Evolutionary Psychology

www.epjournal.net – 2013. 11(1): 263-280

Original Article

Guns, Germs, and Stealing: Exploring the Link Between Infectious Disease and Crime

Ilan Shrira, Department of Psychology, Loyola University, Chicago, IL, USA. Email: ishrira@luc.edu (Corresponding author).

Arnaud Wisman, School of Psychology, University of Kent, Canterbury, Kent, UK.

Gregory D. Webster, Department of Psychology, University of Florida, Gainesville, FL. USA.

Abstract: Can variation in crime rates be traced to the threat of infectious disease? Pathogens pose an ongoing challenge to survival, leading humans to adapt defenses to manage this threat. In addition to the biological immune system, humans have psychological and behavioral responses designed to protect against disease. Under persistent disease threat, xenophobia increases and people constrict social interactions to known in-group members. Though these responses reduce disease transmission, they can generate favorable crime conditions in two ways. First, xenophobia reduces inhibitions against harming and exploiting out-group members. Second, segregation into in-group factions erodes people's concern for the welfare of their community and weakens the collective ability to prevent crime. The present study examined the effects of infection incidence on crime rates across the United States. Infection rates predicted violent and property crime more strongly than other crime covariates. Infections also predicted homicides against strangers but not family or acquaintances, supporting the hypothesis that in-group–out-group discrimination was responsible for the infections–crime link. Overall, the results add to evidence that disease threat shapes interpersonal behavior and structural characteristics of groups.

Keywords: aggression, crime, homicide, infectious disease, pathogens

Introduction

Infectious diseases have some thorny repercussions. Besides the costs they impose on health, quality of life and economic resources, research suggests disease risk also increases interpersonal distrust, prejudice, and aggression. In this paper, we argue that infectious diseases are relevant to a particular type of interpersonal aggression—crime.

First, we review evidence that infection severity affects interpersonal perception and group dynamics, then discuss how these reactions promote aggression and crime. Finally, we present data that tests these ideas.

Infection Severity and Culture

Infections (i.e., pathogens, parasites) have been an ever-present threat to human survival and well-being, and the biological immune system has evolved as our foremost defense against this threat. Immune defenses are especially adept at protecting the body against parasites found in our local environment, but leave us vulnerable to foreign infections that have the potential to inflict serious harm (Diamond, 1997). Because exotic pathogens can invade new environments at any time and spread quickly, people need strategies of limiting their exposure to infections before they can do any damage.

For this, our first line of defense is the *behavioral immune system* (Schaller and Park, 2011), a suite of psychological and behavioral mechanisms that evolved alongside the classic immune system, designed to detect infections in the environment and protect against their transmission. For example, an elevated infection risk triggers hypersensitivity to disease-relevant cues, and produces aversive feelings and behavioral avoidance of potential disease carriers (Stevenson, Case, and Oaten, 2011). Despite the importance of these immune defenses, however, they cannot operate all the time. Immune system activation is physiologically costly, and in many situations avoidant responses are counterproductive (e.g., when mating, exploring the environment). Thus, like many adaptive modules shaped by natural selection, behavioral immune processes are sensitive to environmental inputs, mobilizing our attention and resources when disease risk is high but standing down when threats are minimal (Cosmides and Tooby, 1992; Fleischman et al., 2011; Schaller, 2011).

Another anti-pathogen strategy, and one that is the focus of this research, is to be wary of unfamiliar people (e.g., xenophobia), who pose a greater disease risk. Outsiders are more likely to carry harmful infections to which local residents are not immune (McNeill, 1976), and less likely to conform to local customs which often serve as buffers against disease transmission (e.g., food preparation practices, medical treatment, hygienic and sexual behaviors; Navarrete, Fessler, and Eng, 2007). For these reasons, unfamiliar people should represent a serious threat when disease susceptibility is already elevated.

Consistent with this reasoning, people who perceive themselves as vulnerable to disease harbor more negative sentiments against culturally unfamiliar immigrants (Faulkner, Schaller, Park, and Duncan, 2004; Navarrete and Fessler, 2006). Experiments show that exposing people to infection-related imagery evokes negative attitudes toward immigrants and stronger avoidant responses toward strangers (Faulkner et al., 2004; Mortensen, Becker, Ackerman, Neuberg, and Kenrick, 2010). Hence, activating the immune system elicits out-group avoidance, which may serve to minimize disease exposure. Additionally, behavioral immune responses go a step further by *bracing* for potential disease contraction. Specifically, pathogen threats strengthen in-group affiliation and solidarity (e.g., ethnocentrism, closeness to family), which creates a supportive network should someone in the group become sick (Navarrete and Fessler, 2006).

