.

Science is the study of the causes of the effects making up the natural world. Most fundamentally, the process of discovering such causes is by construction and refinement of correlations. Science progresses toward deeper and deeper knowledge by improving the understanding of correlation between hypothesized cause and effect. In Thornhill and Fincher (2013), we discuss further the fundamental role of correlation in scientific research.

5.7 Ecological Correlations, the Ecological Fallacy, and Multilevel Modeling

Our correlational comparative results presented earlier, as well as other such results throughout the rest of the book, most directly apply to aggregated data on values and parasite stress. We warned against making the naturalistic fallacy in Chap. 1: the logical error of concluding that fact arising from scientific evidence identifies moral direction. Just above we warned against making the partialling fallacy. A third fallacy we want to address is using aggregate results obtained from a group to which individuals belong to infer the features of the individual; this is the so-called ecological fallacy (Robinson 1950), which is discussed widely in the literature and appropriately cautioned against.

There are multiple forms of ecological-fallacy thinking. One has to do with inferring an individual's score on a variable from a regional aggregate score of the same variable. In aggregate or on average, people in some regions experience more infectious disease than in other regions. This is not to say that everyone in high parasite-stress regions experiences a lot of infectious diseases. Some people in such regions will have encountered few, some more, and some many infectious diseases. Some people will have more effective immune systems and thereby not become "infected," even when exposed to the same parasite adversity. A randomly selected individual from a high parasite-stress region could fall in any of these categories. Similarly, we are not saying that all individuals in high parasite-stress regions are extreme collectivists or that all individuals in low parasite-stress regions are ultra-liberal. There will be variation in values among individuals in any such region. We are saying that the measures of values discussed earlier identify the values of individuals in a region on average. We are saying also that the aggregate scores we use have meaning in terms of the general pattern of cultural behavior or of parasite stress experienced in regions and therefore can be used to test predictions of the parasite-stress theory of values.

Robinson (1950) has been criticized for taking an overly individual-centric view because it ignores contextual variation (Subramanian et al. 2009). Many researchers have suggested multilevel modeling is what is needed to account simultaneously for individual level processes and contextual differences (Subramanian et al. 2009; Pollet et al. in press). When these researchers suggest multilevel modeling, what

they typically mean is conducting studies that incorporate simultaneously within a single statistical model both individual-level and group-level data. Multilevel modeling also takes place when researchers synthesize studies conducted at cross-cultural levels and individual levels, even when conducted by different researchers. We have incorporated individual-level and group-level research throughout this book in order to evaluate the basic hypotheses of the parasites-stress theory of sociality.

Another form of ecological-fallacy thinking is when one automatically assumes that two variables generally co-occur within the individual (such as collectivism and parasite stress or individualism and gender equity) in a region. This may or may not be the case for any given set of variables. There is much evidence that our assumption of co-occurrence within the individual of our central variables is the case in general. Vandello and Cohen (1999) showed that their measure of USA state-level collectivism–individualism does not make this form of the ecological fallacy. Also, a range of studies has documented the association between individual-level values and individual-level strength of the behavioral immune response. Clay et al. (2012) have provided evidence that disgust sensitivity and various other measures of concern about infectious diseases are associated positively with collectivism at the level of individual people. Inbar et al. (2012) and Terrizzi et al. (2013) reported this same association, but with conservatism rather than collectivism. This association is an assumption in the cross-cultural research that shows the positive relationship between collectivism and parasite stress. Hence, evidence indicates that it is actually the case that individuals with high pathogen-related disgust and cognitions of contagion concern are more collectivist/conservative, whereas people with low disgust and contagion concern are more individualistic/liberal. Throughout this book we marshal both aggregate and individual-level analyses to make claims about pathogen stress as a causal force.

Although the ecological fallacy warning is typically applied in interpreting comparative correlation results such as those presented earlier, it applies to any finding of difference between two groups or among more groups. This is the case with any method of testing a hypothesis, whether it involves experimentation or other methods, and regardless of the statistical methodology used.

5.8 Family Ties Cross-Nationally

As an additional measure of collectivist and individualist values in our cross-national research, we conducted analyses on the strength of "family ties" within each country—measured as a numerical composite variable of multiple self-report items included in the World Values Survey ($n = 78$ countries). These items quantify allegiance to the extended family, which, as we have emphasized, is a defining feature of collectivistic value systems. The family-ties variable correlates highly with the measures of collectivism–individualism discussed earlier (see Thornhill et al. 2010). Thornhill et al. (2010) reported that parasite stress was positively associated

with a measure of family ties across modern countries. Subsequently, we explored a new and better measure of the strength of family ties at the cross-national level using updated World Values Survey files from a recently produced publicly available dataset unavailable at the time of Thornhill et al. (2010). We now turn to a summary of our research using the updated measure of family ties (for details about methods and results, see Fincher and Thornhill 2012).

5.8.1 Methods

Our new measure of the strength of family ties indexed the importance of family loyalty and interdependence, and was similar to the measure of collectivism–individualism used by the GLOBE project (see above; House et al. 2004) and by Alesina and Giuliano (2010). Our new data came from the 1981–2007 pooled dataset of the World Values Survey (www.worldvaluessurvey.org) for the following five items: (1) how important is family in your life? (We used the proportion of those who chose "very important."); (2) the respondent had to endorse one of two statements: (a) regardless of what the qualities and faults of one's parents are, one must always love and respect them, (b) one does not have the duty to respect and love parents who have not earned it [we used the proportion of those who chose "a"]; (3) the respondent was asked to endorse one of two statements: (a) it is the parents' duty to do their best for their children, even at the expense of their own well-being, (b) parents have a life of their own and should not be asked to sacrifice their own well-being for the sake of their children [we used the proportion of participants that chose "a"]; (4) respondents were asked whether they lived with their parents [we used the proportion who indicated they did live with their parents]; (5) respondents were asked about their goals in life [we used the proportion of respondents who said one of their goals in life was to make their parents proud]. All proportions were arcsine-square-root transformed and then standardized prior to analysis. All five items were highly interrelated (Cronbach's $\alpha = 0.86$, $n = 72$ countries). All five components were summed to become our measure, *Strength of Family Ties*. Larger values indicate stronger family ties, while smaller values indicate weaker family ties. National values are published in the supplementary material to Fincher and Thornhill (2012).

Using multiple measures of parasite stress, we tested the prediction that, across countries, parasite stress would be correlated positively with the strength of family ties. One such measure we used was the WHO variable *Infectious Disease DALY* (mentioned earlier in this chapter), which is a cross-national measure of morbidity and mortality attributed to 28 different "infectious and parasitic diseases" for the year 2002 (e.g., tuberculosis, measles, leprosy, dengue; WHO 2004). As explained earlier, an important element of the parasite-stress theory is the costs associated with acquiring diseases from out-group humans. Thus, infectious diseases that are transmissible between humans are predicted to be more important in evoking collectivism than human infectious diseases that are not transmitted between humans. We summed the number of cases of human-specific and multihost infectious

diseases per country (called nonzoonotic) and the number of cases of zoonotic diseases, based on data from GIDEON for each disease's severity in each country (see Sect. 5.4). This provided measures of parasite severity for the two disease categories. Nonzoonotic disease was correlated positively with zoonotic disease ($r=0.61$, $p<0.0001$, $n=226$). Nonzoonotic disease was correlated positively with *Infectious Disease DALY* ($r=0.76$, $p<0.0001$, $n=192$). Zoonotic disease was correlated positively with *Infectious Disease DALY* ($r=0.16$, $p=0.03$, $n=192$).

Because there is considerable overlap and covariation in our infectious disease measures, we standardized each of the two variables—*Infectious Disease DALY* and nonzoonotic disease severity—and then summed these standardized scores for each country. Zoonotic disease was not included because of its minimal relationship with collectivism–individualism (see Sect. 5.4 and below). This sum we refer to as *Combined Parasite Stress* (Cronbach's $\alpha=0.76$, $n=192$); national values are published in the supplementary material to Fincher and Thornhill (2012).

5.8.2 Findings

The results were as follows. *Strength of Family Ties* was correlated positively and significantly with each of the two separate parasite-stress variables mentioned just earlier; the correlation coefficients were 0.64 for *Infectious Disease DALY* ($n=69$ countries) and 0.57 for nonzoonotic disease ($n=72$; both p's<0.0001). *Combined Parasite Stress* was correlated similarly with the *Strength of Family Ties* ($r=0.63$, $p<0.0001$, $n=69$). *Strength of Family Ties* and zoonotic disease showed a small and insignificant correlation ($r=0.15$, $p>0.22$, $n=72$).

There are variables other than parasite stress that have been proposed in the literature as explanations of the strength of family ties (e.g., economic development is often assumed to reduce family ties). We examined correlations between the possible confounding factors (described below) and our dependent variable *Strength of Family Ties*. Potentially confounding variables that were correlated significantly ($p\leq0.05$) were then entered into multiple regressions with *Combined Parasite Stress* and *Strength of Family Ties* to determine whether the predicted correlation remained after removing the effect of the potentially confounding variables.

We examined the effects of national wealth (GDP per capita over the years 1960–2008; data obtained from data.worldbank.org) and the equitability of resource distribution within a nation. For the equitability of resource distribution, we used the measure produced by Vanhanen (2003). It combines GDP per capita, percentages of university students and literates, the degree to which land ownership is widespread, and the degree of decentralization of nonagricultural economic resources in a single value. We called this variable *Resource Distribution*. Too, we examined the effects of human freedom (e.g., the freedoms of expression and belief) using the average of cross-national scores of civil liberties from Freedom House for the years 1972–2008 (www.freedomhouse.org). This became our variable *Civil Liberty*. In our regression analyses, we used two model specifications. The most general model contained

Combined Parasite Stress, *Civil Liberty*, and *Resource Distribution* as the predictor variables of *Strength of Family Ties. Resource Distribution* includes GDP per capita; however, because of the large amount of cross-cultural research that focuses on GDP per capita, we tested a second model that used GDP per capita and *Combined Parasite Stress* as the predictor variables. All of these potential confounders have been validated and used widely in prior research (see Thornhill et al. 2009).

While we have identified some potentially confounding factors, there may be others that we have not identified. Because we have proposed that parasite stress is an encompassing causal factor, we determined the residual lifespan score for each country by regressing the average life expectancy at birth (for the year 2008) for both sexes combined (data from data.worldbank.org) on nonzoonotic disease ($r^2 = 0.51$, $p < 0.0001$, $n = 190$). *Infectious Disease DALY* was not included because its calculation by WHO incorporates life expectancy. These residual lifespan scores represent the variation in lifespan expectancy that cannot be explained by parasite stress (i.e., potentially, this variation represents other causal factors besides parasite stress). We then used these residual lifespan scores in correlations with the *Strength of Family Ties* to address the potential of causal factors besides parasite stress to account for international variation in strength of family ties. This analysis of residuals is similar in logic to that mentioned earlier.

Among the focal, potentially confounding variables, all had significant correlations with the *Strength of Family Ties*. All correlations between *Combined Parasite Stress* and the *Strength of Family Ties* remained positive and significant after removing the confounding effects of the potential confounders (std. β coefficient for the effect of *Combined Parasite Stress* on *Strength of Family Ties* was 0.36 when controlling *Civil Liberty* and *Resource Distribution*; the std. β coefficient was 0.34 when controlling GDP per capita; p's < 0.01). Thus, the correlation between parasite-stress and family ties was robust to the effects of freedom or economic development and equivalence as captured by Vanhanen's resource distribution, Freedom House ratings, and GDP per capita.

The correlation between residual lifespan and *Strength of Family Ties* was small and insignificant ($r = 0.06$, $p = 0.64$, $n = 71$). Thus, the variation in life expectancy explained by family ties that was independent of that explained by parasite stress was minimal.

To look for patterns of regional variation that might be inconsistent with the overall pattern across the globe, we used the following approach: we divided the countries into six world regions according to the method devised by Murdock (1949), which is based on geographical proximity and cultural historical contact. Then, we conducted correlations using the mean values for each of the variables for each world region. This six-world-region approach allowed us to characterize a region comprised of multiple countries into a single value (thus deflating sample size). The small sample size of six makes the p-values unreliable, but it does allow us to test whether the correlations remain in the predicted direction after reducing the sample size. We also conducted a nested-effect linear regression model that accounts for the nested design of our analysis. In the cross-national case, *Combined Parasite Stress* was nested within each world region as the independent variable and

used to predict the *Strength of Family Ties*. Our findings are repeated in the world regions. At the world region level, *Combined Parasite Stress* and *Strength of Family Ties* correlated highly and positively, and thus in the predicted direction ($r = 0.94$, $n = 6$). Also, the *Strength of Family Ties* was predicted significantly by *Combined Parasite Stress* when nested within world region ($r^2 = 0.47$, $p < 0.0001$, $n = 69$). Hence, there is no good evidence that the overall global pattern of positive relationship between parasite stress and strength of family ties is not repeated across each world region.

Again, the details of methods and results used in our research on family ties across countries are in Fincher and Thornhill (2012).

5.9 United States: Collectivism and Family Ties

This section gives a brief presentation of the study of collectivism in relation to parasite stress across USA states by Fincher and Thornhill (2012); for details about methods and results, consult that paper.

