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Variation and organization in social behavior : infectious disease and human intergroup conflict and warfare; and the organization of foraging behavior in harvester ants

Kenneth Letendre

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**VARIATION AND ORGANIZATION IN SOCIAL BEHAVIOR: INFECTIOUS
DISEASE AND HUMAN INTERGROUP CONFLICT AND WARFARE; AND
THE ORGANIZATION OF FORAGING BEHAVIOR IN HARVESTER ANTS**

BY

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DISSERTATION

Submitted in Partial Fulfillment of the
Requirements for the Degree of

Doctor of Philosophy

Biology

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DEDICATION

For my mother, Carol Letendre, whose unending curiosity about the world is reflected in my desire to learn and do as much as possible.

For my father, Bob Letendre, whose passion for life is reflected in my desire to experience as much of it as I can.

Thanks for making this possible.

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VARIATION AND ORGANIZATION IN SOCIAL BEHAVIOR: INFECTIOUS DISEASE AND HUMAN INTERGROUP CONFLICT AND WARFARE; AND THE ORGANIZATION OF FORAGING BEHAVIOR IN HARVESTER ANTS

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Abstract

Social behavior is an important contributor to the success of widely distributed animal taxa, including such distantly related taxa as humans and ants. There is variation in the features and organization of social systems based on ecological constraints and goals, as these alter the costs and benefits of social behaviors, and select for different optimal behaviors for social groups in different environments. I present two bodies of research: an effort to explain geographic variation in the frequency and intensity of human civil conflict; and an effort to describe and model the foraging behavior of colonies of harvester ants of the genus *Pogonomyrmex*. First, in an investigation of the geographic variation in the frequency of human civil conflict, I show that cross-nationally, intrastate armed conflicts are predicted positively by variation in the intensity of human infectious disease. I present a theoretical model to explain this variation that proposes that risk of exposure to novel infectious diseases imposes costs on intergroup social interaction, and in regions with high intensity of infectious disease these costs

create relative poverty and exacerbate conflict over resources. Second, I show that infectious disease predicts other categories of civil conflict, including clan wars, and revolutions and coups. I present a path analysis of the global peace index to test the plausibility of the hypothesized model, including the various indirect effects linking infectious disease to conflict; this analysis supports the importance of the relationship between infectious disease and conflict cross-nationally. Third, I present the results of a study using a computer model of host-parasite coevolution to examine the mechanisms by which localized host-parasite coevolutionary races impose fitness costs on host intergroup interaction, and lead to the evolution of out-group avoidance in mating decisions. In the fourth chapter I present a field study of the foraging behavior of three sympatric species of harvester ants, and their response to variation in heterogeneity in the distribution of foods. Fifth, I present a computer model of ant colony foraging behavior, which I optimized using genetic algorithms for different distributions of food, and whose resulting foraging behavior I compare to the previous observations of harvester ants in the field.

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CHAPTER 1

INTRODUCTION

Social behavior is an important contributor to the success of widely distributed animal taxa, including such distantly related taxa as humans and ants. There is variation in the features and organization of social systems based on ecological goals and constraints, as these alter the costs and benefits of social behaviors, and select for different optimal behaviors for social groups in different environments. Fundamentally, natural selection favors social living when the benefits exceed the costs of social behavior relative to solitary living; and it designs organisms that express social behaviors that maximize the benefit to cost ratio and maximize inclusive fitness given the organism's particular ecology.

I present two bodies of research on two widely distributed and highly successful, but taxonomically distant, social organisms. First is a study of the geographic distribution of the frequency and intensity of human civil conflict, and its relationship to the geographic distribution of infectious disease and intergroup attitudes. This research makes use of cross-national data to test a causal model of geographic variation in human conflict that results ultimately from the effects of infectious disease on the evolution of human social behavior. I also present an agent-based computer model with which explore the mechanisms by which host-parasite coevolutionary processes can lead to the expression of out-group avoidance, a key component in the link between infectious disease and intergroup conflict.