When a discriminatory social orientation is pervasive across many individuals, the consequences can be seen at the larger group level. For instance, cultures high in parasite

stress are more collectivistic (Fincher, Thornhill, Murray, and Schaller, 2008), where people tend to be less trusting of out-groups, discriminate more readily between in-group and out-group members (Triandis, 1995), and maintain stronger ties to in-groups (Fincher and Thornhill, 2012). Further, as people restrict their interactions to in-group members and insulate themselves from outsiders, social networks become smaller and more disconnected from one another (Fincher and Thornhill, 2008). This fractious dynamic not only exacerbates inter-group distrust, it reduces people's sense of connection to a larger community identity. Over time, perhaps under persistent disease threat, this social organization fosters norms that lead people to care little about those beyond their own in-group (Thornhill, Fincher, and Aran, 2009).

Pathogens and Aggression

Extreme in-group–out-group discrimination has a further implication: it produces conditions that facilitate aggression (Letendre, Fincher, and Thornhill, 2010; Thornhill et al., 2009). Xenophobia, ethnocentrism, and social distancing reduce moral concern for out-group members, and in extreme cases, lead to their dehumanization (Bandura, 1999; Navarrete and Fessler, 2006). Xenophobia and ethnocentrism foster antipathy and perceived superiority over others, while social distancing depersonalizes out-groups so that moral prescriptions will no longer apply to them. Moral detachment and dehumanization in turn weaken inhibitions against harming others and reduce people's guilt over doing so. For example, when faced with a choice of how to resolve opposing interests, people who distrust one another will be predisposed to pursue aggressive (e.g., violent) solutions over cooperative ones.

Letendre et al. (2010) found that pathogen severity predicted the frequency of intrastate political violence, especially smaller skirmishes between groups. Thornhill and Fincher (2011) extended these results to the realm of inter*personal* aggression. They hypothesized that because parasite stress elicits collectivism, both of these factors would aggravate interpersonal conflicts and elevate the likelihood of lethal aggression. Using U.S. states as their unit of analysis, they hypothesized that infection severity and collectivism would increase the incidence of: (a) romantic partner homicides, (b) argument-related homicides between male acquaintances, and (c) felony-related homicides.

Regarding romantic partner homicides, Thornhill and Fincher (2011) pointed out that partner violence is elevated under collectivistic values because these values tend to uphold gender inequalities, patriarchal norms, and greater acceptability of violence against women who violate these norms. These features of collectivism can be considered expressions of ethnocentrism and xenophobia, and are therefore relevant to pathogen severity, which should exacerbate conflicts and increase the risk that they escalate into homicide.

Collectivistic societies also hold stronger honor norms, which attach greater import to one's reputation, and for men, hyper-masculine gender expectations. In honor cultures, a man's esteem is tied to his tough-guy image and his ability to defend his family and property. This causes men to be more sensitive to insults from others, which are usually perceived as threats to their masculine persona (Vandello and Cohen, 2003). Many partner homicides are initiated when a woman threatens her partner's image and the man attempts

to reassert control over the woman in response to this. Strong honor norms are also central to argument-related homicides between male acquaintances, often triggered by a potential loss of face (e.g., public insults, cuckoldry) by one man and the unwillingness of both men to back down from the confrontation (Nisbett and Cohen, 1996). Similarly, felony-related homicides sometimes occur after an exchange of insults between males (who are typically strangers) in the context of a less serious crime.

Thus, in all of these encounters, the perceived threat posed by the other person is elevated under collectivistic values, and should be still greater under parasite stress, which amplifies interpersonal divisions and increases the risk these encounters will lead to deadly violence. Thornhill and Fincher (2011) found that infection severity and collectivism (examined separately) each predicted homicides between partners, male acquaintances, and strangers in the context of a felony.

The Present Research

We built on Thornhill and Fincher's (2011) thesis that parasite stress can increase aggression, with an approach designed to test a few novel hypotheses. First, because parasite stress increases xenophobia, we expected aggression to be directed toward unfamiliar rather than familiar people (i.e., strangers as opposed to known others). Therefore, we distinguished homicides based on the perpetrator–victim relationship, examining those between family members, acquaintances, and strangers. We expected pathogen severity to predict homicides against strangers but not against family or acquaintances. If this hypothesis is supported, it would suggest that the link between pathogen and aggression applies primarily to unfamiliar out-groups.

Second, we assume that infection severity is relevant not only to homicide, but to crime more generally. If persistent disease threat erodes concern for others' welfare, then there should be fewer qualms about harming and exploiting others (e.g., committing violent and property crimes against them) regardless of the context. Hence, we looked at the influence of pathogen severity on all types of felony crimes.

Finally, our study differed methodologically from Thornhill and Fincher's, which used one control variable (i.e., the Gini index) when testing how infections and collectivism (separately) predicted homicide. Though we did not examine collectivism, we did include an assortment of economic and demographic control variables that have been previously linked to crime, to help rule out the effect of other third variables (Moody and Marvell, 2010). Although standard U.S. crime data do not provide information on the perpetrator–victim relationship disaggregated across different geographic units, homicide records do. Therefore, homicide records enabled us to test whether relationship type—family, acquaintances, or strangers—moderated the effect of pathogens on crime across states.