5.9.1 Methods

In order to investigate family ties in the United States, we used a measure of USA state-level collectivism compiled by Vandello and Cohen (1999), because collectivism includes strong family ties; it also includes preferential assortment with ingroup members outside the extended family (Triandis 1995; Hofstede 2001; Gelfand et al. 2004; Fincher et al. 2008; Thornhill et al. 2009). Vandello and Cohen (1999) provided a validated measure of collectivism across the USA states by standardizing and summing the following items obtained from USA state data archives: percentage of people living alone (reversed), percentage of elderly people (65+) living alone (reversed), percentage of households with grandchildren in them, divorce to marriage ratio (reversed), percentage of people with no religious affiliation (reversed), average percentage voting Libertarian over the four presidential elections during 1980–1992 (reversed), ratio of people carpooling to work to people driving alone, and percentage of self-employed workers (reversed). Values ranged from 31 for Montana (highest individualism) to 91 for Hawaii (highest collectivism). We predicted a positive correlation between parasite stress and Vandello and Cohen's (1999) measure, which we called *Collectivism*.

Furthermore, from the original state data sources, we extracted the components that address most directly family ties (as described by Vandello and Cohen 1999). These were the items percentage of people living alone (reversed), percentage of elderly people (65+) living alone (reversed), and the percentage of households with grandchildren in them. All three items were interrelated (Cronbach's $\alpha = 0.73$, $n = 50$). The three items were summed to become the variable *Strength of Family Ties USA*.

We obtained the annual *Morbidity and Mortality Weekly Report*'s "Summary of Notifiable Diseases, United States" from the Centers for Disease Control (CDC) for the years 1993–2006 (available at http://www.cdc.gov). For each year, we adjusted the number of cases of all infectious diseases tracked by the CDC for that year by the CDC-reported population size for each state. We only included infectious diseases that the CDC had information on from each state. Thus, some diseases that were unreported in some states (meaning that, for some states, there was no information on the disease's presence or absence, not just that there were zero cases reported) were not included in the index. For each state, we determined the average z-score of this population adjusted disease incidence score for all years. This approach was necessary because the infectious diseases tracked by the CDC can vary between years, though there was often great similarity between years. The standardization allowed us to pinpoint a state's position along a parasite gradient relative to the other states. This index of parasite severity, *Parasite Stress USA*, is ecologically validated by the fact that it shows a negative correlation with latitude (-0.45, $n=50$, $p=0.001$; or after removing the latitudinal outliers Alaska and Hawaii, -0.71, $n=48$, $p<0.0001$), as do global measures of parasite stress (Cashdan 2001a; Guernier et al. 2004). Further ecological validation is demonstrated by the negative correlation of *Parasite Stress USA* with the average lifespan expectancy at birth for both sexes in the year 2000 (data collected from www.census.gov): $r=-0.67$, $p<0.0001$, $n=50$). (A similar pattern between infectious disease stress and lifespan expectancy is found in cross-national analyses; see Chap. 8). The list of diseases across years that comprise *Parasite Stress USA* as well as the actual values of the variable for each state is in the supplementary materials published with Fincher and Thornhill (2012).

5.9.2 Results

The results were as follows. Across US states, collectivism and family ties are predicted positively by parasite severity. *Parasite Stress USA* was correlated positively with *Collectivism* ($r=0.60$, $p<0.0001$, $n=50$) and the *Strength of Family Ties USA* ($r=0.46$, $p=0.0007$, $n=50$).

For addressing potentially confounding variables in the USA analysis, we followed a similar approach as in our cross-national analysis described earlier. We examined correlations between *Collectivism* and *Strength of Family Ties USA*, and potentially confounding factors. Variables that were significantly correlated ($p \leq 0.05$) then were entered into partial correlations with *Parasite Stress USA* to examine whether the predicted correlations between parasite stress and the dependent variables remained after partialling the potentially confounding factors. The potentially confounding factors we considered were GDP per capita and Gini. GDP per capita is an average of the values for years 1999–2007 obtained from the Bureau of Economic Analysis (http://www.bea.gov). Gini was measured at the family level for 1999, the last year available at the time for the variable at the US Census Bureau

(http://www.census.gov). Of the two potentially confounding variables, only Gini correlated significantly with *Collectivism* and *Strength of Family Ties USA*. Given these significant zero-order correlations, the effect of Gini was partialled from the correlations between *Parasite Stress USA* and *Collectivism* and between *Parasite Stress USA* and *Strength of Family Ties USA*. After statistically controlling the effect of Gini, *Parasite Stress USA* remained positively correlated with *Collectivism* ($r=0.46$, $p=0.0009$, $n=50$) and *Strength of Family Ties USA* ($r=0.35$, $p=0.014$, $n=50$). Thus, the correlations between parasite stress and family ties or collectivism were not confounded with the effects of economic inequality and development as captured by the Gini index and GDP per capita.

As in the cross-national analysis, we regressed average life expectancy at birth for both sexes combined for the year 2000 (obtained from www.census.gov) on parasite stress. This regression was significant ($r^2=0.45$, $p<0.0001$, $n=50$). The residuals of the regression represent the variation in life expectancy that is not explained by our measure of parasite stress—that is, residual lifespan. The finding of statistically significant covariation between residual lifespan and any of the dependent variables would imply causation other than parasite stress. No such covariation was detectable. Residual lifespan was not correlated significantly with *Collectivism* ($r=0.11$, $p=0.44$, $n=50$) or *Strength of Family Ties USA* ($r=0.22$, $p=0.13$, $n=50$). Therefore, parasite stress accounts for much of the state-level variation in collectivism and family ties as they relate to life expectancy.

For the USA regional analysis, we divided the states into the nine geographic regions used by the Census Bureau and used both approaches as we did for the cross-national analysis described earlier. The correlation between the dependent variables and *Parasite Stress USA* at the regional level all were in the predicted direction (*Collectivism*: $r=0.83$; *Strength of Family Ties USA*: $r=0.51$, $n=9$ for both). The *Strength of Family Ties USA* was predicted significantly by *Parasite Stress USA* when nested within USA region ($r^2=0.34$, $p=0.033$, $n=50$). *Collectivism* was predicted significantly by *Parasite Stress USA* when nested within USA region ($r^2=0.45$, $p=0.0021$, $n=50$). Hence, the overall pattern across the USA states is a positive relationship between parasite severity and each of the two variables, collectivism and family ties, and this relationship is not specific only to certain regions of the USA.

Again, see Fincher and Thornhill (2012) for the details about the methods and results discussed earlier.

Following the publication of Fincher and Thornhill (2012), we explored the potentially different effects of zoonotic and nonzoonotic infectious-disease severities for the USA. Appendix 2 contains the list of USA infectious diseases and their classification into nonzoonotic (multihost and human-specific comprise nonzoonotic) or zoonotic. The classification scheme was based on Smith et al. 2007 and Fincher and Thornhill 2012, or on our additional research when a disease was not listed in either of those sources. Appendix 3 contains the severity scores for each USA state. Nonzoonotic infectious diseases were correlated positively with *Collectivism* ($r=0.53$, $p<0.0001$, $n=50$), and so were zoonotic infectious diseases, but much less and with marginal significance ($r=0.32$, $p=0.03$, $n=50$). Nonzoonotic

infectious diseases were correlated positively with the *Strength of Family Ties USA* ($r=0.47$, $p=0.0005$, $n=50$), and so were zoonotic infectious diseases, but much less so and not significantly ($r=0.23$, $p=0.11$, $n=50$). Additional regression analyses showed that nonzoonotic diseases were strongly predictive of *Collectivism* and *Strength of Family Ties USA* in comparison to zoonotic diseases: when predicting *Collectivism* (nonzoonotic std. $\beta=0.50$, $p=0.0001$; zoonotic std. $\beta=0.24$, $p=0.05$), when predicting *Strength of Family Ties USA* (nonzoonotic std. $\beta=0.45$, $p=0.0009$; zoonotic std. $\beta=0.16$, $p=0.21$). Thus, just as we found when comparing nations, across the USA states nonzoonotic infectious diseases are more paramount for explaining collectivism and the strength of family ties than are zoonotic infectious diseases.

We also conducted regression analyses to examine the unique predictive effects of each of the indices of transmission types for the USA states (zoonotic, multihost, and human-specific). For *Collectivism*, only human-specific infectious diseases were significantly associated (zoonotic std. $\beta=0.20$, $p=0.10$; multihost std. $\beta=0.04$, $p=0.75$; human-specific std. $\beta=0.53$, $p=0.0001$). For the *Strength of Family Ties USA*, only human-specific infectious diseases were significantly associated (zoonotic std. $\beta=0.16$, $p=0.23$; multihost std. $\beta=0.21$, $p=0.13$; human-specific std. $\beta=0.34$, $p=0.02$). Thus, for both dependent variables, human-specific diseases have a greater effect than either of the other two disease transmission categories. Unlike the cross-national comparisons, multihost diseases, considered alone, were not predictive of collectivism and the strength of family ties within the USA states.

5.10 Summary: Cross-National and USA States

In summary, our cross-national analysis showed that the strength of family ties was correlated positively with parasite stress. As predicted also, the cross-national analysis showed that the strength of family ties was correlated more strongly with nonzoonotic infectious diseases than with zoonotic infectious diseases. Within the United States, the strength of family ties and collectivism were correlated positively with parasite stress and more strongly with nonzoonotic infectious diseases than with zoonotic infectious diseases. The potential confounds examined did not change these conclusions. The patterns appear robust at regional levels both cross-nationally and across the USA.

5.11 Minkov (2011)

Minkov (2011) recently proposed a new measure of collectivism–individualism across countries that is called exclusionism–universalism, with high values equaling high exclusionism (collectivism) and low universalism (individualism). He offered it as a new collectivism–individualism variable to be added to three traditional

measures we discussed at the beginning of this chapter (*Hofstede Individualism, Suh Individualism, Gelfand In-group Collectivism*). Exclusionism is characterized by strong social ties of relatives between generations and within groups of relatives, as well as a low value placed on the interests and well-being of strangers. In contrast, universalism is weak ties of relatives between generations and within groups of relatives, as well as a high degree of interest in the needs and welfare of strangers. Given the composition of Minkov's variable, it is a standard collectivism–individualism metric. Indeed, it is highly correlated with the three traditional measures mentioned from an r of −0.71 with *Hofstede*'s *Individualism* to an r of 0.81 with *Gelfand In-group Collectivism.* Importantly, exclusionism–universalism correlates strongly with *Combined Parasite Stress* ($r = 0.72$, $p < 0.0001$, $n = 86$ countries).

5.12 Cashdan and Steele (2013): The Standard Cross-Cultural Sample

Cashdan and Steele (2013) have conducted an important first study of collectivism–individualism in relation to parasite severity in the 186 indigenous societies comprising the Standard Cross-Cultural Sample. They used a measure of these values based on how children are inculcated across the societies as reported by the ethnographers working in the societies. In this sample, child training ranges from high obedience to parents and other authorities (collectivist inculcation) to high self-reliance (individualist inculcation). They used a parasite severity measure similar to that used by Low (1988) for the same societies. Their results supported the parasite-stress theory of values: in societies experiencing high parasite stress, children are taught obedience whereas in low parasite stress conditions children are taught self-reliance.

5.13 Convergence of Evidence

So far in this chapter, we have provided convergent evidence that parasite stress directly predicts collectivist values across countries, USA states, and societies in the Standard Cross-Cultural Sample, or, said differently, parasite stress negatively predicts individualism across all three of these levels of analysis. The findings cross-nationally and across the USA that strong family ties are correlated with parasite stress complements our earlier work, discussed earlier, that linked collectivism–individualism with parasite stress. Our finding of the strong positive correlation between Vandello and Cohen's measure of collectivism and parasite stress within the USA is also an important complement to the cross-national findings of the same relationship. Finally, the findings of Cashdan and Steele (2013) showing that collectivism is positively related to parasite stress across indigenous societies complements all these other sources of evidence for the relationship between collectivism–individualism and parasite adversity.

5.14 Collectivism and Family Ties: Other Implications

5.14.1 Life History

All the findings presented earlier are major cross-cultural extensions to the ethnographic research on indigenous societies that has documented the important role of nepotistic and other in-group altruistic connections and support for surviving parasitic infections (discussed in Chap. 3) and conducted by Gurven et al. (2000), Sugiyama and Sugiyama (2003), Sugiyama (2004), and Hill and Hurtado (2009). However, whenever parasite stress is extremely high, collectivistic, including nepotistic, investment may not be optimal because the extreme parasite stress yields extrinsic mortality (Quinlan 2007 and references therein). In the area of evolutionary theory dealing with life history patterns, extrinsic mortality is mortality independent of individuals' efforts to combat it. Because extrinsic mortality, by definition, cannot be reduced by collectivist social investment, comparatively low levels of investment are expected from life history theory when extrinsic mortality is high. Therefore, we expect reduced collectivist investment in offspring, other kin, and the in-group in general in the face of extremely high parasite stress. In this setting, as in other contexts of high extrinsic mortality, early reproduction with minimum nepotistic investment per family member (e.g., offspring) is expected from life history theory (Charnov 1993; Kaplan and Gangestad 2005). Consistent with this thinking, Quinlan (2007) found in a sample of traditional societies that maternal investment in the form of nursing duration increased along with pathogen stress, but then began to decrease after pathogen stress became extreme. We reasoned that this same pattern would be seen in human value systems as well.