The second body of research is a study of foraging behavior of harvester ants of the genus *Pogonomyrmex*. This research includes field observations of colonies of harvester ants foraging on experimental baits; and computer modeling of mechanisms of organization of colony foraging activity, in which I optimize computer models for a range of colony sizes and food distributions to explore the ways these factors affect optimal colony foraging behavior; and finally comparison of the foraging behavior of optimized models to the behavior of ants in the field.

In Chapter 2, I present an analysis of cross-national variation in the frequency intrastate armed conflicts, including small-scale conflicts and large-scale civil wars. I propose a model to explain this variation that results ultimately from geographic variation in the intensity of human infectious disease. In warm, humid climates that are conducive to the transmission of infectious disease, the high burden of disease imposes risks and costs to intergroup social interaction. These risks and costs lead to the expression of xenophobic and ethnocentric intergroup attitudes in these places and, in addition to the direct costs of the morbidity and mortality of disease, leads to relative poverty because these xenophobic and ethnocentric groups are unwilling to cooperate or contribute to public goods that will be shared across groups. This ultimately results in increased frequency of intergroup conflict within nations when groups come into conflict over scarce resources and are unwilling to resolve these conflicts through cooperative means. I found that infectious disease predicts the frequency of infectious disease even when controlling for a battery of demographic, economic, and political variables. I report support for a direct relationship between infectious disease and the frequency of small-

scale intrastate armed conflicts, and support for an indirect relationship between infectious disease and the frequency of large-scale civil wars.

In Chapter 3, I extend my findings about the relationship of infectious disease and intrastate armed conflicts to other categories of within-state conflict, including non-state wars (or clan wars), revolutions and coups, and the Global Peace Index, a composite measure of conflict within nations. I found that the variation in the frequency of infectious disease predicts the frequency of non-state wars, revolutions and coups, and the Global Peace Index. I present a path analysis of the causal model relating infectious disease intensity to conflict, which supports the indirect effects of infectious disease on ethnocentrism and national wealth, and additionally suggests a further direct, positive effect of infectious disease on conflict.

In Chapter 4, I use a computer model of host-parasite coevolution to explore the mechanisms by which infectious disease selects for out-group avoidance, which is an important link in the relationship between infectious disease and inter-group conflict. I model a heterogeneous population of sexually reproducing hosts and their asexual parasites, with both host and parasite gene flow between sub-populations dependent on inter-group mating by hosts. Host immunity is under selection to detect and escape the locally common parasites, while parasites are under selection to evade detection and more effectively parasitize hosts; thus hosts and parasites are locked in an antagonistic coevolutionary race. I model the evolution of host mate choice preferences in this system, and find that hosts consistently evolve a preference for genetically dissimilar mates, but a bias against mates from outside the local subpopulation. Thus host-parasite

coevolutionary races can lead to the evolution and expression of out-group avoidance despite the advantages of out-breeding in these systems.

In Chapter 5, I present a field study of the foraging behavior of harvester ants of the genus *Pogonomyrmex*. I baited colonies of three sympatric species of harvester ants, which vary in colony size over an order of magnitude, with seeds in distributions varying in degree of heterogeneity. I recorded the time of arrival of bait seeds at the nest and analyzed how the rate of seed collection varies with colony size and with heterogeneity of the food distribution. I found that foraging rate varies with food distribution, with foraging rate roughly doubling as seed pile sizes are quadrupled; but, contrary to the hypothesis that larger colonies should make use of their larger forager population and more complex social structure to more effectively exploit heterogeneous food sources, I found that the treatment of heterogeneous foods relative to random foods does not vary with colony size.