Out-group derogation, however, by itself does not provide a compelling causal explanation for higher crime rates—such a process would require strong intervening forces to produce an outcome as extreme as crime. We propose that this connection can be best understood, at a proximate level, in terms of a social disorganization theory of crime. Social disorganization theory asserts that strong relationships and mutual trust among residents of an area enable them to collectively achieve common goals, such as crime prevention (Sampson, Raudenbush, and Earls, 1997). Tightly knit residents maintain strong

informal social control over crime; for example, they are more likely to monitor and control teenage peer groups, organize neighborhood watch programs, and communicate with each other when potential trouble arises (Messner, Baumer, and Rosenfeld, 2004). In contrast, when residents feel estranged from one another and do not look out for each other's interests, the collective ability to solve problems all but vanishes. Residents view one another, even neighbors, as part of a generalized out-group (Charles, 2003). We believe these conditions resemble the fractious, distrusting dynamics caused by infection severity (e.g., Price-Smith, 2009). This makes social disorganization theory a useful lens for understanding how pathogen severity and discriminatory sociality can lead to crime.

Materials and Methods

Our pathogen measure for each state was based on the incidence of common infections aggregated over the years 1995–1999, and crime rates from 2000–2007. Our regression analyses also contained a set of control variables, described below.

Felony Crimes

Crime records were taken from the Uniform Crime Reports (UCR; Federal Bureau of Investigation, 2009). We examined the rate of violent crimes (aggravated assault, robbery, rape, homicide) and property crimes (larceny, motor vehicle theft, burglary). Crime rates were computed by taking the total number of crime incidents in a state from 2000 to 2007, and dividing this value by the state's aggregated population over those eight years (U.S. Bureau of the Census, 2010). The homicides in the UCR encompassed those across all perpetrator–victim relationships.

Homicide by Perpetrator–Victim Relationship

The Supplementary Homicide Reports are a subsystem of the UCR that report detailed characteristics of homicides (Fox and Swatt, 2009), including the perpetrator–victim relationship. Consistent with previous research (e.g., Kovandzic, Vieraitis, and Yeisley, 1998), separate rates were calculated for family, acquaintance, and stranger homicides, using the homicide count as the numerator and the total population as the denominator (per 100,000). Family members included spouses and other relatives (e.g., parents, children, siblings); acquaintances were non-family members who had some connection to one another (e.g., neighbors, friends, co-workers); strangers made up all other relationships. Homicide rates were normalized using a natural log transformation to correct for positive skew. The Supplementary Homicide Reports did not have data for Florida over these years, so our results are based on the other 49 states.

Pathogen Severity

Our pathogen index reflected the most common infections reported in the Centers for Disease Control and Prevention's (CDC) National Notifiable Diseases Surveillance System (CDC, 2010). Only diseases with over 20,000 new cases in at least one of the five years examined (1995–1999) were used. This criterion left us with eight diseases: AIDS, Chlamydia, gonorrhea, syphilis, Hepatitis A, salmonellosis, shigellosis, and tuberculosis.

These eight diseases accounted for over 90% of all the infection counts reported in this system over these years; the incidence of the other diseases (e.g., cholera, malaria) tended to be very low.

First, we calculated a pooled rate for each of the eight diseases. For each one, we counted all new cases of a disease over the five-year period, and divided this total by the state's (pooled) population over the five years (U.S. Bureau of the Census, 2000). The five-year pooled rate for each disease was then standardized (*z*-scored) across states, and these eight values were averaged together to produce an overall standardized infection rate for each state. There was a high degree of overlap in the rates of the eight diseases, with the index exhibiting good internal reliability (α = .77; see Fincher and Thornhill, 2012, for a different state-level disease index).[1] To make the numbers more interpretable and to avoid negative values, we transformed them by multiplying 20 then adding 50 (scores are listed in the Appendix). New York State did not have complete records for Chlamydia incidence during these years, so the index for NY State was derived from the other seven diseases.

Control Variables

We sought to include a broad range of control variables in our analyses, so we used the variables from the classic Land et al. (1990) homicide study. These variables were originally selected by Land et al. because they were the most common co-variates of crime examined in the literature (see also McCall, Land, and Parker, 2010). These included (a) resource deprivation (defined below), (b) population structure (also defined below), (c) unemployment rate, (d) percentage of the population aged 15–29 years, (e) the percentage of males over 15 who are divorced (vs. not divorced), and (f) a dichotomous variable for the Southern U.S. Census region. All our variables reflected either the year 1999 or 2000 (Minnesota Population Center, 2004; U. S. Bureau of Labor Statistics, 2000; U.S. Bureau of the Census, 2000, 2005).

Resource deprivation. One way to minimize the problem of multicollinearity is to combine predictors that have a high degree of overlap into a single composite measure. Accordingly, Land et al. (1990) created their resource deprivation index from five variables: median family income, the Gini index for families, the percentage of families below the poverty line, the proportion of residents who are black or African-American, and the proportion of children not living with both parents. They conducted principal components analyses to show that these variables all loaded on the same factor.