Supporting this reasoning, when focusing on Murdock's six world regions, we reported in Fincher and Thornhill (2012) that the correlation in Africa between *Combined Parasite Stress* and a variable we called *In-group Assortativeness* was negative (–0.31), instead of positive as in the other five world regions. *In-group Assortativeness* is a combination variable that we have constructed and explored cross-nationally in Fincher and Thornhill (2012). It combines strength of family ties and religiosity and is a validated measure of embeddedness in the in-group and in-group favoritism. We will discuss this variable further when we treat the relationship between religiosity and parasite stress (Chap. 9). The exceptional case of Africa mentioned might be explained by the fact that parasite stress is exceptionally high in Africa—and therefore generally yields extrinsic mortality—as compared to the five other world regions. (A *post-hoc* Tukey HSD means test showed Africa to be distinctly high in parasite stress: Africa, mean (M) = 3.36, A (world areas not followed by the same letter are significantly different); South America, $M = 0.85$, B; East Eurasia, $M = 0.53$, BC; North America, $M = -0.51$, BC; Insular Pacific, $M = -0.65$, C; West Eurasia, $M = -2.28$, D.) Finding the different pattern in Africa shows the importance of regional analysis to identify patterns that may be contrary to worldwide relationships of variables and masked without regional analytical follow-up.

In the final chapter of this book we return to the idea of extremely high parasite stress as extrinsic mortality and provide additional evidence that under very high parasite stress collectivism shows the predicted decline.

5.14.2 Implications for Future Regional Analyses

The history of infectious-disease reduction in the USA and other Western regions is reviewed in Thornhill et al. (2009) and involved a multitude of factors varying from antibiotic availability, vaccination programs, chlorinated and fluoridated public drinking water availability, vector control, food handling regulations, and other sanitation changes and technology. As we discuss more fully in Chap. 10, we have argued that the reduction in parasite stress, beginning in the early to mid-1900s (depending on the particular health intervention), accounts for the cultural revolution in the huge increase in liberalization of values in the 1960s and 1970s in the West—the sexual revolution, antiauthoritarianism, women's rights, gay rights, changes in divorce law, civil rights, and so on (Thornhill et al. 2009). Although these health improvements had the overall effect of reducing parasitic infections across the USA (and the West in general) and associated morbidity and mortality, the degree of their effect remains variable across the USA, as seen in the data we presented earlier on the power of parasite-stress variation for predicting variation in values across the states of the USA.

It would be relevant to explore regional analysis within other countries that contain significant parasite gradients. For example, Japan's northern island of Hokkaido rivals the high individualism in the United States (Kitayama et al. 2006); likely, Hokkaido has a much lower level of parasite stress than does southern Japan, given the negative covariation of parasite stress and latitude (Guernier et al. 2004). Also, in China, historically, much of China's innovation came from the northern side, which was much lower in parasite adversity than the southern portion below the Yellow River (McNeill 1998). Innovation—both its generation and the willingness of people to adopt it—corresponds to individualistic values (Thornhill et al. 2009, Chap. 11). The regional development of innovation in China and elsewhere could be studied more thoroughly in its relation to pathogen stress and associated evoked value systems. In the USA, we found significant regional variation in values in spite of generally low parasite adversity in the country. We expect this pattern to be repeated within nations across the world in cases in which a nation contains a parasite gradient.

5.14.3 The Demographic Transition

One aspect of family life that has been studied often is the demographic transition from large families to smaller families. One of the more convincing explanations for this phenomenon comes from Newson et al. (2005), who argue that the demographic

transition arose from an increase over time in the ratio of nonkin to kin in individuals' social networks. We agree, but offer a reinterpretation of the meaning of the demographic transition. Based on our studies on collectivism and family ties discussed earlier, it is reasonable to assume that this increase in the non-kin-to-kin ratio is related to a decrease in parasite stress over time and corresponding increase in individualism and associated nuclear family focus. Moreover, as predicted by the parasite-stress theory of values, those countries where the demographic transition has occurred are the same ones that have experienced a relatively recent emancipation from infectious disease (Thornhill et al. 2009).

5.15 Philopatry

Philopatry—the preference for remaining in the natal location for reproduction (absence of dispersal)—reduces contact with out-groups and their habitats that may contain new parasites to which the in-group has no or limited immunity. Or, said differently, dispersal has the potential cost of contacting infectious diseases that could be avoided by remaining philopatric. In areas of high pathogen stress, compared with those of low pathogen stress, limited dispersal will be favored by natural selection and attractive for cultural adoption by individuals owing to the corresponding advantage of increased association with immunologically similar individuals and decreased contact with more distant, and differently parasitized, other individuals. Freeland (1976, 1979), Møller et al. (1993), and Loehle (1995) all discuss how limited dispersal in nonhuman animals reduces exposure to a diversity of types of infectious diseases and argue for the importance of territoriality and restricted home ranges, forms of limited dispersal, as adaptive means for reducing contact with dissimilar conspecifics that may carry novel diseases.

In humans, philopatry keeps people near to their natal locale and social community, and hence contributes to collectivism, ethnocentrism, and in-group assortative sociality in general. Alesina and Giuliano (2010) demonstrated that, across countries, adults who are more embedded in their extended family (and demonstrably more collectivist) disperse for shorter distances from their natal locale and are, hence, more philopatric than are less embedded adults (individualists). They reported that the relatively limited dispersal of collectivists was apparent both in their adult presence in the natal region and in adult residence in their natal house. The research outside of humans, mentioned earlier, as well as that of Alesina and Giuliano on people, supports the hypothesis that infectious diseases cause values or preferences pertaining to dispersal behavior—where infectious disease is more stressful, animals, including humans, disperse over shorter distances than where infectious disease is less stressful. The human research indicates that high philopatry is a core value of collectivists and low philopatry is a core value of individualists.

Below we present research on human movement patterns in relation to parasite stress. First, we summarize our research findings on movement patterns in ethnographic societies reported in Fincher and Thornhill (2008b). We then examine the pattern of interstate residential movement across states of the USA.

5.15.1 Ethnographic Societies

5.15.1.1 Methods

According to the parasite-stress theory of values, the area of land that individuals inhabit should correlate negatively with pathogen stress, reflecting limited dispersal in the face of high contagion risk associated with contact with out-groups and their habitats. In order to further test this proposition's application to human movement, we examined the relationship between Binford's (2001) measure of societal range size, reflecting the aggregate use of space by individuals within a society, for his large sample of 339 traditional societies across the globe, and an estimate of pathogen stress for each of these societies (Fincher and Thornhill 2008b). Binford's measures are based on his estimates taken from ethnographic sources. We estimated pathogen stress by first establishing the linear equation that best predicted parasite severity (the number of parasitic disease cases), as measured by Low (1994), for the 186 societies in the Standard Cross-Cultural Sample (SCCS, Murdock and White 1969). We examined absolute latitude, mean annual temperature, and mean yearly rainfall as predictor variables from data provided by Binford (2001) for each of the 339 societies in his sample. The data for the SCCS were collected from the *World Cultures* journal website (www.worldcultures.org). The best single predictor of parasite severity was absolute latitude ($r = -0.51$, $p < 0.0001$). Both temperature and rainfall significantly predicted parasite severity, but to lower degrees than absolute latitude; therefore, we generated a linear equation to predict parasite severity based on absolute latitude. Then, we used this equation to estimate parasite severity for each of the 339 societies from data on the latitudinal location of each society in Binford (2001).

Societies with more individuals also might have larger home ranges; thus, we examined the potentially confounding effect of population size using data provided by Binford (2001). Also, societal range size might be related positively to reliance on hunting of terrestrial animals for subsistence; thus, we examined the potentially confounding effect of the proportion of subsistence derived from hunting of terrestrial animals, as provided by Binford (2001).

Additionally, we considered the patterns of mobility of each society with respect to estimated parasite severity because high levels of infection and associated lethargy and incapacitation may reduce mobility of people, generating a negative relationship between societal range size and parasite adversity irrespective of philopatric values serving to minimize dispersal. We did this by analyzing the average distance moved annually and the average annual number of moves (1 was added to the number of moves prior to ln-transformation to eliminate negative values).

Lastly, we used an analysis of variance with country in which a traditional society was located, as provided in Binford (2001), as the independent variable predicting societal range size to examine the possibility of regional dissimilarity in results. This method is similar in logic to our use of world regions, cultural regions, and USA census regions in other analyses we have discussed earlier in this chapter. (All values analyzed for each of the 339 societies are contained in Fincher and Thornhill 2008b.)

5.15.1.2 Findings

We found that societal range size was negatively and significantly correlated with parasite severity ($r=-0.48$, $p<0.0001$, $n=339$). After partialling the effect of population size or the proportion of subsistence from hunting, the correlation between range size and parasite severity remained negative and significant (partialling population size: $r=-0.42$; partialling proportion hunting: $r=-0.38$; $p<0.0001$, $n=339$ for each correlation). Removing the effects of population size and the proportion of subsistence from hunting simultaneously, the correlation between parasite severity and home range size was negative and significant ($r=-0.27$, $p<0.0001$, $n=339$). Thus, as predicted by the parasite-stress theory, there was a robust reduction in range size and associated limitation of dispersal in areas with greater pathogen severity compared to societies residing under reduced parasite severity.

In general, a society's range size was predicted by the country in which the society occurred ($p<0.0001$). Thus, we computed the average value of home range size, parasite severity, population size, and the proportion of subsistence from hunting for the cultures of each country and conducted correlations with these composite values. At the country level, the correlation between home range size and parasite severity was $r=-0.60$ ($p<0.0001$, $n=67$ countries). After partialling the effects of population size and proportion of subsistence from hunting, the correlation between home range size and parasite severity at the country level was $r=-0.43$ ($p<0.0004$, $n=67$). Considered separately, partialling the effect of proportion of subsistence from hunting and the effect of population size, the correlation between home range size and parasite severity was $r=-0.51$ ($p<0.0001$; $n=67$) and $r=-0.52$ ($p<0.0001$; $n=67$), respectively.

Considering mobility patterns within home ranges, the number of moves (ln) annually was positively correlated ($r=0.12$, $p<0.03$), while the distance moved annually was negatively correlated ($r=-0.21$, $p<0.0001$) with parasite severity ($n=339$ for both). Moreover, partialling the effect of distance moved increases the correlation between the number of moves and parasite severity to 0.45 ($p<0.0001$), while partialling the effect of number of moves increases the correlation between parasite severity and distance moved to $r=-0.47$ ($p=0.0001$). Thus, people in traditional societies in high parasite-stress areas move more often, but over shorter distances. Conversely, in areas with lower parasite stress, they move less often but over longer distances.

5.15.1.3 Discussion and Conclusions

The main finding in Fincher and Thornhill (2008b) was that range sizes for ethnographic societies are smaller in areas of the world where parasite severity was more intense. Moreover, this relationship is not confounded by population size or the proportion of subsistence from hunting. This finding is consistent with the notion that human societal range sizes, as well as species' range sizes, are generally reduced in the tropics. Ecologists call this pattern for species' ranges "Rapoport's

rule" (Stevens 1989). Our research gives this ecological rule a causal explanation, which it has lacked, that is supported by the increasing evidence that parasite adversity affects dispersal behavior. We have hypothesized that this general pattern results from the evolved response of limited dispersal in tropical regions and other high pathogen areas owing to strong selection against out-group contact (Fincher and Thornhill 2008a). We discuss Rapoport's rule further in Chap. 13.

According to the data, individuals in ethnographic societies in areas with high pathogen stress move often, but over shorter distances. These findings certainly negate the common-sense notion that limited societal range size is due primarily to incapacitation, lethargy, and physical inability to move due to a heavy infectious-disease burden. We have hypothesized that this pattern of short, but frequent movement of peoples in high parasite areas is also an aspect of evolved antipathogen behavior (Fincher and Thornhill 2008b). Individuals within these societies may move strategically often within a restricted territory to optimally distance themselves from parasites, especially intestinal parasites, that persist in the soil (McNeill 1981; Loehle 1995). Freeland (1976) discusses similar localized movements of ungulates that seem to function this way.

Cashdan and Steele's (2013) study on collectivism–individualism in the 186 societies in the Standard Cross-Cultural Sample (discussed above) included an analysis of a variable measuring adult movement of residence between communities. They reported that, in indigenous peoples, parasite stress negatively predicts this movement. Hence, in comparison to people living in low parasite-stress locales, people in high parasite-stress locales have smaller home ranges and lower intercommunity residential movement, as expected from the parasite-stress theory of values.