In Chapter 6, I use a computer model of ant colony foraging to explore the ways in which colony size and food distribution affect the evolution and organization of social foraging in ants. I built an agent-based model of ant foraging and recruitment, and used genetic algorithms to optimize this model for a range of colony sizes and food distributions. I find that a common model of ant colony foraging and recruitment, in which colony foraging effort is thought to converge on the best foraging patches as a result of reduced search times in high quality patches, is sub-optimal in foraging for seeds as in harvester ants. Another model, in which successful foragers decide whether to lay a pheromone trail on the return trip to the nest based on local information about the

presence of other seeds, performs better, and empirically behaves more similarly to harvester ants in the field.

CHAPTER 2

DOES INFECTIOUS DISEASE CAUSE GLOBAL VARIATION IN THE FREQUENCY OF INTRASTATE ARMED CONFLICT AND CIVIL WAR?

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SUMMARY

Geographic and cross-national variation in the frequency of intrastate armed conflict and civil war is a subject of great interest. Previous theory on this variation has focused on the influence on human behaviour of climate, resource competition, national wealth, and cultural characteristics. We present the parasite-stress model of intrastate conflict, which unites previous work on the correlates of intrastate conflict by linking frequency of the outbreak of such conflict, including civil war, to the intensity of infectious disease across countries of the world. High intensity of infectious disease leads to the emergence of xenophobic and ethnocentric cultural norms. These cultures suffer greater poverty and deprivation due to the morbidity and mortality caused by disease, and as a result of decreased investment in public health and welfare. Resource competition among xenophobic and ethnocentric groups within a nation leads to

increased frequency of civil war. We present support for the parasite-stress model with regression analyses. We find support for a direct effect of infectious disease on intrastate armed conflict, and support for an indirect effect of infectious disease on the incidence of civil war *via* its negative effect on national wealth. We consider the entanglements of feedback of conflict into further reduced wealth and increased incidence of disease, and discuss implications for international warfare and global patterns of wealth and imperialism.

INTRODUCTION

Warfare accounts for a staggering amount of human mortality. From 1945 to 1999, interstate warfare resulted in over three million deaths. During that same period, civil wars resulted in over five times that number, more than 16 million deaths (Fearon & Laitin, 2003). This estimate of the carnage associated with civil war is based on immediate mortality; however, as Ghobarah, Huth & Russett (2003) put it, this is only the “tip of the iceberg.” Civil wars maim and kill many more, long after the shooting stops, through disability and disease (Ghobarah *et al.*, 2003). It is no wonder, then, that civil war is an area of intense interest for political scientists and behavioural researchers alike. However, warfare and civil war are not strictly modern phenomena. Estimates of the rates of mortality *per capita* as a result of warfare through human history indicate that as astonishing as the numbers mentioned above may be, throughout most of human history death directly attributable to warfare has been a more significant source of mortality than it was during the 20th Century (Keeley, 1996). As a result, selection has been intense for

the evolution of psychological and behavioural adaptations that minimize the costs of warfare and maximize the benefits (Bowles, 2009).

Although war is a ubiquitous feature of human societies (Keeley, 1996; Wrangham, 1996), there is large variation among societies in the frequency of warfare. Researchers interested in the causes of war have focused on these differences in order to study the features of societies and cultures that may lead to a greater or lesser frequency of warfare (e.g. Ember & Ember, 1992b; Fearon & Laitin, 2003; Schwartz, 1968; Van de Vliert *et al.*, 1999). Here, we review previous research on this variation, and propose and test a new theory, the parasite-stress theory, to explain cross-national variation in the frequency of intrastate armed conflict and civil war.

(1) Prior theory on intrastate armed conflict and civil war

Numerous scholars have noted geographic patterns in the frequency and intensity of violence and warfare. Some of these studies focus attention on particular geographic regions (e.g. Collier & Hoeffler, 2002; Elbawadi & Sambanis, 2000) while others focus attention on the effect of climatic variables, such as temperature (Anderson, 1989; Schwartz, 1968; Van de Vliert *et al.*, 1999) and rainfall and humidity (Schwartz, 1968). Schwartz's (1968) analysis of the relation of ecological variables with the frequency of political violence found highest levels of violence in countries with the most rainfall, highest humidity, and moderately warm temperatures. Van de Vliert *et al.* (1999) also report a positive curvilinear relationship between temperature and domestic political violence.