When running a principal component analysis on our data, we found median family income did not load with the other four variables. These four variables had high factor loadings together, so we excluded median income and combined the other four as our

[1] Fincher and Thornhill's (2012) index included counts of many of the rarer infections from this surveillance system. However, the number of different infection types that made up their index varied from year to year (range: 23–50), depending on whether all 50 states reported that particular infection count to the CDC that year. Our index was highly correlated with Fincher and Thornhill's (*r* = .87).

resource deprivation measure. Higher values reflected greater resource deprivation (see Table 1 for principal component analysis).

Population structure. This variable was a composite of total state population and population density (both log–transformed).

Table 1. Principal components analysis for Resource Deprivation factors

	Factor Score
Percentage families in poverty	.797
Gini coefficient	.844
Percentage Black/African-American	.815
Percentage children not living with both parents	.924

Results

Pathogen severity and the six control variables were entered as predictors of each crime and homicide rate. Results for the felony crimes and homicides are reported separately because these data came from different sources.

Felony Crimes

Table 2 shows the zero-order correlations between all the predictors and violent and property crime rates. Pathogen severity was positively correlated with both categories of crime.

Table 3 reports the regression results. Pathogens were the strongest predictor of violent and property crimes, as well as most of the individual crimes. Despite some of the high inter-correlations among the predictor variables, the largest variance inflation factor (VIF) in all our analyses was 5.3 (tolerance = 0.19), which was well below the value of 10, above which indicates multicollinearity could threaten the validity of the results (Cohen, Cohen, West, and Aiken, 2003; Neter, Wasserman, and Kutner, 1989).

Because resource deprivation was highly correlated with pathogen severity, though, we ran multiple regression analyses on violent and property crime rates both with and without resource deprivation as a predictor variable. We found pathogen severity was positively associated with both violent and property crimes regardless of whether resource deprivation was included as a predictor. Table 3 reports these additional regressions with deprivation excluded from the analysis.

Pathogen severity was weakly but significantly (p = .049) related to the homicide rate. However, the UCR dataset did not allow us to disaggregate the results by perpetrator–victim relationship, whereas the Supplementary Homicide Reports did.

Table 2. Zero-order correlations of predictors with violent and property crime

	Violent crime	Property crime	Pathogen severity	Resource deprivation	Population structure	Unemploy rate	% 15-29	% men divorced
Property crime	.57**	—						
Pathogen severity	.72**	.55**	—					
Resource deprivation	.59**	.35*	.80**	—				
Population structure	.35*	.08	.50**	.45**	—			
Unemployment rate	.32*	.31*	.24	.43**	-.09	—		
% population 15-29	-.01	.33*	.18	.03	-.31*	.15	—	
% men divorced	.12	.33*	-.22	.01	-.44**	.33*	-.04	—
Southern region	.43**	.29*	.53**	.67**	.24	.12	.09	.09

Notes: *$p < .05$; **$p < .01$.

Table 3. Regression results for felony crimes

Predictor variable	Violent crime	Violent crime excluding deprivation	Property crime	Property crime excluding deprivation	Aggravated Assault	Robbery	Rape	Homicide	Larceny	Car theft	Burglary
Pathogen severity	.95***	.76***	.96***	.65***	.86***	.85***	.61**	.32*	.90***	1.04***	.57***
Resource deprivation	-.37†	—	-.61**	—	-.13	-.38*	-.50†	.51*	-.69**	-.63**	-.11
Population structure	.13	.08	.23†	.14	-.03	.53***	-.30†	.14	.09	.41**	.28*
Unemployment	.18	.07	.12	-.05	.15	.15	.26†	-.001	.12	.16	.01
% 15-29	-.15	-.11	.25*	.30*	-.15	-.13	.04	.11	.26*	.06	.23*
% men divorced	.32**	.30*	.62***	.58***	.30*	.24**	.35*	.27*	.50***	.59***	.62***
Southern region	.10	-.02	.04	-.16	.15	-.01	-.01	-.04	.10	-.31*	.16
R^2	.66	.63	.67	.60	.53	.80	.41	.72	.53	.63	.72

Notes: Values are standardized betas. Violent crime is the aggregate of aggravated assault, robbery, rape, and homicide; property crime is the aggregate of larceny, car theft, and burglary.
†$p < .10$; *$p < .05$; **$p < .01$; ***$p < .001$

Homicide by Perpetrator–Victim Relationship

The zero-order correlations between the predictor variables and each type of homicide are shown in Table 4.

Regression results appear in Table 5. Pathogen rate was not significantly related acquaintance homicides (p = .18) and only marginally related to family homicides (p = .09), but consistent with our hypothesis, pathogens strongly predicted homicides against strangers. These findings bolster the claim that aversion to out-groups explains the relationship between infection severity and crime.