5.15.2 Residential Movement Between USA States

Based on the hypothesis that higher levels of parasite stress will evoke philopatry, we predicted that people in the USA will move from one state to another to establish a new residence less often in areas of high parasite stress than in areas of low parasite stress. We tested this prediction in a study first reported here. We collected migration data from the Census Bureau for the year 2005 (source: http://www.census.gov/hhes/migration/data/acs/state-to-state.html). We chose 2005 as a point prior to the economic upheaval in 2008. Our measure is the number of people leaving a given state (emigrants) within the year 2005 to establish residence in any other state within the USA. Because of outlying data in the samples, we used Spearman's correlations to examine the relationships between variables. Unsurprisingly, the number of emigrants was correlated positively with the state's population size for year 2005 ($\rho = 0.97$, $p < 0.0001$, $n = 50$) (source for population size: http://www.census.gov/popest/data/historical/2000s/vintage_2005/index.html). Therefore, we calculated an *Emigration* variable for each state that is the number of emigrants from the state divided by the state's population size. As predicted, the correlation between *Parasite Stress USA* (introduced in Sect. 5.9.1) and *Emigration* was significantly

negative ($\rho = -0.31$, $p = 0.03$, $n = 50$ states). This means that there were relatively fewer residential emigration events from states that have high parasite stress; conversely, there were more emigrations from states that have lower parasite stress. There are other potential explanations of emigration rate. One particularly cognizant one is the average wealth within a state. We collected data on GDP per capita for the year 2005 from the Bureau of Economic Analysis (source: www.bea.gov). State-level GDP per capita was unimportant for explaining *Emigration* ($\rho = -0.01$, $p = 0.95$, $n = 50$). Thus, we have here concordant evidence across USA states that philopatry is more prevalent in areas with high parasite adversity than in areas of low parasite adversity. We add, however, that the relationship between Vandello and Cohen's (1999) measure of collectivism and *Emigration* is in the predicted direction, but statistically insignificant ($\rho = -0.19$, $p = 0.18$).

5.15.3 Overview: Philopatry

In sum, we hypothesized that reduced dispersal (high philopatry) reflects adaptation to reduce contact with novel parasites, and that increased dispersal is the optimal preference under low parasite stress. Various lines of evidence support this hypothesis. Across nations, collectivist people are more philopatric than individualists, and correspondingly collectivists more often live in areas of higher parasite severity than do individualists. Across indigenous societies, range size is related negatively to parasite stress. People in indigenous societies move a lot under high parasite stress, but not very far. Furthermore, interstate movement of residence by people in the USA is reduced in states with high parasite adversity compared to states with low parasite adversity. A similar pattern is seen in the small-scale societies in the Standard Cross-Cultural sample, but involved reduced intercommunity residential movement under high parasite stress. A pattern mentioned in Chap. 4 is also relevant to the difference in philopatry of collectivists versus individualists. Liberals are more interested in and engage in more travel from their home region. This is seen from scores of degree of conservatism/liberalism of individuals (Carney et al. 2008). Finally, the parasite-stress theory of movement gives new meaning to an important general rule of ecological science, Rapoport's rule.

5.16 Collectivism, Family Ties, and Cooperative Breeding

Collectivism and associated strong family ties are not restricted to humans; indeed, they appear to be widespread across animal taxa. We have argued that parasite adversity was one of the main forces of selection responsible for adaptation that functions in extended family investment. Thus, we proposed that variation in parasite prevalence is a cause of the large variation across animal species in the degree of extended nepotism exhibited outside the social unit of parent(s) and offspring (Fincher and Thornhill 2012).

The literature on the evolution of family life is voluminous, with important reviews provided by Andersson (1984), Brockmann (1984), and Emlen (1994, 1995, 1997). The study of family life first became fully encompassed by evolutionary biology with Hamilton's realization that an individual's fitness is more than its phenotypic design for production of descendant relatives—that is, more than the individual's classical or Darwinian fitness (Chap. 2). Indeed, one's inclusive fitness is the classical component plus design for assisting nondescendant kin by nepotism and thereby promoting one's reproduction. However, current inclusive fitness theory, the fundamental component of modern evolutionary social theory, does not account for why nepotism is variable across social systems. Why is nepotism limited to the nuclear family in many systems, but extended beyond the nuclear family in others to entail varying degrees of cooperative breeding? In this chapter, we provide considerable evidence that parasite stress explains this variation across human social systems. Family ties, or collectivism, measures the investment in the extended family and hence measures cooperative breeding. We proposed in Fincher and Thornhill (2012) that a general theory of family life across taxa is accomplished by coupling the parasite-stress theory of sociality with Hamilton's theory.

The social organization of animal species varies along a cooperative breeding continuum, or said differently, a continuum of eusociality (Andersson 1984; Sherman et al. 1995). A mother alone investing in her offspring or, much less commonly across species, a father alone investing in offspring, is on the highest asociality end of the continuum. Species in which both mother and father nepotistically assist the offspring (so-called biparental species) are more social in degree on the continuum. This is followed by varying degrees of nepotism extended outside parental care (i.e., extra-parental nepotism). Sterility, shown by adult members of the group who serve as helpers and thereby assist relatives of varying degrees or in some cases nonrelatives instead of producing their own offspring, occurs in certain taxa of vertebrates, including the human species, and certain taxa of invertebrates. Depending on the species, this sterility ranges from temporary to permanent. The temporary case is delayed striving to produce descendent kin while, instead, engaging in extra-parental nepotism and other in-group altruism (e.g., certain human groups (Hill and Hurtado 2009) and certain species of wasps, birds, and carnivores). The permanent case is lifelong exclusive extra-parental nepotism (as is characteristic of worker and soldier castes in ants and termites). Both temporary and permanent cases are cooperative breeding, a feature of in-group assortative sociality. Also, the relatively eusocial species on the continuum—i.e., the more cooperative in terms of group breeding—exhibit, in general, strong sedentism, delayed or no dispersal from the natal location, and territory defense by the family group or in some cases by the larger in-group (e.g., Arnold and Owens 1998). According to the parasite-stress theory, the sedentism and limited dispersal are analogs (similarity resulting from independent evolution by selection, i.e., convergent evolution) or in some cases homologs (similarity resulting from common ancestry) of human philopatry. The territoriality is the analog or homolog, depending on the comparison, of human xenophobia.

At a minimum, our conjecture is supported by the fact that cooperative breeding in birds and eusocial systems in insects are more common, or in the case of

eusociality, more eusocial, in tropical regions for many different taxa (e.g., birds (Brown 1987; Ekman 2006; Blumstein and Møller 2008), wasps (Wilson 1971)). The incidence of cooperative breeding in birds has been shown to correlate positively with temporal variation in certain climatic factors, especially rainfall (Jetz and Rubenstein 2011). It is not known, however, whether parasite stress in birds also corresponds to temporal variation in the factors Jetz and Rubenstein studied, but it is established that climatic factors correlate strongly with human infectious disease adversity (e.g., Dunn et al. 2010). Data that would allow a comparison of parasite stress in cooperative and noncooperative breeding nonhuman vertebrates are unavailable. A test of our hypothesis that cooperative breeding is favored by selection under high parasite adversity would include measures of allocation to the immune system between the two types of social systems. Møller (1998) reported that tropical bird species show greater immune-system allocation than do temperate bird species, which is expected on the basis of greater parasite adversity in the tropics. The prediction that cooperative breeders will invest more in immune defense than closely related species that breed in pairs (noncooperative breeders) was supported by a study of 66 species of African birds, of which 18 were cooperative breeders (Spottiswoode 2008). Similar comparisons could be conducted on cooperative-breeding species versus non-cooperative-breeding species in other taxa containing cooperative breeders.

5.17 Reciprocity

In this and the previous chapter, we have seen that, for humans, evidence strongly supports the following: in comparison to individualists, collectivists are less widely egalitarian, favor in-group over out-group, dislike and avoid out-groups, define sharp and permanent in- versus out-group boundaries, and are less motivated to help strangers. As degree of collectivism increases across human cultural groups, so do each of these features of in-group assortative sociality. The ethnocentrism component of in-group assortative sociality involves more than just investment in the nuclear and extended family. It extends to nonrelated others with like values and hence, in human evolutionary history, to other in-group people with similar immunity. As explained in Chap. 3's discussion of the basic components of the parasite-stress theory of values, the ethnocentrism functionally is for both avoidance and management of infectious diseases. The management part of ethnocentrism is designed to produce and maintain socially supportive networks, based on nepotism and pure reciprocity (reciprocity without a kinship component), with other people of similar values and norms, which are therefore safe for social interaction from the standpoint of reduced risk of infection by a novel parasite. This network was the only insurance against the morbidity and mortality resulting from infectious disease in evolutionary ancestral times of the hominin lineage. Hence, the quality and reliability of this support network affected differential inclusive reproductive success of individuals. Those individuals with high quality and reliable networks

out-reproduced those without such networks, and hence became modern human's evolutionary ancestors.

In altruistic social interactions, as the coefficient of relatedness between benefactor and recipient declines, nepotistic behavior becomes increasingly similar to pure reciprocity—the altruist's return benefits affecting the adaptiveness of altruism increasingly depend upon resources returned, rather than the return arising from the recipient's enhanced reproductive success that results from the altruism. Hence, nepotism grades into pure reciprocity; this graded social life was part of the social evolutionary legacy of people, even in the kin-based groups of humans' deep-time background.

We have emphasized that human values are conditionally adopted and used in social navigation. In comparison to other species, *Homo sapiens* is adapted to unique degrees to conditionally interact and ally with people in distinct out-groups with dissimilar phenotypes, including dissimilarity in values, behavior, and appearance. The important condition making this xenophilia adaptive, according to the parasite-stress theory, is low parasite stress.

As explained in Chap. 3, parasite adversity is variable on the localized spatial and temporal scale. Hence, in any given locale, within their lifetime, individuals face varying amounts of parasite stress. We suggest that, in human evolutionary history, it was the regularity of relatively low parasite-stress conditions, in which out-group interactions, transactions, and alliances were adaptive, that crafted human psychological adaptation functioning in altruistic reciprocation among nonrelatives, both in direct and indirect reciprocity (also see Thornhill et al. 2009). As explained in Chap. 2, altruism among individuals unrelated by recent common descent is favored by selection when the altruist receives return benefits from another or others that exceed the costs of the altruism. We propose that, in human evolutionary history, this condition was met consistently when contagion risk associated with inter-group contact was relatively low.

The structure seen in indigenous foraging societies/hunter–gatherers—the social organization that characterized human evolutionary history—is that individuals are surrounded primarily by close and distant genetic relatives. In addition, a minority of the group members comes from the outside through marriage, capture, or other sources of immigration (e.g., Van den Berghe 1981; Low 2000). Although the composition of the group in human evolutionary history included very distant relatives and some nonrelatives, giving context for natural selection favoring reciprocity in the broader social network, we propose that an important context for the evolution of reciprocity was in gaining benefits from out-group interactions during periods of relatively low disease threat. Thus, the parasite-stress theory of values offers a novel perspective to explain the evolution of human reciprocal altruism. Accordingly, natural selection is expected to have designed our reciprocity activities to be conditionally sensitive to the variable risk of contagion in the local ecological setting. Under low such risk, reciprocity is more beneficial than under high risk.

Generally consistent with this reasoning is the favorable attitude of liberals versus the unfavorable attitude of conservatives about out-group transactions. Also consistent is the experimental evidence we have discussed of a within-individual

shift to values of interpersonal avoidance upon encountering cues of parasite threat in the immediate environment. Finally, in Chap. 11 we take up topics central to understanding the variation in patterns of diffusion of innovations. Such diffusion relies on willingness to interact with out-groups and hence, we argue, is reflective of reciprocity psychological adaptation designed by an evolutionary history of variable parasite stress locally.

5.18 Human-Specific Cognitive Ability

The parasite-stress theory of sociality, moreover, provides a new hypothesis for the uniquely sophisticated cognitive ability of humans. The hypothesis that natural selection, in the context of interactions with conspecifics, was a major evolutionary force responsible for increased brain size and the concomitant increased intelligence and associated sophistication of social behavior in various bird and mammalian taxa, including primates—the "social brain hypothesis"—is well supported (see recent overviews by Dunbar and Shultz 2007; Shultz and Dunbar 2007). A version of this idea applied to the cognitive abilities of humans was discussed by Alexander (1987, 1990) and Flinn et al. (2005). Alexander argued that, as we became ecologically dominant during human evolution as a result of the evolved psychological capacity for inventing weapons and other technology, the most important selective agents were not "… climate, weather, food shortages, or parasites—not even predators" (Alexander 1990, p. 4). Rather, he proposed that, as we gained relative freedom (compared to other species) from these forms of mortality, other humans became the greatest force of Darwinian selection. This led to runaway social selection in the human lineage, generating many aspects of human mental uniqueness, such as consciousness, theory of mind, creativity, exquisite linguistic and deceptive abilities, reputation building, and many others (Flinn et al. 2005).

We specifically take issue with Alexander's (1990) notion that our ecological dominance freed us from the importance of infectious diseases as agents of selection. We suggest a different scenario: as our physical environmental problems and predators became less important as mortality agents, parasites became more important as agents of selection. Indeed, only since the inventions of vaccines, antibiotics and modern sanitation have humans achieved any significant dominance over parasites. Also, such dominance is seen primarily only in the West. As discussed in Chap. 3, in many geographic areas infectious disease appears to be the leading factor bringing about natural selection on contemporary humans, and infectious disease is likely the leading cause of juvenile mortality in indigenous peoples in the ethnographic record. Even now in the USA, a relatively low parasite-stress country, much of the variation in human lifespan may result from parasitic disease (see above and Chap. 8). Moreover, we suggest that it was the salience of the variability of local parasite stress that accounts for the runaway social selection in the human lineage and thus for important aspects of human uniqueness in cognitive ability.