Schwartz (1968) investigated and discussed folk-wisdom as hypotheses for the relation between climate and violence, including: the “temperate weather hypothesis,”

which holds that people are generally more active in temperate weather, and are therefore naturally more likely to engage in violent activities as well; and the “long, hot summer hypothesis,” which holds that people are irritable, and therefore more aggressive, in uncomfortably hot weather. Although Schwartz's (1968) analysis provided some support for a “discomfort-irritability-aggression” effect, he expressed hope for more refined questions and hypotheses in future research. We object to these folk-wisdom hypotheses on the evolutionary theoretical grounds that as an important and costly feature of human political behaviour, variation among societies in the frequency of warfare is not likely to be merely a by-product of humans' preference for pleasant weather. As an extremely costly activity, warfare must have emerged as a human-typical behaviour by having provided benefits – in the form of increased survival and reproduction, i.e. inclusive fitness – that consistently outweighed those costs to individuals who engage in it. Fortunately, in the years since Schwartz's (1968) analysis, more refined hypotheses and analyses have emerged.

One well-established correlate of warfare is competition for resources. The connection between resource competition and war has been investigated and demonstrated in numerous samples, including samples from traditional societies (Durham, 1976; Ember & Ember, 1992b; Manson & Wrangham, 1991), as well as data on warfare in modern societies (Collier & Hoeffler, 2004; Fearon & Laitin, 2003; Van de Vliert *et al.*, 1999). From an evolutionary perspective, resource scarcity, whether measured by reported hunger in ethnographic samples (e.g. Ember & Ember, 1992b) or by relatively low Gross Domestic Product (GDP) *per capita* in modern societies (e.g. Fearon & Laitin, 2003), represents the same cause for warfare: limitation in resources

necessary for reproduction (and primarily for that of males, the primary or sole actors in warfare; Manson & Wrangham, 1991), which leads to potential benefits in intergroup aggression to seize resources or reduce competition for them. While in traditional societies these resources may be represented by goods which contribute more directly to reproduction (e.g. food sources, or brideprice goods such as livestock, women; Manson & Wrangham, 1991), studies of modern societies often focus on the more abstract *GDP per capita* as a general measure of resource availability within a society. Despite the more indirect link between *GDP per capita* and reproduction, nevertheless *GDP per capita* represents reproductive resources because, from an evolutionary perspective, all important resources are those that can be converted to reproductive success. (See Low, 2000, for a more general discussion of resources and human reproduction.)

Although the behaviours on which warfare depend must have been selected and maintained through greater inclusive fitness for individuals who participate in warfare (Durham, 1976), on the level of human populations, warfare has the effect of reducing competition by introducing an extra source of mortality. This operates in addition to the mortality caused by resource deprivation itself, acting as a feedback mechanism moderating the amount of resources available (Vayda, 1967; Zhang *et al.*, 2007).

Diamond (2005) implicates economic collapse, population pressure, and shortage of land in the civil war and genocide between Hutu and Tutsi factions in Rwanda in 1994. André & Platteau (1998) note, “It is not rare, even today, to hear Rwandans argue that a war is necessary to wipe out an excess of population and to bring numbers into line with the available land resources.”

Although resource competition is an important cause of war, it alone can not explain persistent variation in the frequency of warfare among societies. When human populations expand beyond the human carrying capacity of an area, the increased mortality caused by resource deprivation and resulting warfare should reduce the human population until an equilibrium is reached. With populations at the carrying capacity determined by their environments' particular availability of resources, warfare may occur periodically, but there is no theoretical reason to expect that it should happen more or less frequently in any society or in any particular region than in others. Elbawadi & Sambanis (2000) attribute the high frequency of civil wars in Africa to high levels of poverty and weak political institutions; Fearon & Laitin (2003) argue for similar causes for the global incidence of civil war in the latter half of the 20th Century. This explanation is not entirely satisfying because it leads to the question: why is there persistently more poverty, and thus more conflict, in some regions than in others? This is a question we will return to later.