Table 4. Zero-order correlations of predictors with homicide types across perpetrator-victim relationship

	Family homicide	Acquaintance homicide	Stranger homicide
Acquaintance homicide	.90**	—	
Stranger homicide	.67**	.77**	—
Pathogen severity	.46**	.57**	.76**
Resource deprivation	.56**	.68**	.67**
Population structure	-.03	.14	.43**
Unemployment rate	.41**	.44*	.33*
% population 15-29	.29*	.24	.10
% men divorced	.48**	.34*	.03
Southern region	.57**	.52**	.45**

Notes: Intercorrelations of the predictor variables are reported in Table 2.
*p < .05; **p < .01

Parallel Analyses with Fincher and Thornhill's (2012) Pathogen Index

We sought to verify that our results were comparable when using Fincher and Thornhill's (2012) pathogen index, so we substituted their index with ours for all our regression analyses. Most of the results were similar. Their pathogen index continued to predict violent crime (β = .66, p < .01) and property crime (β = .59, p < .01). Like ours, their index was also related to stranger homicides (β = .43, p < .05) and unrelated to family homicides (β = .27, *ns*). However, their measure was associated with acquaintance homicides (β = .40, p < .05), whereas our pathogen index was not. We discuss this discrepancy below.

Table 5. Regression results for homicides

	Family Homicide	Acquaintance Homicide	Stranger Homicide
Pathogens	.30†	.24	.75***
Resource deprivation	.08	.36†	-.17
Population Structure	.03	.12	.31*
Unemployment	.07	.05	.14
% population 15-29	.18	.19†	-.01
% men divorced	.51***	.44***	.33**
Southern region	.29*	.07	.07
R^2	.66	.66	.69

Notes: Values are standardized betas.
†$p < .10$; *$p < .05$; **$p < .01$; ***$p < .001$

Discussion

In-group favoritism and out-group distancing are ubiquitous features of social life, and this is especially true when parasite stress is elevated. Disease threat activates responses that function to guard against the threat, such as out-group avoidance and in-group preference. When these responses are widespread among many individuals—for example, in places where infection risk is chronically elevated—they foster xenophobia and produce a fragmented social structure that increases the potential for out-group aggression (Letendre et al., 2010). We found that infection rates explained a substantial proportion of the variance in violent and property crimes, and in most cases was a stronger predictor than established crime covariates. Interestingly, the homicide results showed that the aggression was directed primarily at out-group members, with pathogens predicting stranger homicides more strongly than any of the control variables.

We believe these results contribute to our understanding of parasite stress in several ways. First, we conceptualize crime as an extreme consequence of xenophobia and ethnocentrism, in which aggression is directed toward unfamiliar out-groups. This account is distinct from Thornhill and Fincher's (2011) thesis, which theorized that pathogens increase homicides against both known and unknown others in honor-relevant contexts. A second contribution is our assertion that pathogen threats are relevant not just to homicide, but to crime in general. If parasite stress erodes moral concern for out-group members, people will have weaker self-restraints against harming them in any number of ways.

Finally, our inclusion of a broad set of control variables strengthened support for these hypotheses. These additional controls may have also been one reason for the inconsistency between our homicide results and Thornhill and Fincher's (2011), which linked pathogens to violence against intimates and acquaintances. For example, their

research found that infections predicted romantic partner homicides, whereas we found infections were unrelated to family homicides. We suspect our finding was null because our control variables explained the bulk of the variance in these homicides. If this was indeed the case, then we should expect that *excluding* these control variables from our analysis would yield a significant relationship between pathogens and family homicides. In a separate analysis, therefore, we regressed family homicide on our pathogen measure and the Gini index (Thornhill and Fincher's control variable). The result confirmed our hypothesis: family homicide was now significantly predicted by infection severity ($\beta = .43$, $p < .05$), though not the Gini ($\beta = .05$, *ns*). Taken together, this suggests that our additional control variables—not pathogens—better explained the variation in family homicide in our primary results.

When using Fincher and Thornhill's (2012) pathogen index, it predicted acquaintance homicides, whereas this relationship was non-significant with our pathogen measure. The reason for this discrepancy was unclear. We find it interesting, though, that the only discrepancy occurred for homicides against acquaintances, relationships that range from very close (e.g., one's best friends) to barely recognizable (e.g., co-workers with whom one has never spoken). This raises the larger question of how "in-group" and "out-group" are to be distinguished, a definition that has varied a great deal across research traditions. For example, in-group–out-group status has been defined in terms of perceived similarity (Dasgupta, Banaji, and Abelson, 1999; Daly and Wilson, 1997) and conflicting interests (Oates and Wilson, 2002; Sherif, Harvey, White, Hood, and Sherif, 1961). Our distinction was based on degree of familiarity, an attribute considered a simple but powerful heuristic about whom to trust and whom to avoid (Zajonc, 1980). Nevertheless, the conflicting results for acquaintance homicide suggest that our distinction between stranger and non-stranger might be too absolute; instead, a graded degree of familiarity is likely a better way to characterize in-group–out-group status.