This relative increase in the importance of parasites versus other mortality and morbidity factors for a species may mark a novel event in the entire evolution of the diversity of life. If we are correct about collectivism–individualism and related values being causally related to pathogen stress, then human ecological dominance (relative freedom from predators and the physical environment, but of increased importance of parasites) would lead to the same social features considered important in Alexander's (1990) hypothesis: intensive and extensive nepotism, male kin and nonkin coalitions, male philopatry, pervasive intergroup and intragroup conflict, raiding, war, complex reciprocity, and the like. Thus, much of human uniqueness may arise from our ecological dominance generating runaway social selection in concert with the avoidance and management of parasites. Flinn et al. (2005) provided evidence that the parts of the brain evolutionarily enlarged in humans, and which account for the brain-size differences between chimpanzees and humans, function in human social life. We agree with Flinn et al.'s (2005) emphasis on coevolutionary, antagonistic social races in the human lineage. We add that these races were fueled by variable parasite stresses that gave rise to adaptive variation through time and space in the use of collectivist and individualist tactics in defining and delimiting social networks and in-group and out-group interactions. It is the variation in parasite stress in a locale that gives rise to the social complexity and difficulty involved in adaptive use of these tactics in social interactions. In essence, the unique cognitive abilities of *Homo sapiens* may reflect, in large part, mental and associated behavioral adaptations that evolved in the context of the relatively greater selection from pathogen stresses in humans compared to other species, and it was these abilities, we argue, that provided adaptive solutions to the complex and contingently optimal social decisions arising from variable pathogen stresses.

Thus, we disagree, in part, with a major, prior theory for the evolution of unique features of human cognition. We agree that social coevolutionary races were salient; therefore, what accounts for the comparatively lofty mental capabilities of humans is past selection in dealing with conspecifics. Parasite-stress variation, however, is central to adopting adaptive behavioral contingencies for social behavior. We propose that the context of spatially and temporally variable parasite stress and associated in-group and out-group behavioral solutions to the variation was a chief adaptive problem that ultimately created human abilities in intensive and extensive nepotism, complex reciprocity involving distant relatives and unrelated others, and the psychology involved in intergroup interactions, antagonisms, and alliances.

Anders Møller, whose research contributions extend across so many areas of biology, including parasitology, proposed recently a hypothesis that is relevant to our hypothesis of the relatively greater impact of parasites as selection agents in human evolution than in other lineages. After summarizing numerous research findings that predators differentially kill and eat parasitized prey, compared to their depredation of healthy prey items, he proposed that as predator abundance declines, parasites evolve higher virulence (pathogenicity) (Møller 2008). Møller's reasoning was that, under elevated natural selection on a host species from predators, the greater predation on parasitized prey would select for reduced pathogenicity in the parasites. This is because parasites with reduced virulence would be less likely to

die before transmission to a new host as a result of their host being depredated. If this is correct, then the relative emancipation of human evolutionary ancestors from predators resulting from their weaponry and other technology contributed to the relatively greater impact of parasite adversity in human evolution through increased parasite virulence (A. Møller, personal communication, August 5, 2010).

Hence, the great reduction in impact of many typical sources of Darwinian selection, including predation, on human ancestral populations makes the human lineage unique, compared to other lineages of living things, in terms of the elevated impact of parasites as agents of selection. Also, the decline in the relative importance of predation in human ancestral generations specifically may have made the parasites of humans more virulent, compared to parasites of other species with higher rates of predation. These factors combined lend theoretical support to our hypothesis that parasites were fundamentally salient as causes in evolutionary history of human sociality and may account for many of the unique aspects of human social life, intellectual capacity, and behavioral immunity.

Parasites, besides perhaps providing the Darwinian selection that created human-unique mental capacity, appear to be important in another aspect of human mental life. The large brains and human-specific lofty mental capabilities of *Homo sapiens* are very energetically costly to produce and maintain during their ontogeny. These costs, we have argued, result in a negative ontogenetic interaction between cognitive ability and classical immunity. Parasitic infections result in a greater allocation to the classical immune system, and hence limit the energy available for cognitive development. These infections also reduce energy availability more directly. In research with Chris Eppig, we have shown that, both cross-nationally and across the states of the USA, parasite stress is strongly, negatively related to cognitive ability (IQ) (Eppig et al. 2010, 2011; Chap. 11). This, we argue, helps to explain why conservatives have lower cognitive ability than liberals and why collectivist cultures, relative to individualist ones, are more economically depressed (see Chaps. 4 and 11). Although parasites ultimately made humankind's large brains, these brains are susceptible to indirect degradation by parasites on a proximate developmental timescale.

5.19 Patriotism

We compiled a cross-national measure we call *Strength of National Ties* from the World Values Survey. (See Fincher and Thornhill 2012 for its composition and data within the supplementary materials.) This measure taps into the value placed on an individual for adopting the customs, being born in, and having ancestors from a particular country in order to make a claim of citizenship in that country. The higher the *Strength of National Ties*, the more importance placed on a parochial background and knowledge of local customs for granting citizenship. As we reported in Fincher and Thornhill (2012), the *Strength of National Ties* was correlated positively and strongly with the *Strength of Family Ties* ($r=0.74$, $p<0.0001$, $n=30$

countries) and *Combined Parasite Stress* ($r=0.71$, $p<0.0001$, $n=40$ countries). This relationship could be studied more thoroughly to explore the role of parasite adversity and associated collectivism in nationalism and other similar patriotic cultural features.

5.20 Xenophobia

We have stressed that xenophobic attitudes cross-nationally are correlated positively with parasite adversity (as expected from the parasite-stress theory of sociality). Here we mention briefly analyses, reported first in Fincher and Thornhill (2012), based on this relationship that used *Combined Parasite Stress* and *Strength of Family Ties*. Participants in the World Values Survey were asked about different types of people that they would not want as a neighbor. The proportion of those who said they did not want to live next to someone of a different race was associated positively with *Combined Parasite Stress* ($r=0.35$, $p=0.0009$, $n=88$ countries; see also Schaller and Murray 2011) and *Strength of Family Ties* ($r=0.45$, $p<0.0001$, $n=71$ countries). Similar questions are posed in the World Values Survey with similar relationships to *Combined Parasite Stress* and *Strength of Family Ties* (e.g., *Combined Parasite Stress* in relation to the proportion not wanting to live next to someone who speaks a different language: $r=0.42$, $p=0.004$, $n=44$ countries).

Throughout the book, we treat ethnocentrism and xenophobia as though they are positively associated. However, xenophobia and ethnocentrism can arise from separate causes, leading to cases where they may be uncorrelated or potentially even negatively correlated (Brewer 1999; Cashdan 2001b). Cashdan (2001b) demonstrated that ethnocentrism was high in traditional societies that experienced catastrophic food shortage, while xenophobia was high where the threat of intergroup violence was great. Further extension of the parasite-stress model of sociality can provide a basis for making more refined predictions about the patterns of xenophobia and ethnocentrism. For example, in a given area, zoonotic diseases may generate high morbidity and mortality, but nonzoonotics low morbidity and mortality; in this setting, ethnocentrism is predicted to be high, but xenophobia low, because zoonotic infections are not transmitted between human hosts. We discuss these issues in more detail in Chap. 14.

5.21 Moral Foundations Theory

We conclude this chapter, and before summarizing it, with a discussion of how we interpret the connection between the parasite-stress theory of values and the recently proposed Moral Foundations Theory of Haidt and Graham (2007). Where a person stands in terms of moral foundations is measured by questionnaires. We mentioned

in the previous chapter that liberals moralize in relation to an "individualizing" moral foundation that prioritizes individual autonomy and success, whereas conservatives moralize more from a "binding" foundation of morals that prioritizes the well-being, loyalty, and integrity of the collective. There, too, we tied these two factors of moral foundations theory to individualism and collectivism. Van Leeuwen et al. (2012) have connected aspects of moral foundations theory to the parasite-stress theory in showing across a large sample of nations that parasite stress is related positively and significantly to the three subfactors of the binding moral foundation (specifically, endorsement of in-group loyalty, respect of authority, and purity and holiness). This is as expected by the parasite-stress theory, because collectivism includes high value given to in-group embeddedness and loyalty, authoritarianism, and religiosity. No significant relationship was found by Van Leeuwen et al. (2012) between parasite stress and either of the two individualizing subfactors: harm or care, fairness/reciprocity. These two subfactors address how people ought (morally) to treat other people. We suggest that there is a methodological problem with the measures of both of the individualizing subfactors. From the parasite-stress-theory perspective, it is essential to distinguish between in-group and out-group harm or care or fairness/reciprocity. Hence, the binded people (collectivists) primarily care for in-group members, whereas individualists show more care toward a broad network of people. If the distinction between in-group and out-group altruism were included in a future questionnaire, we predict that high parasite stress would be associated primarily with in-group care, fairness and reciprocity, and low parasite stress predominantly with out-group care, fairness, and reciprocity.

5.22 Summary

The cross-national cultural variable collectivism–individualism is a major dimension for describing cross-cultural differences. This value dimension has been studied in some detail and measured in multiple highly correlated ways. Fincher et al. (2008) hypothesized that regional differences in parasite adversity cause this variable, with the following reasoning. Individualism confers benefits upon individuals such as independent thinking, openness to new and nontraditional ideas and ways, and willingness to interact with a diversity of people. These same traits, however, have the cost of an enhanced likelihood of contracting infectious disease. Thus, the lower the parasite stress, the greater the benefits of individualism relative to its costs. In contrast, the behaviors that define collectivism, such as ethnocentrism, xenophobia, and adherence to traditional ideas and ways, function in antipathogen defense, and thus are optimal under conditions of high parasite stress.

Across multiple measures of collectivism–individualism, Fincher et al. (2008) found that worldwide variation in parasite stress robustly predicted cross-national values of collectivism–individualism. Within regions with high severity of infectious diseases, human cultures are characterized by high collectivism whereas in

regions of low parasite stress cultures are highly individualistic. This pattern remained significant when controlling statistically for potential confounding variables. Moreover, the pattern was strong when broad cultural regions (rather than individual countries) were used in analysis.

Subsequently, Thornhill et al. (2010) computed separate indices assessing the richness (number) of three functionally distinct categories of human parasitic diseases (human-specific, multihost, zoonotic), and examined the extent to which each index uniquely predicted cross-national differences in collectivism–individualism. The parasite-stress theory of values proposes that infectious disease transmissible among humans (human-specific and multihost parasites) will be more important in predicting collectivism–individualism than those that humans can contract only from nonhuman animals (zoonotics). As predicted, both human-specific and multihost parasite richness predicted uniquely cross-national differences in collectivist–individualist values. Zoonotic parasite richness contributed little, if at all, to cross-national relationships between parasite adversity and these values. Thus, worldwide variation in these values predicted by parasite adversity appears to be attributable almost entirely to the prevalence of nonzoonotic diseases.

These cross-national results for richness of diseases in the transmission categories in relation to collectivism–individualism were repeated with parasite-severity measures (measures of number of infectious-disease cases). Nonzoonotic severity related more strongly to collectivism–individualism than did zoonotic severity. Also the measures of parasite richness were correlated nearly perfectly with measures of parasite severity.

Also, across states of the USA and societies in the Standard Cross-Cultural Sample, collectivism correlated positively with parasite stress. Furthermore, as with the cross-national results, collectivism across the USA states correlated more strongly with nonzoonotic than zoonotic human diseases.

The strength of family ties, a measure of collectivism we compiled, was correlated positively with parasite stress. This was found in cross-national analysis and analysis across states of the USA. And, as predicted, the cross-national analysis and the analysis across USA states showed that the strength of family ties was correlated more strongly with nonzoonotic infectious diseases than with zoonotic infectious diseases.

The potential confounds examined did not change these conclusions. Also, the basic relationships of values and parasite stress are robust at regional levels both cross-nationally and across the USA.

We hypothesized that reduced dispersal (high philopatry) is a defense against contact with novel parasites in out-groups and their habitats, and that reduced dispersal is the optimal preference under high parasite stress. Evidence in support of this hypothesis is seen in movement patterns across nations, states of the USA, and indigenous societies. Moreover, the parasite-stress theory of sociality provides an explanation for a general rule of ecological science, Rapoport's rule: the positive relationship between latitude and species' range size.

The social organization of animal species varies along a cooperative-breeding continuum. Cultures with high degrees of family ties have high degrees of

cooperative breeding. Evidence is provided that parasite stress accounts for this variation across human social systems. We propose that the parasite-stress theory of sociality offers a general theory of family life across humans as well as nonhuman animal taxa.

We propose that a major context for the evolution of reciprocity was in gaining benefits from out-group interactions during periods of relatively low disease threat. Thus, the parasite-stress theory of values offers a novel perspective to explain the evolution of reciprocity. Accordingly, natural selection is expected to have designed our reciprocity activities to be conditionally sensitive to risk of contagion in the local ecological setting. Under low contagion risk, reciprocity is more beneficial than under high risk.

We suggest that, in human evolutionary history, as the physical environmental problems and predators became less important as mortality agents, parasites became more important as agents of natural selection. The human lineage may be unique among branches in the Tree of Life in the relatively great importance of parasite stress as a source of Darwinian selection. Also, the reduction in predator-based natural selection in human evolutionary history may have selected for relatively high virulence in human parasites. Moreover, we suggest that it was local change in parasite adversity, and challenges for adoption of values to cope with this change, that accounts for the runaway social selection in the human lineage that produced important aspects of human uniqueness in cognitive ability.