Another predictor of warfare is resource unpredictability – as opposed to chronic resource scarcity – which Ember & Ember (1992b) suggest may be a better predictor of war than chronic resource scarcity, because it may produce anxiety over acute shortage, leading to inter-group aggression to alleviate or preempt it. In an analysis of the standard cross-cultural sample (Murdock & White, 1969), Ember & Ember (1992b) found that the occurrence of natural disasters (and resulting, non-chronic resource scarcity) positively predicts the occurrence of warfare. Moreover, they found that anxiety about the occurrence of natural disasters predicts warfare as well as their actual occurrence, in societies where people expressed worry about disasters to ethnographers who witnessed

no disasters during their data-collection period. Thus, perceived risk of future resource scarcity can be as potent a cause of warfare as current resource scarcity.

Zhang *et al.* (2007) performed an analysis of the effect of long-term temperature variation (and its effect on land carrying capacity) on the historical frequency of warfare and war-related mortality in China. They found that temporal decreases in mean temperature led to increased rates of warfare and mortality, as the carrying capacity of the region was reduced, leading to intensified resource competition. Interestingly, their finding is apparently contradictory to the generally recognized positive relationship across cultures between temperature and violence (Anderson, 1989). Thus there is reason to believe that different mechanisms may account for temporal *versus* geographic and cross-cultural variation in warfare frequency.

We propose that the most satisfying model for the frequency of warfare will be one that takes into account the environment's effect on evolved human psychology, and the adaptive expression of conditional motivations and behaviours suited to particular environments. There have been notable attempts to incorporate these elements into causal models of intergroup conflict. In addition to resource unpredictability, Ember & Ember (1992b) found that socialization for mistrust has a weaker, but significant influence on the occurrence of warfare. Under their model, real or anticipated acute shortages encourage intergroup aggression as groups attempt to alleviate or preempt shortage by the capture of resources from other groups. Meanwhile, mistrust of others, in part due to resource unpredictability and the occurrence of warfare, causes parents to socialize children to be mistrustful as well, and while this is partly caused by the

environment, according to Ember & Ember (1992b), it is an independent, significant cause of warfare.

While Ember & Ember's (1992b) model is a step in the right direction, the patterns of variation in frequency of civil war cross-culturally have not yet been explained fully. Their model fails to fully account for geographic variation in the frequency of civil war; its predictive power ultimately rests entirely in variation in resource unpredictability, but offers no explanation why this should vary systematically with geography or temperature. Why should hotter countries be poorer (Williamson & Moss, 1993) and experience more civil war on average, given a general negative correlation between latitude and net primary production (Field *et al.*, 1998) which, all other things being equal, should lead to the expectation that hotter countries are more resource abundant and wealthier (Templet, 1995)? Explanations for civil war that depend entirely on resource shortage or unpredictability are further contradicted by the argument that the need for intergroup cooperation in areas with high resource scarcity or unpredictability leads to peaceful intergroup relations, as seen, e.g. in the !Kung and Eskimos (Durham, 1976). What influences the decision to respond to resource deprivation with increased intergroup aggression rather than increased intergroup cooperation?

(2) The parasite-stress theory of intrastate conflict

The recently revealed association between cross-national variation in the unidimension collectivism-individualism and the severity of infectious diseases (Fincher *et al.*, 2008) suggests a model with the potential to integrate the diversity of published ideas and findings with respect to the incidence of civil war. Collectivism-individualism

is a cultural dimension that describes people's tendency to place individual goals above those of society or the group as a whole (individualism), or to see oneself as an integral part of a group whose goals supersede that of the individual (collectivism) (Hofstede, 2001). Individualist societies are characterized by individuals who look after themselves and their immediate, nuclear families, have many, but less intimate, interactions with a greater diversity of social partners, and who make few distinctions between in-group and out-groups; whereas collectivist societies are characterized by ethnocentric and xenophobic values, and therefore by individuals integrated into strong, cohesive in-groups, more extended family nepotism, fewer but more intimate social relations, stronger distinctions between in-group and out-groups (see review in Gelfand *et al.*, 2004), and decreased willingness to engage in social contact with out-group members (Sagiv & Schwartz, 1995).