Comparison to other theories

Our findings should also be considered in the context of other theories that invoke evolutionary mechanisms to understand crime. For example, Daly and Wilson (1988) view homicide as a marker of heightened competition between men over status, material resources, and ultimately, over reproductive opportunities. Because male reproductive success is enhanced by attaining status and resources relative to other men, greater resource inequities should intensify competition, and Daly and Wilson have shown that societal income inequality (e.g., the Gini index) is one of the best predictors of homicide between unrelated males (Daly and Wilson, 1988). Competition should be especially fierce in men who lack resources, and the demographic profile of criminals supports this idea—criminals are more likely to be young, low-resource, unmarried men. Not surprisingly, aggression decreases substantially once men get married, when their reproductive prospects are no longer a concern.

Barber (2008) also argues that violent crime is a marker of aggressive competition that functions to improve fitness. Using a social learning framework, Barber proposes that harsh rearing environments—such as poverty, lack of parental investment—tend to socialize hyper-competitive behavior in children, a phenotype that will ultimately boost

fitness under similarly harsh conditions they are likely to encounter as adults (Belsky, Steinberg, and Draper, 1991). However, an aggressive disposition also increases the likelihood a person will commit crimes.

Our analyses controlled for some of the presumed causes of the elevated competition—income inequality, single-parent households, other resource variables—suggesting that other forces were also contributing to crime. Rather than viewing competition as a proximate explanation, we believe crime is rooted in the xenophobia and out-group distancing that serves as adaptive responses to pathogens. At the proximate level, this connection between pathogens and crime make sense in the context of social disorganization theory, which shows that crime is pervasive when social trust and communal bonds between people are weak (Kubrin and Weitzer, 2003). Distrust and weak communal bonds correspond to the symptoms of parasite stress. Thus, we propose that pathogens produce cultural dynamics similar to social disorganization (Price-Smith, 2009), which in turn increases the likelihood of crime against unknown others. One implication of this model is that social disorganization theory may be a better framework for understanding crime against strangers than non-strangers (see Sampson, 1987). Furthermore, these two types of crimes may be caused by very different factors.

Conclusions

Overall, the costs of infectious disease are steeper than they appear to be. They not only compromise health and survival, they also erode any collective benefit that depends on trust as its currency. Given such far-reaching consequences, infection control efforts may deserve an even higher priority than they are already given (Powell, Clarke, and Savulescu, 2012; Thornhill et al., 2009). We also need a clearer understanding of pathogen-avoidant responses in order to integrate them into the disease modeling of epidemics and optimize public health responses to outbreaks (Durham and Casman, 2012).

Despite the damage inflicted by pathogens in the past, there are grounds for optimism. The control over infectious diseases in the modern world has been one of the triumphs of the 20th century. Over these years, the U.S. mortality rate from infections decreased by a magnitude of 13 (Armstrong, Conn, and Pinner, 1999), and today, fewer than 6% of all deaths are attributed to them (CDC, 2009). In an entirely different realm, another reason for optimism has been the progressive decline of violence in society. In recent centuries and even decades, the world has seen dramatic decreases in wartime casualties, homicide, capital punishment, ritual sacrifices, torture, slavery, child abuse, and domestic violence, while pacifism has established itself as a movement. This trend is due in part to the growing sphere of individuals with whom we have come to empathize and deem worthy of moral consideration (Pinker, 2011; Singer, 1981). The promotion of human rights has generated greater consideration for others regardless of race, gender, nationality, lifestyle, disability, and even species. Put another way, our in-group has grown larger while our out-groups have shrunk. These two trends—the decreases in infection severity and societal violence—may simply be coincidental, each a natural byproduct of modernization. Yet we wonder whether these changes could also have some causal connection to one another, a hypothesis that is well worth a closer examination.

Received August 16, 2012; Revision submitted February 13, 2013; Accepted February 16, 2013

References

Armstrong, G. L., Conn, L. A., and Pinner, R. W. (1999). Trends in infectious disease mortality in the United States during the 20th century. *Journal of American Medical Association, 281*, 61–66.

Bandura, A. (1999). Moral disengagement in the perpetration of inhumanities. *Personality and Social Psychology Review, 3,* 193–209.

Barber, N. (2008). Evolutionary social science: A new approach to violent crime. *Aggression and Violent Behavior*, *13*, 237-250.

Belsky, J., Steinberg, L., and Draper, P. (1991). Childhood experience, interpersonal development, and reproductive strategy: An evolutionary theory of socialization. *Child Development*, *62*, 647-670.

Centers for Disease Control and Prevention. (2009). *Compressed Mortality File, 2005* [database online]. Atlanta, GA: Centers for Disease Control and Prevention. CDC Wonder. Available at: http://wonder.cdc.gov/mortSQL.html.

Centers for Disease Control and Prevention. (2010). *Summary of notifiable diseases–United States, 1995–1999*. Atlanta, GA: Centers for Disease Control and Prevention. Available at: http://www.cdc.gov/mmwr/summary.html.

Charles, C. Z. (2003). The dynamics of racial residential segregation. *Annual Review of Sociology*, 167-207.

Cohen, J., Cohen, P., West, S. G., and Aiken, L. S. (2003). *Applied multiple regression/correlation analysis for the behavioral sciences* (3rd ed.). Mahwah, NJ: Erlbaum.