The parasite-stress theory of values suggests useful new research directions for the study of the demographic transition, patriotism, xenophobia, ethnocentrism, and moral foundations theory. The demographic transition's association with increases in non-kin-to-kin ratio in people's social networks may simply reflect individualism and corresponding reduction of parasite adversity. Patriotism may be a manifestation of collectivist values and concomitant high parasite stress. Xenophobia and ethnocentrism often covary positively, but there are circumstances identified by the parasite-stress theory in which they should not do so. So-called moral foundations theory could be improved by distinguishing the in-group and out-group components of altruism and how each relates to parasite stress.

Certain misunderstandings of correlational findings generated by comparative methodology are discussed. It is explained that all scientific results are correlational, including those from experiments. Many of the results supporting the parasite-stress theory of sociality are from application of the comparative method and associated statistical correlation with statistical controls; other research supporting it uses experimental and/or observational methods. The scientific value of any finding depends upon the control of confounders, not the type of method itself. Thus, the method of testing is always, in itself, irrelevant. When thorough controls are in place, correlation documents causation whether the correlation arises from experimentation, comparative methodology, or observational analysis.

We discuss ecological correlations and the ecological fallacy and how they relate to testing of the parasite stress theory of sociality.

5.23 Appendix 1

Cross-national scores of human infectious-disease richness (number of diseases) by type of transmission to humans. The type "Nonzoonotic" is the sum of "Human-specific" and "Multihost"

Country	Human-specific	Multihost	Nonzoonotic	Zoonotic
Afghanistan	105	23	128	53
Albania	100	21	121	53
Algeria	104	22	126	55
American Samoa	101	21	122	42
Andorra	100	20	120	44
Angola	107	25	132	61
Anguilla	100	21	121	40
Antigua and Barbuda	101	23	124	41
Argentina	102	25	127	68
Armenia	102	21	123	59
Aruba	100	21	121	41
Australia	101	22	123	64
Austria	99	22	121	52
Azerbaijan	100	24	124	57
Azores	98	21	119	41
Bahamas, The	99	22	121	41
Bahrain	100	21	121	42
Bangladesh	103	27	130	52
Barbados	100	22	122	40
Belarus	99	23	122	60
Belgium	99	22	121	51
Belize	101	25	126	53
Benin	107	27	134	53
Bermuda	99	22	121	39
Bhutan	101	23	124	51
Bolivia	104	29	133	59
Bosnia and Herzegovina	99	24	123	54
Botswana	104	22	126	53
Brazil	107	30	137	86
British Virgin Islands	101	21	122	40
Brunei	102	21	123	46
Bulgaria	100	22	122	60
Burkina Faso	108	27	135	58
Burundi	106	24	130	56
Cambodia	105	26	131	58
Cameroon	109	27	136	63
Canada	100	21	121	70
Canary Islands	100	21	121	48
Cape Verde	105	21	126	46
Cayman Islands	100	21	121	41

(continued)

(continued)

Country	Human-specific	Multihost	Nonzoonotic	Zoonotic
Central African Republic	109	26	135	71
Chad	110	25	135	58
Chile	102	23	125	57
China	105	27	132	87
Christmas Island	100	20	120	38
Colombia	107	29	136	71
Comoros	104	21	125	50
Congo, Democratic Republic of the	108	25	133	71
Congo, Republic of the	107	24	131	69
Cook Islands	101	22	123	41
Costa Rica	103	27	130	58
Cote d'Ivoire	110	28	138	63
Croatia	100	21	121	60
Cuba	101	21	122	52
Cyprus	100	21	121	45
Czech Republic	100	21	121	62
Democratic People's Republic of Korea (North)	101	28	129	59
Denmark	99	20	119	51
Djibouti	105	23	128	52
Dominica	101	23	124	43
Dominican Republic	103	23	126	51
East Timor	107	27	134	75
Ecuador	105	30	135	63
Egypt	104	23	127	69
El Salvador	102	24	126	56
Eritrea	107	25	132	63
Estonia	100	21	121	56
Ethiopia	108	26	134	65
Falkland Islands	100	20	120	43
Fiji	101	22	123	44
Finland	100	20	120	53
France	100	22	122	66
French Guiana	103	27	130	54
French Polynesia	101	21	122	44
Gabon	107	24	131	67
Gambia, The	105	26	131	60
Georgia	101	21	122	56
Germany	100	20	120	60
Ghana	109	26	135	61
Gibraltar	100	21	121	44
Greece	100	22	122	59
Greenland	100	20	120	42
Grenada	100	21	121	42
Guadeloupe	102	21	123	49
Guam	100	21	121	44

(continued)

(continued)

Country	Human-specific	Multihost	Nonzoonotic	Zoonotic
Guatemala	104	28	132	58
Guinea	107	27	134	57
Guinea-Bissau	108	24	132	51
Guyana	104	26	130	63
Haiti	104	22	126	49
Honduras	103	26	129	56
Hong Kong	103	22	125	50
Hungary	100	21	121	58
Iceland	100	20	120	42
India	105	27	132	86
Indonesia	107	27	134	79
Iran	103	23	126	68
Iraq	104	22	126	55
Ireland	100	20	120	48
Israel	100	22	122	55
Italy	100	21	121	64
Jamaica	100	21	121	49
Japan	101	24	125	69
Jordan	102	22	124	51
Kazakhstan	100	21	121	64
Kenya	107	27	134	72
Kiribati	101	22	123	40
Kuwait	100	22	122	48
Kyrgyzstan	101	21	122	60
Laos	104	25	129	58
Latvia	100	21	121	53
Lebanon	100	22	122	50
Lesotho	103	21	124	51
Liberia	106	25	131	60
Libya	102	22	124	54
Liechtenstein	100	20	120	46
Lithuania	100	21	121	53
Luxembourg	100	20	120	45
Macau	100	23	123	42
Macedonia	100	21	121	58
Madagascar	106	22	128	62
Malawi	107	23	130	57
Malaysia	106	27	133	72
Maldives	104	21	125	45
Mali	109	25	134	61
Malta	100	21	121	45
Marshall Islands	101	21	122	43
Martinique	101	22	123	47
Mauritania	107	23	130	55
Mauritius	103	21	124	47

(continued)

(continued)

Country	Human-specific	Multihost	Nonzoonotic	Zoonotic
Mexico	104	28	132	74
Micronesia, Federated States of	102	22	124	43
Moldova	100	21	121	56
Monaco	100	20	120	43
Mongolia	101	24	125	53
Montserrat	100	22	122	40
Morocco	103	24	127	53
Mozambique	107	27	134	57
Myanmar	104	30	134	59
Namibia	104	25	129	54
Nauru	100	22	122	39
Nepal	103	26	129	51
Netherlands	99	21	120	54
Netherlands Antilles	99	22	121	41
New Caledonia	100	22	122	45
New Zealand	99	21	120	45
Nicaragua	102	28	130	51
Niger	108	27	135	57
Nigeria	110	31	141	71
Niue	100	22	122	38
Norfolk Island	99	21	120	38
Northern Mariana Islands	100	22	122	42
Norway	99	21	120	52
Oman	103	23	126	48
Pakistan	104	29	133	58
Palau	100	22	122	42
Panama	104	31	135	61
Papua New Guinea	103	25	128	54
Paraguay	100	29	129	53
Peru	104	32	136	64
Philippines	104	27	131	65
Pitcairn Islands	99	21	120	38
Poland	99	22	121	56
Portugal	99	24	123	57
Puerto Rico	101	22	123	50
Qatar	101	23	124	43
Republic of Korea (South)	103	28	131	63
Reunion	101	22	123	47
Romania	100	24	124	57
Russia	103	27	130	81
Rwanda	106	27	133	57
Saint Helena	99	21	120	40
Saint Kitts and Nevis	102	22	124	41
Saint Lucia	102	23	125	41
Saint Vincent and Grenadines	101	23	124	39

(continued)

(continued)

Country	Human-specific	Multihost	Nonzoonotic	Zoonotic
Samoa	100	22	122	40
San Marino	99	21	120	41
Sao Tome and Principe	104	22	126	49
Saudi Arabia	105	24	129	55
Scotland	99	22	121	53
Senegal	108	29	137	62
Seychelles	100	22	122	42
Sierra Leone	107	26	133	55
Singapore	101	24	125	50
Slovakia	99	22	121	58
Slovenia	99	24	123	58
Solomon Islands	101	24	125	42
Somalia	105	27	132	56
South Africa	103	25	128	70
Spain	99	24	123	59
Sri Lanka	103	26	129	57
Sudan	109	30	139	68
Suriname	105	28	133	57
Swaziland	103	22	125	50
Sweden	99	21	120	51
Switzerland	99	21	120	55
Syria	100	23	123	47
Taiwan	100	27	127	61
Tajikistan	101	23	124	56
Tanzania	107	27	134	64
Thailand	104	32	136	73
Togo	107	27	134	53
Tokelau	100	22	122	38
Tonga	100	22	122	41
Trinidad and Tobago	102	25	127	51
Tunisia	102	25	127	54
Turkey	102	24	126	62
Turkmenistan	100	24	124	55
Turks and Caicos Islands	100	22	122	39
Tuvalu	100	23	123	39
Uganda	109	29	138	72
Ukraine	100	23	123	60
United Arab Emirates	101	22	123	47
United Kingdom	99	22	121	55
United States	103	26	129	86
Uruguay	99	22	121	57
Uzbekistan	101	24	125	55
Vanuatu	101	23	124	41
Venezuela	107	30	137	65
Vietnam	103	30	133	64

(continued)

(continued)

Country	Human-specific	Multihost	Nonzoonotic	Zoonotic
Virgin Islands, US	100	23	123	40
Wake Island	99	21	120	38
Wallis and Futuna Islands	100	22	122	40
Western Sahara	99	21	120	43
Yemen	104	25	129	51
Zambia	106	25	131	58
Zimbabwe	105	23	128	65

5.24 Appendix 2

Notifiable human infectious diseases within the United States tracked by the Centers for Disease Control and Prevention (CDC) from the years 1993 to 2007 classified by transmission type (Human-specific, Multihost, or Zoonotic) or if the disease was not included in this classification (labeled here as "Removed"). There is some redundancy in disease names because in some cases a disease was variably named by the CDC across years

Disease name	Transmission type
AIDS	Human-specific
Chancroid	Human-specific
Chlamydia	Human-specific
Cholera	Human-specific
Diphtheria	Human-specific
Gonorrhea	Human-specific
Granuloma inguinale	Human-specific
Haemophilus influenzae	Human-specific
Haemophilus influenzae—Age <5 years—Nonserotype b	Human-specific
Haemophilus influenzae—Age <5 years—Serotype b	Human-specific
Haemophilus influenzae—Age <5 years—Unknown serotype	Human-specific
Haemophilus influenzae—All ages all serotypes	Human-specific
Haemophilus influenzae, invasive disease (age <5 years) unknown serotype	Human-specific
Haemophilus influenzae, invasive disease (all ages, serotypes)	Human-specific
Haemophilus influenzae, invasive disease (age <5 years) Nonserotype b	Human-specific
Haemophilus influenzae, invasive disease (age <5 years) Serotype b	Human-specific
Hepatitis B	Human-specific
Hepatitis C/Non-A, Non-B	Human-specific
Hepatitis Non-A, Non-B	Human-specific
Hepatitis unsp.	Human-specific
Lymphogranuloma venereum	Human-specific
Malaria	Human-specific
Measles—Imported	Human-specific

(continued)

(continued)

Disease name	Transmission type
Measles—Indigenous	Human-specific
Meningococcal disease (all serogroups)	Human-specific
Meningococcal disease (other serogroup)	Human-specific
Meningococcal disease (Serogroup A, C, Y, and W-135)	Human-specific
Meningococcal disease (Serogroup B)	Human-specific
Meningococcal disease (Serogroup unknown)	Human-specific
Meningococcal disease, invasive—All serogroups	Human-specific
Meningococcal disease, invasive—Other serogroup	Human-specific
Meningococcal disease, invasive—Serogroup A, C, Y, and W-135	Human-specific
Meningococcal disease, invasive—Serogroup B	Human-specific
Meningococcal disease, invasive—Serogroup unknown	Human-specific
Meningococcal infections	Human-specific
Mumps	Human-specific
Pertussis	Human-specific
Poliomyelitis, paralytic	Human-specific
Rubella	Human-specific
Rubella—Congenital syndrome	Human-specific
SARS-CoV[a]	Human-specific
Streptococcus pneumoniae, invasive disease, drug-resistant (age <5years)	Human-specific
Syphilis—All stages	Human-specific
Syphilis—Congenital (<1 year)	Human-specific
Syphilis—Primary and secondary	Human-specific
Typhoid fever	Human-specific
Varicella deaths	Human-specific
Cryptosporidiosis[b]	Multihost
Escherichia coli O157:H7	Multihost
Hansen disease	Multihost
Hepatitis A	Multihost
Influenza-associated pediatric mortality	Multihost
Listeriosis	Multihost
Novel influenza A virus infections	Multihost
Shigatoxin-producing *E. coli* (STEC)	Multihost
Shigellosis	Multihost
Tuberculosis	Multihost
Yellow fever	Multihost
Anthrax	Zoonotic
Botulism—Infant	Zoonotic
Botulism—Foodborne	Zoonotic
Botulism—Other	Zoonotic
Brucellosis	Zoonotic
California serogroup virus disease (neuro-invasive)	Zoonotic
Domestic arboviral diseases—California serogroup—Neuro-invasive	Zoonotic
Domestic arboviral diseases—California serogroup—Non-neuro-invasive	Zoonotic
Domestic arboviral diseases—Eastern Equine—Neuro-invasive	Zoonotic
Domestic arboviral diseases—Eastern Equine—Non-neuro-invasive	Zoonotic