Ember & Ember's (1992b) finding that socialization for mistrust independently predicts warfare suggests that the heightened ethnocentrism and xenophobia of collectivist societies may be salient; their source for this variable (Barry *et al.*, 1976) describes it as mistrust for members of the community who are outside the family. While collectivists, compared to individualists, socialize more intimately with members of their in-group, particularly members of extended families, they draw sharper distinctions between their in-group and out-groups (Gelfand *et al.*, 2004). Further, Barry *et al.* (1976) indicates that “sorcery and witchcraft generally indicate a low rating of trust.” (p. 95) The positive association between collectivism and in-group religious beliefs (Gelfand *et al.*, 2004; Hofstede, 2001) further suggests that the findings of Ember & Ember (1992b) may be explained by cross-cultural differences in collectivism-individualism.

In a cross-national analysis, Fincher *et al.* (2008) demonstrated that individualism correlates negatively (and collectivism correlates positively) with the intensity of human infectious disease in the environment. Previous studies have documented an association between ethnocentrism (Navarrete & Fessler, 2006) and xenophobia (Faulkner *et al.*, 2004) and perceived vulnerability to disease. This association suggests that the focus of social contact on in-group members, and avoidance of contact with out-group members, may be an important mechanism for management and avoidance of the risk of infection. Fincher *et al.* (2008) put forward and support the theory that collectivism is a cultural manifestation of this evolved anti-pathogen psychology.

There is significant geographic variation in human pathogens; they are especially diverse and severe in low latitudes (Guernier, Hochberg & Guegan, 2004; Low, 1990). Human populations become locally adapted to local pathogens through the process of parasite-host coevolution (Ewald, 1994; May & Anderson, 1983; see Fincher & Thornhill, 2008a, for evidence of localized immune defences among human groups). This local coadaptation between humans and their pathogens makes inter-group contact costly, by increasing the risk of contracting a pathogen to which an individual is not adapted. The intense in-group social interaction characteristic of ethnocentric societies also serves as an important buffer against the morbidity and mortality of infectious disease (Sugiyama, 2004); in-group social support may be critical in surviving periods of debility due to disease. As a result, in conditions of high infectious disease prevalence, selection favours people who adaptively express the evolved, conditional psychology that promotes avoidance of out-group members, and who concentrate their social interactions more intensely on the local in-group: those in the extended family and others with similar

immunological defences. Because the intensity of infectious disease is greatest at low latitudes, localization of immunological defence is greatest at low latitudes as well, as are the xenophobic and ethnocentric attitudes these evoke.

Fincher & Thornhill (2008a,b) further propose that the ethnocentric and xenophobic values of cultures in areas of high pathogen stress generate cultural diversity. As the process of localized parasite-host coevolution proceeds within a cultural group, spatial variation emerges both in the immune systems of the group members, and in the pathogens they carry. This spatial variation leads to mismatch between the immune systems of members of a local group and the pathogens carried by outsiders. This process leads to the emergence of new social boundaries based on language, norms, and values, as people assort themselves in order to avoid infection by novel pathogens to which their immune systems are not adapted. As a result, groups fission and diverge culturally, producing novel cultural diversity (Fincher & Thornhill, 2008a,b). It is likely that this fissioning process is not always a peaceful one, as the xenophobia that discourages out-group contact and novel contagion exposure makes it less likely that conflict between fissioning groups over previously shared territories and resources will be resolved in a cooperative manner.