Cosmides, L., and Tooby, J. (1992). Cognitive adaptations for social exchange. In J. H. Barkow, L. Cosmides, and J. Tooby (Eds.), *The adapted mind: Evolutionary psychology and the generation of culture* (pp. 163-228). New York: Oxford University Press.

Daly, M., and Wilson, M. (1988). *Homicide*. New York: Aldine de Gruyter.

Daly, M., and Wilson, M. (1997). Crime and conflict: Homicide in evolutionary psychological perspective. *Crime and Justice*, 22, 51-100.

Dasgupta, N., Banaji, M. R., and Abelson, R. P. (1999). Group entitativity and group perception: Associations between physical features and psychological judgment. *Journal of Personality and Social Psychology*, *77*, 991-1003.

Diamond, J. (1997). *Guns, germs, and steel: The fates of human societies*. New York: Norton.

Durham, D. P., and Casman, E. A. (2012). Incorporating individual health-protective decisions into disease transmission models: a mathematical framework. *Journal of the Royal Society Interface, 9*, 562–570.

Faulkner, J., Schaller, M., Park, J. H., and Duncan, L. A. (2004). Evolved disease-avoidance mechanisms and contemporary xenophobic attitudes. *Group Processes and Intergroup Relations, 7*, 333–353.

Federal Bureau of Investigation. (2009). *Uniform crime reports for the United States 2000–2007*. Washington, DC: Government Printing Office.

Fincher, C. L., and Thornhill, R. (2008). Assortative sociality, limited dispersal, infectious disease and the genesis of the global pattern of religious diversity. *Proceedings of the Royal Society B: Biological Sciences, 275*, 2587–2594.

Fincher, C. L., and Thornhill, R. (2012). Parasite-stress promotes in-group assortative sociality: The cases of strong family ties and heightened religiosity. *Behavioral and Brain Sciences, 35*, 1–19.

Fincher, C. L., Thornhill, R., Murray, D. R., and Schaller, M. (2008). Pathogen prevalence predicts human cross-cultural variability in individualism/collectivism. *Proceedings of the Royal Society B: Biological Sciences, 275*, 1279–1285.

Fleischman, D. S., Webster, G. D., Judah, G., de Barra, M., Aunger, R., and Curtis, V. A. (2011). Sensor recorded changes in rates of hand washing with soap in response to the media reports of the H1N1 pandemic in Britain. *BMJ Open, 1,* e000127.

Fox, J. A., and Swatt, M. L. (2009). *Uniform crime reports [United States]: Supplemental Homicide reports with multiple imputations, cumulative files, 1976-2007* [Computer file]. Ann Arbor, MI: Inter-university Consortium for Political and Social Research. Available at: http://icpsr.umich.edu.

Kovandzic, R. V., Vieraitis, L. M., and Yeisley, M. R. (1998). The structural covariates of urban homicide: Re-assessing the impact of income inequality and poverty in the post-Reagan era. *Criminology*, *36*, 569–600.

Kubrin, C. E., and Weitzer, R. (2003). New directions in social disorganization theory. *Journal of Research in Crime and Delinquency, 40*, 374–402.

Land, K. C., McCall, P. L., and Cohen, L. E. (1990). Structural covariates of homicide rates: Are there any invariances across time and social space? *American Journal of Sociology, 95*, 922–963.

Letendre, K., Fincher, C. L., and Thornhill, R. (2010). Does infectious disease cause global variation in the frequency of intrastate armed conflict and civil war? *Biological Reviews, 85*, 669–683.

McCall, P. L., Land, K. C., and Parker, K. F. (2010). An empirical assessment of what we know about structural covariates of homicide rates: a return to a classic twenty years later. *Homicide Studies*, *14*, 221–245.

McNeill, W. H. (1976). *Plagues and people*. New York: Anchor.

Messner, S. F., Baumer, E. P., and Rosenfeld, R. (2004). Dimensions of social capital and rates of criminal homicide. *American Sociological Review*, *69*, 882-903.

Minnesota Population Center. (2004). *National Historical Geographic Information System: Pre-Release Version 0.1*. Minneapolis: University of Minnesota. Available at: http://www.nhgis.org.

Moody, C. E., and Marvell, T. B. (2010). On the choice of control variables in the crime equation. *Oxford Bulletin of Economics and Statistics*, *72*, 696-715.

Mortensen, C. R., Becker, D. V., Ackerman, J. M., Neuberg, S. L., and Kenrick, D. T. (2010). Infection breeds reticence: The effects of disease salience on self-perceptions of personality and behavioral tendencies. *Psychological Science, 21*, 440–447.

Navarrete, C. D., and Fessler, D. M. T. (2006). Disease avoidance and ethnocentrism: The effects of disease vulnerability and disgust sensitivity on intergroup attitudes. *Evolution and Human Behaviour, 27*, 270–282.