(continued)

(continued)

Disease name	Transmission type
Domestic arboviral diseases—Powassan—Neuro-invasive	Zoonotic
Domestic arboviral diseases—Powassan—Non-neuro-invasive	Zoonotic
Domestic arboviral diseases—St. Louis—Neuro-invasive	Zoonotic
Domestic arboviral diseases—St. Louis—Non-neuro-invasive	Zoonotic
Domestic arboviral diseases—West Nile—Neuro-invasive	Zoonotic
Domestic arboviral diseases—West Nile—Non-neuro-invasive	Zoonotic
Eastern equine encephalitis (neuro-invasive)	Zoonotic
Eastern equine encephalitis (non-neuro-invasive)	Zoonotic
Encephalitis—California serogroup viral	Zoonotic
Encephalitis—Eastern Equine	Zoonotic
Encephalitis—Postinfectious	Zoonotic
Encephalitis—Powassan	Zoonotic
Encephalitis—Primary infections	Zoonotic
Encephalitis—St. Louis	Zoonotic
Encephalitis—West Nile	Zoonotic
Leptospirosis	Zoonotic
Lyme disease	Zoonotic
Murine typhus fever	Zoonotic
Plague	Zoonotic
Powassan virus disease (neuro-invasive)	Zoonotic
Powassan virus disease (non-neuro-invasive)	Zoonotic
Psittacosis	Zoonotic
Rabies—Animal	Zoonotic
Rabies—Human	Zoonotic
Rocky Mountain spotted fever (RMSF)	Zoonotic
Salmonellosis	Zoonotic
St. Louis encephalitis (neuro-invasive)	Zoonotic
St. Louis encephalitis (non-neuro-invasive)	Zoonotic
Trichinosis	Zoonotic
Tularemia	Zoonotic
West Nile virus disease (neuro-invasive)	Zoonotic
West Nile virus disease (non-neuro-invasive)	Zoonotic
Legionellosis	Removed
Tetanus	Removed
Toxic-shock syndrome	Removed

[a]SARS-CoV was classified as multihost in Fincher and Thornhill (2012)
[b]Cryptosporidiosis was classified as zoonotic in Fincher and Thornhill (2012)

5.25 Appendix 3

Standardized pathogen severity scores for USA states for different transmission types with or without the District of Columbia (DC). The transmission type "Nonzoonotic" is the sum of "Human-Specific" and "Multihost"

State	Human-specific with DC	Multihost with DC	Nonzoonotic with DC	Zoonotic with DC	Human-specific without DC	Multihost without DC	Nonzoonotic without DC	Zoonotic without DC
Alabama	0.90	−0.10	0.79	−0.19	1.40	−0.08	1.33	−0.20
Alaska	0.43	0.28	0.71	−0.65	0.60	0.31	0.91	−0.65
Arizona	−0.15	1.61	1.46	−0.15	−0.10	1.68	1.58	−0.16
Arkansas	0.24	−0.01	0.23	0.86	0.51	0.02	0.53	0.85
California	−0.11	0.88	0.77	−0.43	−0.05	0.94	0.90	−0.44
Colorado	−0.27	−0.16	−0.43	−0.17	−0.27	−0.13	−0.40	−0.18
Connecticut	−0.23	−0.71	−0.94	1.91	−0.17	−0.69	−0.86	1.88
Delaware	0.55	−0.07	0.48	0.88	0.90	−0.03	0.87	0.86
District of Columbia	4.82	1.52	6.33	−0.44				
Florida	0.09	0.58	0.67	0.45	0.30	0.64	0.94	0.44
Georgia	1.01	0.76	1.77	0.66	1.56	0.82	2.38	0.65
Hawaii	−0.16	0.36	0.21	0.88	−0.15	0.40	0.25	0.87
Idaho	−0.93	−0.14	−1.07	−0.29	−1.13	−0.13	−1.26	−0.29
Illinois	0.46	0.09	0.56	−0.43	0.76	0.13	0.89	−0.43
Indiana	−0.15	−0.47	−0.62	−0.87	−0.07	−0.44	−0.52	−0.87
Iowa	−0.63	−0.12	−0.75	−0.43	−0.75	−0.09	−0.84	−0.43
Kansas	−0.37	−0.41	−0.78	−0.35	−0.38	−0.39	−0.77	−0.35
Kentucky	−0.53	−0.21	−0.74	−0.90	−0.57	−0.18	−0.75	−0.91
Louisiana	1.28	0.00	1.29	0.19	1.93	0.04	1.97	0.18
Maine	−1.11	−1.09	−2.20	−0.33	−1.38	−1.09	−2.47	−0.34
Maryland	0.59	−0.13	0.46	0.49	1.01	−0.09	0.92	0.48
Massachusetts	−0.64	−0.31	−0.95	0.87	−0.73	−0.28	−1.01	0.86
Michigan	0.21	−0.45	−0.23	−0.95	0.41	−0.42	−0.01	−0.95
Minnesota	−0.64	−0.15	−0.79	−0.28	−0.75	−0.12	−0.87	−0.29
Mississippi	1.87	0.05	1.92	0.98	2.74	0.07	2.81	0.97
Missouri	0.29	0.41	0.70	−0.52	0.53	0.44	0.98	−0.53
Montana	−0.83	−0.61	−1.44	−0.37	−1.04	−0.60	−1.64	−0.38
Nebraska	−0.47	−0.05	−0.53	−0.44	−0.54	−0.03	−0.57	−0.45

Nevada	−0.20	−0.03	−0.23	−0.66	−0.16	0.00	−0.16	−0.67
New Hampshire	−1.11	−0.95	−2.05	−0.11	−1.36	−0.94	−2.31	−0.12
New Jersey	−0.37	−0.23	−0.60	0.32	−0.33	−0.20	−0.53	0.30
New Mexico	0.01	0.89	0.90	−0.07	0.08	0.93	1.01	−0.08
New York	0.24	0.33	0.57	0.64	0.50	0.38	0.88	0.63
North Carolina	0.63	−0.17	0.46	0.24	1.06	−0.14	0.91	0.23
North Dakota	−0.86	−0.47	−1.33	0.12	−1.09	−0.46	−1.54	0.12
Ohio	0.27	−0.45	−0.18	−0.64	0.48	−0.44	0.04	−0.64
Oklahoma	0.06	1.44	1.50	−0.29	0.21	1.53	1.74	−0.29
Oregon	−0.71	0.15	−0.56	−0.83	−0.85	0.18	−0.67	−0.83
Pennsylvania	−0.19	−0.59	−0.78	0.17	−0.14	−0.56	−0.70	0.16
Rhode Island	−0.41	−0.50	−0.91	1.10	−0.45	−0.48	−0.93	1.08
South Carolina	1.12	−0.18	0.94	0.50	1.71	−0.14	1.57	0.49
South Dakota	−0.58	0.90	0.32	0.87	−0.72	0.92	0.21	0.86
Tennessee	0.62	0.21	0.83	−0.59	1.03	0.25	1.28	−0.59
Texas	0.26	1.27	1.53	−0.36	0.50	1.35	1.85	−0.37
Utah	−0.87	0.34	−0.53	−0.25	−1.08	0.36	−0.72	−0.26
Vermont	−1.00	−0.86	−1.86	0.51	−1.22	−0.85	−2.07	0.50
Virginia	−0.04	−0.40	−0.44	0.17	0.09	−0.37	−0.28	0.16
Washington	−0.52	0.00	−0.52	−0.69	−0.60	0.03	−0.57	−0.69
West Virginia	−0.95	−1.18	−2.14	−0.60	−1.16	−1.19	−2.34	−0.60
Wisconsin	−0.06	−0.10	−0.16	0.23	0.02	−0.07	−0.05	0.21
Wyoming	−0.90	−0.79	−1.69	0.24	−1.11	−0.80	−1.90	0.23

References

Alesina, A., & Giuliano, P. (2010). The power of the family. *Journal of Economic Growth* 15: 93–125.

Alexander, R. D. (1978). Evolution, creation and biology teaching. *American Biology Teacher* 40: 91–107.

Alexander, R. D. (1987). *The Biology of Moral Systems*. Aldine de Gruyter, New York, NY.

Alexander, R. D. (1990). *How Did Humans Evolve? Reflections on the Uniquely Unique Species*. Special Publication No. 1. Museum of Zoology, The University of Michigan, Ann Arbor, MI.

Andersson, M. (1984). The evolution of eusociality. *Annual Review of Ecology and Systematics* 15: 165–189.

Arnold, K. E., & Owens, I. P. F. (1998). Cooperative breeding in birds: A comparative test of the life history hypothesis. *Proceedings of the Royal Society B* 265: 739–745.

Binford, L. R. (2001). *Constructing Frames of Reference: An Analytical Method for Archaeological Theory Building Using Hunter Gatherer and Environmental Data Sets.* University of California Press, Berkeley, CA.

Blumstein, D. T., & Møller, A. P. (2008). Is sociality associated with high longevity in North American birds? *Biology Letters* 4: 146–148.

Brewer, M. B. (1999). The psychology of prejudice: Ingroup love or outgroup hate? *Journal of Social Issues* 55: 429–444.

Brockmann, H. J. (1984). The evolution of social behaviour in insects. In *Behavioural Ecology: An Evolutionary Approach* (eds. J. R. Krebs & N. B. Davies), pp. 340–361. Sinauer Associates, Inc., Sunderland, MA.

Brown, J. L. (1987). *Helping and Communal Breeding in Birds: Ecology and Evolution*. Princeton University Press, Princeton, NJ.

Carney, D. R., Jost, J. T., Gosling, S. D. et al. (2008). The secret lives of liberals and conservatives: Personality profiles, interaction styles, and the things they leave behind. *Political Psychology* 29: 807–840.

Cashdan, E. (2001a). Ethnic diversity and its environmental determinants: Effects on climate, pathogens, and habitat diversity. *American Anthropology* 103: 968–991.

Cashdan, E. (2001b). Ethnocentrism and xenophobia: A cross-cultural study. *Current Anthropology* 42: 760–765.

Cashdan, E., & Steele, M. (2013). Pathogen prevalence, group bias, and collectivism in the standard cross-cultural sample. *Human Nature* 24: 59–75.

Census Bureau. (2005). http://www.census.gov/hhes/migration/data/acs/state-to-state.html.

Census Bureau. (2005). http://www.census.gov/popest/data/historical/2000s/vintage_2005/index.html.

Charnov, E. L. (1993). *Life History Invariants: Some Explorations of Symmetry in Evolutionary Ecology*. Oxford University Press, Oxford, U.K.

Clay, R., Terrizzi Jr., J. A., & Shook, N. J. (2012). Individual differences in the behavioral immune system and the emergence of cultural systems. *Journal of Social Psychology* 43: 174–184.

Cohen, D. (2001). Cultural variation: considerations and implications. *Psychological Bulletin* 127: 451–471.

Cukur, C. S., De Guzman, M. R. T., & Carlo, G. (2004). Religiosity, values, and horizontal and vertical individualism–collectivism: A study of Turkey, the United States, and the Philippines. *Journal of Social Psychology* 144: 613–634.

Dunbar, R., & Shultz, S. (2007). Evolution in the social brain. *Science* 317: 1344–1347.

Dunn, R. R., Davies, T. J., Harris, N. C. et al. (2010). Global drivers of human pathogen richness and prevalence. *Proceedings of the Royal Society B* 27: 2587–2595.

Ekman, J. (2006). Family living among birds. *Journal of Avian Biology* 37: 289–298.

Emlen, S. T. (1994). Benefits, constraints and the evolution of the family. *Trends in Ecology and Evolution* 9: 282–285.