We hypothesize that the process of group fissioning may often take the form of intrastate armed conflict or civil war. Similarly, the xenophobia of collectivist groups leads to hostility between neighbouring groups within a region; this contributes to the occurrence of intrastate conflict where political boundaries encompass groups that regard themselves as distinct from their neighbours (e.g. the Hutus and Tutsis of Rwanda). Schaller & Neuberg (2008) discussed the relationship of evolved intergroup prejudices to

intergroup conflict, and hypothesized that the xenophobic attitudes evoked by the risk of disease may contribute to intergroup aggression. This hypothesis is further suggested by Hofstede's (2001) characterization of “high risk of domestic intergroup conflict” in collectivist societies (in regions with high intensity of infectious disease; Fincher *et al.*, 2008) as a key difference from individualist societies (Hofstede, 2001, p. 251). This hypothesis predicts that more frequent intrastate armed conflict and civil war will be associated with increasing intensity of infectious disease.

In addition to the contribution to inter-group hostility by the ethnocentrism and xenophobia of cultures in regions of high intensity of infectious disease, disease has been shown to have a negative effect on national wealth. This can happen through two pathways. First, infectious disease can incapacitate workers on a large scale; this depresses economic growth and accounts for a significant amount of variation in wealth (Gallup & Sachs, 2001; Price-Smith, 2001). Second, infectious disease may have an indirect, negative effect on wealth through its evocation of ethnocentric and xenophobic cultures. The negative association between collectivism and wealth across countries is well recognized (e.g. Hofstede, 2001). There has been debate as to the direction of causality of this association (review in Gelfand *et al.*, 2004), but Thornhill, Fincher & Aran (2009) argue that it is individualism that leads to increased wealth (and therefore collectivism, and associated xenophobia and ethnocentrism, lead to relative poverty), as individualist cultures have higher democratization, and thus people are more willing to invest in public welfare, health, infrastructure, and other public goods; whereas in collectivist societies, people are unwilling to invest in public goods that will be shared beyond their extended family or ethnic group. This differential investment in public

welfare maintains widespread poverty in collectivist societies while individualist societies show relative prosperity.

Given the well-established negative correlation between national wealth, as measured by GDP *per capita*, and civil war (e.g. Ember & Ember, 1992b; Fearon & Laitin, 2003) we predict that infectious disease will make a further, indirect, positive contribution to the frequency of intrastate armed conflict and civil war, through its influence on decreasing national wealth. Price-Smith (2001) investigated the association between re-emerging infectious disease and various measures of state capacity, including GDP *per capita*, and argued for the important influence of infectious disease on intra- and inter-state warfare. However, he used infant mortality rates and life expectancy as proxies for pathogen severity, and did not investigate the association between pathogen severity and conflict *per se*. Here, we will examine this relationship specifically.

The causal pathway we suggest is one that begins with climate's influence on the intensity of infectious disease. Geographic variation in temperature and rainfall causes geographic variation in the intensity of human parasites (Guernier *et al.*, 2004). In this model, infectious disease causes the emergence of societies with ethnocentric and xenophobic cultural values in areas of high parasite stress, such as in low latitudes, and relatively xenophilic societies in areas of low intensity of infectious disease. This produces geographic and regional variation in the wealth of nations, and leads to differences in the frequencies of intrastate, inter-group conflict as groups within these nations compete for resources. Thus, this model unifies a number of the diverse variables known to correlate with the frequency of warfare, and completes the causal pathway from climate and infectious disease to the occurrence of intrastate conflict. In order to test this

model, we conducted a series of regression analyses of the cross-national incidence of intrastate armed conflict and civil war and its causes, which we present below.

METHODS

(1) Dependent variables

(a) Uppsala/PRIO intrastate armed conflict

Strand (2006) produced a dataset of onsets of intrastate armed conflict during the time period 1946 to 2004, based on the Uppsala/PRIO Armed Conflict Dataset (ACD). The ACD defines armed conflict as a contested political incompatibility that involves at least two organized parties, at least one of which is a state government, and which results in at least 25 battle deaths in one year (Gleditsch *et al.*, 2002). The ACD focuses on the contested political incompatibility in its definition in order to distinguish armed conflict from crime, one-sided violence, and communal violence. The definition also enables determination of the end of one conflict and the beginning of a new one, so that an ongoing conflict that is joined by a new organization is not considered a new conflict if it is over the same incompatibility (Strand, 2006).