Navarrete, C. D., Fessler, D. M. T., and Eng, S. J. (2007). Elevated ethnocentrism in the first trimester of pregnancy. *Evolution and Human Behavior, 28,* 60–65.

Neter, J., Wasserman, W, and Kutner, M. H. (1989). *Applied linear regression models* (2nd ed.). Homewood, IL: Irwin.

Nisbett, R. E., and Cohen, D. (1996). *Culture of honor: The psychology of violence in the South*. Westview Press.

Oates, K., and Wilson, M. (2002). Nominal kinship cues facilitate altruism. *Proceedings of the Royal Society of London, Series B: Biological Sciences*, *269*, 105-109.

Pinker, S. (2011). *The better angels of our nature: Why violence has declined*. New York: Viking Adult.

Powell, R., Clarke S., and Savulescu, J. (2012). An ethical and prudential argument for prioritizing the reduction of parasite-stress in the allocation of health care resources. *Behavioral and Brain Sciences, 35*, 90–91.

Price-Smith, A. T. (2009). *Contagion and chaos: Disease, ecology and national security in the era of globalization*. Cambridge: MIT Press.

Sampson, R. J. (1987). Personal violence by strangers: An extension and test of the opportunity model. *The Journal of Criminal Law and Criminology, 78,* 327–356.

Sampson, R. J., Raudenbush, S., and Earls, F. (1997). Neighborhoods and violent crime: A multi-level study of collective efficacy. *Science, 277,* 918–924.

Schaller, M. (2011). The behavioural immune system and the psychology of human sociality. *Proceedings of the Royal Society B: Biological Sciences, 366*, 3418–3426.

Schaller, M., and Park, J. H. (2011). The behavioral immune system (and why it matters). *Current Directions in Psychological Science, 20*, 99–103.

Sherif, M., Harvey, O. J., White, B. J., Hood, W. R., and Sherif, C. W. (1961). *Intergroup conflict and cooperation: The Robbers Cave experiment* (Vol. 10). Norman, OK: University Book Exchange.

Singer, P. (1981). *The expanding circle: Ethics and sociobiology*. Oxford: Oxford University Press.

Stevenson, R. J., Case, Y. I., and Oaten, M. J. (2011). Proactive strategies to avoid infectious disease. *Proceedings of the Royal Society B: Biological Sciences, 366*, 3361–3363.

Thornhill, R., and Fincher, C. L. (2011). Parasite stress promotes homicide and child maltreatment. *Philosophical Transactions of the Royal Society B, 366*, 3466–3477.

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

Tooby, J., and Cosmides, L. (1992). The psychological foundations of culture. In J. H. Barkow, L. Cosmides, and J. Tooby (Eds.), *The adapted mind: Evolutionary psychology and the generation of culture* (pp. 19-136). New York: Oxford University Press.

Triandis, H.C. (1995). *Individualism and collectivism*. Boulder: Westview Press.

U. S. Bureau of Labor Statistics. (2000). Unemployment rates for states. Available at: http://www.bls.gov/lau/lastrk00.htm.

U. S. Bureau of the Census. (2000). *Census of population. Vols. 1 and 2. Characteristics of the population*. Washington, DC: Government Printing Office. Available at: http://www.census.gov.

U.S. Bureau of the Census. (2005). *Table S4. Gini Ratios by State: 1999*. Washington, DC: U.S. Department of Commerce. Available at: http://www.census.gov/hhes/www/income/data/historical/state/state4.html.

U. S. Bureau of the Census. (2010). Available at: http://www.census.gov.

Vandello, J. A., and Cohen, D. (2003). Male honor and female fidelity: Implicit cultural scripts that perpetuate domestic violence. *Journal of Personality and Social Psychology*, *84*, 997-1010.

Zajonc, R. B. (1980). Feeling and thinking: Preferences need no inferences. *American Psychologist*, *35*, 151-175

Appendix

Pathogen values for each state

State	Pathogen value
New Mexico	72.4
Mississippi	68.6
New York	66.1
Georgia	65.0
Louisiana	64.7
Texas	64.5
Maryland	64.1
Arizona	61.2
South Carolina	60.9
Tennessee	59.7
Delaware	59.5
California	57.6
Oklahoma	56.2
Florida	56.1
North Carolina	53.7
Hawaii	52.7
Illinois	51.7
Missouri	50.5
Alabama	50.3
Arkansas	48.2
New Jersey	47.9
Nevada	44.3
Virginia	43.3
Connecticut	42.8

Utah	42.3
Michigan	42.3
Rhode Island	41.8
Colorado	41.1
Oregon	40.8
Wisconsin	40.7
Massachusetts	40.5
Pennsylvania	40.2
Alaska	38.6
Ohio	38.2
Washington	38.1
Kentucky	36.6
Kansas	35.6
Nebraska	34.8
South Dakota	34.1
Indiana	33.1
Idaho	32.4
Iowa	31.4
Minnesota	30.4
Wyoming	28.6
Montana	28.0
North Dakota	26.5
Vermont	24.0
West Virginia	23.9
New Hampshire	23.2
Maine	20.4