Emlen, S. T. (1995). An evolutionary theory of the family. *Proceedings of the National Academy of Sciences USA* 92: 8092–8099.
Emlen, S. T. (1997). The evolutionary study of human family systems. *Social Science Information Sur Les Sciences Sociales* 36: 563–589.
Eppig, C., Fincher, C. L., & Thornhill, R. (2010). Parasite prevalence and the worldwide distribution of cognitive ability. *Proceedings of the Royal Society of London B* 277: 3801–3808.
Eppig, C., Fincher, C. L., & Thornhill, R. (2011). Parasite prevalence and the distribution of intelligence among the states of the USA. *Intelligence* 39: 155–160.
Faulkner, J., Schaller, M., Park, J. H. et al. (2004). Evolved disease-avoidance mechanisms and contemporary xenophobic attitudes. *Group Processes and Intergroup Relations* 7: 333–353.
Fincher, C. L., & Thornhill, R. (2008a). A parasite-driven wedge: Infectious diseases may explain language and other biodiversity. *Oikos* 117: 1289–1297.
Fincher, C. L., & Thornhill, R. (2008b). Assortative sociality, limited dispersal, infectious disease and the genesis of the global pattern of religion diversity. *Proceedings of the Royal Society of London, Biological Sciences* 275: 2587–2594.
Fincher, C. L., & Thornhill, R. (2012). Parasite-stress promotes in-group assortative sociality: The cases of strong family ties and heightened religiosity. *Behavioral and Brain Sciences* 35: 61–79.
Fincher, C. L., Thornhill, R., Murray, D. R. et al. (2008). Pathogen prevalence predicts human cross-cultural variability in individualism/collectivism. *Proceedings of the Royal Society of London Biological Sciences* 275: 1279–1285.
Flinn, M. V., Geary, D. C., & Ward, C. V. (2005). Ecological dominance, social competition, and coalitionary arms races: Why humans evolved extraordinary intelligence. *Evolution and Human Behavior* 26: 10–46.
Freedom House. (2008). http://www.freedomhouse.org.
Freeland, W. J. (1976). Pathogens and the evolution of primate sociality. *Biotropica* 8: 12–24.
Freeland, W. J. (1979). Primate social groups as biological islands. *Ecology* 60: 719–728.
Fumagalli, M., Sironi, M., Pozzoli, U. et al. (2011). Signatures of environmental genetic adaptation pinpoint pathogens as the main selective pressure through human evolution. *PLoS Genetics* 7: e1002355.
Gangestad, S. W., & Buss, D. M. (1993). Pathogen prevalence and human mate preference. *Ethology and Sociobiology* 14: 89–96.
Gangestad, S. W., Haselton, M. G., & Buss, D. M. (2006). Evolutionary foundations of cultural variation: Evoked culture and mate preferences. *Psychological Inquiry* 17: 75–95.
Gelfand, M. J., Bhawuk, D. P. S., Nishii, L. H. et al. (2004). Individualism and collectivism. In *Culture, Leadership, and Organizations: The GLOBE Study of 62 Societies* (eds. R. J. House, P. J. Hanges, M. Javidan et al.), pp. 437–512. Sage Publications, Thousand Oaks, CA.
Gordon, R.A. (1968). Issues in Multiple Regression. *The American Journal of Sociology* 73: 592–616.
Greger, M. (2007). The human/animal interface: Emergence and resurgence of zoonotic infectious diseases. *Critical Review of Microbiology* 33: 243–299.
Guernier, V., Hochberg, M. E., & Guégan, J. -F. (2004). Ecology drives the worldwide distribution of human diseases. *PLoS Biology* 2: 740–746.
Gupta, V., & Hanges, P. J. (2004). Regional and climate clustering of societal cultures. In *Culture, Leadership, and Organizations: The GLOBE Study of 62 Societies* (eds. R. J. House, P. J. Hanges, M. Javidan et al.), pp. 178–218. Sage Publications, Thousand Oaks, CA.
Gurven, M., Allen-Arave, W., Hill, K. et al. (2000). "It's a Wonderful Life": Signaling generosity among the Ache of Paraguay. *Evolution and Human Behavior* 21: 263–282.
Haidt, J., & Graham, J. (2007). When morality opposes justice: Conservatives have moral intuitions that liberals may not recognize. *Social Justice Research* 20: 98–116.
Heine, S. J. (2008). *Cultural psychology.* Norton, New York, NY.
Hill, K., & Hurtado, A. M. (2009). Cooperative breeding in South American hunter-gatherers. *Proceedings of the Royal Society B* 276: 3863–3870.

Hofstede, G. (2001). *Culture's Consequences: Comparing Values, Behaviors, Institutions, and Organizations Across Nations*, 2nd ed. Sage Publications, Thousand Oaks, CA.

House, R. J., Hanges, P. J., Javidan, M. et al., Eds. (2004). *Culture, Leadership, and Organizations: The GLOBE study of 62 Societies.* Sage Publications, Thousand Oaks, CA.

Inbar, Y., Pizarro, D. A., Iyer, R. et al. (2012). Disgust sensitivity, political conservatism, and voting. *Social Psychological and Personality Science* 5: 537–544.

Jetz, W., & Rubenstein, D. R. (2011). Environmental uncertainty and the global biogeography of cooperative breeding in birds. *Current Biology* 21: 72–78.

Jones, K. E., Patel, N. G., Levy, M. A. et al. (2008). Global trends in emerging infectious diseases. *Nature* 451: 990–993.

Kaplan, H. S., & Gangestad, S. W. (2005). Life history theory and evolutionary psychology. In *The Handbook of Evolutionary Psychology* (ed. D. M. Buss), pp.68–95. John Wiley and Sons, Inc., New York, NY.

Kashima, E. S., & Kashima, Y. (1998). Culture and language: The case of cultural dimensions and personal pronoun use. *Journal of Cross-Cultural Psychology* 29: 461–486.

Kitayama, S., Ishii, K., Tmada, T. et al. (2006). Voluntary settlement and the spirit of independence: Evidence from Japan's "Northern Frontier." *Journal of Personality and Social Psychology* 91: 369–384.

Loehle, C. (1995). Social barriers to pathogen transmission in wild animal populations. *Ecology* 76: 326–335.

Low, B. S. (1988). Pathogen stress and polygyny in humans. In *Human Reproductive Behavior: A Darwinian Perspective* (eds. L. Betzig, M. Borgerhoff Mulder, & P. Turke), pp. 115–127. Cambridge University Press, Cambridge, U.K.

Low, B. S. (1990). Marriage systems and pathogen stress in human societies. *American Zoologist* 30: 325–339.

Low, B. S. (1994). Pathogen severity cross-culturally. *World Cultures* 8: 24–34.

Low, B. S. (2000). *Why Sex Matters: A Darwinian Look at Human Behavior*. Princeton University Press, Princeton, NJ.

Maudlin, I., Eisler, M., & Welburn, S. (2009). The neglected zoonoses. *Philosophical Transactions of the Royal Society B* 364: 2777–2787.

Mayr, E. (1982). *The Growth of Biological Thought: Diversity, Evolution, and Inheritance*. Harvard University Press, Cambridge, MA.

McNeill, W. H. (1998). *Plagues and Peoples*. Anchor, Harpswell, ME.

Minkov, M. (2011). *Cultural Differences in a Globalizing World*. Emerald Group Publishing Ltd., Bingley, U.K.

McNeill, W. H. (1981). Migration patterns and infection in traditional societies. In *Changing Disease Patterns and Human Behavior* (eds. N. F. Stanley & R. A. Joske), pp. 27–36. Academic Press, Salt Lake, UT.

Møller, A. P. (1998). Evidence of larger impact of parasites on hosts in the tropics: Investment in immune function within and outside the tropics. *Oikos* 82: 265–270.

Møller, A. P. (2008). Interactions between interactions: Predator–prey, parasite–host, and mutualistic interactions. *Annals of New York Academy Sciences* 1133: 180–186.

Møller, A. P., Dufva, R., & Allander, K. (1993). Parasites and the evolution of host social behaviour. *Advances in the Study of Behavior* 22: 65–102.

Murdock, G. P. (1949). *Social Structure*. MacMillan, New York, NY.

Murdock, G. P., & White, D. R. (1969). Standard cross-cultural sample. *Ethnology* 8: 329–69.

Murray, D. R., & Schaller, M. (2010). Historical prevalence of infectious diseases within 230 geopolitical regions: A tool for investigating origins of culture. *Journal of Cross-Cultural Psychology* 41: 99–108.

Murray, D. R., Trudeau, R., & Schaller, M. (2011). On the origins of cultural differences in conformity: Four tests of the pathogen prevalence hypothesis. *Personality and Social Psychology Bulletin* 37: 318–329.

Newson, L., Postmes, T., Lea, S. E. G. et al. (2005). Why are modern families small? Toward an evolutionary and cultural explanation for the demographic transition. *Journal of Personality and Social Psychology* 9: 360–375.

Oishi, S., Schimmack, U., Diener, E. et al. (1998). The measurement of values and individualism–collectivism. *Personality and Social Psychology Bulletin* 24: 1177–1189.

Pearce-Duvet, J. M. C. (2006). The origin of human pathogens: evaluating the role of agriculture and domestic animals in the evolution of human disease. *Biological Reviews* 81: 369–382.

Pollet, T. V., Tybur, J. M., Frankenhuis, W. E., & Rickard, I. J. (in press). What can cross-cultural correlations teach us about human nature? *Human Nature*

Quinlan, R. J. (2007). Human parental effort and environmental risk. *Proceedings of the Royal Society B* 274: 121–125.

Robinson, W. S. (1950). Ecological correlations and the behavior of individuals. *American Sociological Review* 15: 351–357.

Schaller, M., & Murray, D. R. (2011). Infectious disease and the creation of culture. In *Advances in Culture and Psychology* (eds. M. Gelfand, C.-y. Chiu, & Y.-y. Hong), pp. 99–151. Oxford University Press, New York, NY.

Sherman, P. W., Lacey, E. A., Reeve, H. K. et al. (1995). The eusociality continuum. *Behavioral Ecology* 6: 102–108.

Sherman, P. W., & Billing, J. (1999). Darwinian gastronomy: Why we use spices. *BioScience* 49: 453–463.

Shultz, S., & Dunbar, R. I. M. (2007). The evolution of the social brain: Anthropoid primates contrast with other vertebrates. *Proceedings of the Royal Society B: Biological Sciences* 274: 2429–2436.

Smith, K. F., Sax, D. F., Gaines, S. D. et al. (2007). Globalization of human infectious disease. *Ecology* 88: 1903–1910.

Spottiswoode, C. N. (2008). Cooperative breeding and immunity: A comparative study of PHA response in African birds. *Behavioral Ecology and Sociobiology* 62: 963–974.

Stearns, S. C. (1976). Life-history tactics: A review of the ideas. *Quarterly Review of Biology* 51: 3–47.

Stevens, G. C. (1989). The latitudinal gradient in geographical range: How so many species coexist in the tropics. *American Naturalist* 133: 240–256.

Subramanian, S. V., Jones, K., Kaddour, A. et al. (2009). Revisiting Robinson: the perils of individualistic and ecological fallacy. *International Journal of Epidemiology* 38: 342–360.

Sugiyama, L. S. (2004). Illness, injury, and disability among Shiwiar forager–horticulturalists: Implications of human life history. *American Journal of Physical Anthropology* 123: 371–389.

Sugiyama, L. S., & Sugiyama, M. S. (2003). Social roles, prestige, and health risk: Social niche specialization as a risk-buffering strategy. *Human Nature* 14: 165–190.

Suh, E., Diener, E., Oishi, S. et al. (1998). The shifting basis of life satisfaction judgments across cultures: Emotions versus norms. *Journal of Personality and Social Psychology* 74: 482–493.

Terrizzi Jr., J. A., Shook, N. J., & McDaniel, M. A. (2013). The behavioral immune system and social conservatism: A meta-analysis. *Evolution and Human Behavior* 34: 99–108.

Thornhill, R. (1984). Scientific methodology in entomology. *Florida Entomologist* 67: 74–96.

Thornhill, R., & Fincher, C. L. (2013). The comparative method in cross-cultural and cross-species research. *Evolutionary Biology* 40: 480–493.

Thornhill, R., Fincher, C. L., & Aran, D. (2009). Parasites, democratization, and the liberalization of values across contemporary countries. *Biological Reviews* 84: 113–131.

Thornhill, R., Fincher, C. L., Murray, D. R. et al. (2010). Zoonotic and non-zoonotic diseases in relation to human personality and societal values: Support for the parasite-stress model. *Evolutionary Psychology* 8: 151–169.

Thornhill, R., & Palmer, C. T. (2000). *A Natural History of Rape: Biological Bases of Sexual Coercion*. MIT Press, Cambridge, MA.

Triandis, H. C. (1995). *Individualism and Collectivism*. Westview Press, Boulder, CO.

U.S. Bureau of Economic Analysis (BEA), U.S. Department of Commerce. http://www.bea.gov.
U.S. Census Bureau, U.S. Department of Commerce, http://www.census.gov.
Vandello, J. A., & Cohen, D. (1999). Patterns of individualism and collectivism across the United States. *Journal of Personality and Social Psychology* 77: 279–292.
Van den Berghe, P. L. (1981). *The Ethnic Phenomenon*. Elsevier, New York, NY.
Vanhanen, T. (2003). *Democratization: A Comparative Analysis of 170 Countries*. Routledge, New York, NY.
Van Leeuwen, F., Park, J. H., Koenig, B. L. et al. (2012). Regional variation in pathogen prevalence predicts endorsement of group-focused moral concerns. *Evolution and Human Behavior* 33: 429–437.
Wilson, E. O. (1971). *The Insect Societies.* Belknap Press of Harvard University Press, Cambridge, MA.
Wilson, E. O. (1998). *Consilience: The Unity of Knowledge*. Knopf, New York, NY.
World Bank. (2008). http://data.worldbank.org/topic/education.
World Cultures. http://www.worldcultures.org.
World Health Organization. (2004). *Global Burden of Disease: 2004 Update,* http://www.who.int. World Health Organization, Geneva, Switzerland.