Strand's (2006) intrastate conflict onset dataset builds on the ACD by expanding it into a list of years for each independent state in the interstate system (Gleditsch & Ward, 1999). There is one entry for each year during which each country was extant during the period 1945 to 2004. For example, the United States was extant during this entire period, and therefore has 60 entries in the dataset; whereas the Republic of Mali gained its independence in 1960, and therefore has 44 entries. Each country-year is coded as "0" (zero) if no armed conflict began that year in that country, or "1" (one) if an intrastate

armed conflict began. Date of onset is defined by the date on which a conflict surpassed the minimum 25 battle deaths required for inclusion in the ACD.

The onset dataset further builds on the ACD by coding reoccurring conflict with a threshold that varies from two to nine years of inactivity before a conflict is recorded as a new onset. It also distinguishes conflicts that meet only the minimum number of casualties for inclusion in the ACD from intermediate conflicts that resulted in a total of at least 1000 battle deaths, and from conflicts that resulted in at least 1000 battle deaths in one year (Strand, 2006). This latter category includes conflicts that meet the definition of a civil war (e.g. Singer & Small, 1994; and discussed below).

Strand's (2006) onset dataset includes 177 independent states, and 7889 country-years. We chose a two-year threshold for distinguishing reoccurring conflict from the onset of a new conflict. In our logistic regression analyses, we will independently consider conflicts that meet the minimum requirement of 25 battle deaths, and conflicts that meet the requirement of 1000 deaths in a year. There are 250 conflicts that meet the 25 battle death threshold; 148 conflicts in 57 independent states meet the higher threshold of 1000 deaths in one year, and we will refer to these as civil wars; 102 conflicts in 58 independent states never resulted in 1000 deaths in a year.

Using the onset data described above, our logistic regression analyses will treat country-year as the unit of analysis, as is customary in political science treatments of onset data (Hegre & Sambanis, 2006). In addition we wanted to analyze intrastate conflict using countries as the unit of analysis. Initially we thought to collapse these onset data into counts of conflicts experienced by each country for use in multiple regression analyses and path analysis. However, the resulting variable was non-normal

and could not be normalized by transformation. In particular, the count data were heavily zero-inflated. We therefore used the onset data to create a dummy variable coded “0” for countries in which there were no onsets of armed conflict during the period 1946 to 2004, or as a “1” for countries in which there were one or more onsets during this period. Our dummy variable for intrastate armed conflicts contains 58 countries that experienced one or more conflicts, and 119 that experienced no armed conflicts. Our dummy variable for civil wars contains 57 countries that saw one or more civil wars, and 120 that experienced none.

(b) Correlates of War intra-state wars

Fearon & Laitin (2003) created an onset dataset based on the Correlates of War (COW) intra-state war dataset (Singer & Small, 1994). Conflicts included in the COW dataset meet the following criteria: (1) fighting between agents of a state and organized, non-state groups who sought to take control of the government, a region, or to change government policies; (2) the conflict killed at least a total of 1,000, with a minimum yearly average of 100 casualties; and (3) at least 100 were killed on both sides.

Similar to Strand's (2006) armed conflict onset dataset discussed above, Fearon & Laitin (2003) coded a COW onset variable as “1” for country-years in which a civil war started and “0” for all others. This onset dataset spans the period 1945 to 1992. Data are available for 157 independent states, and a total of 6610 country-years. There are 92 civil wars involving a total of 51 states.

As described above for Uppsala armed conflicts and civil wars, we used these onset data to create a dummy variable for use in analyses that treat countries, rather than

country-year, as the unit of analysis. We coded countries “0” if they experienced no civil war, or “1” if they experienced one or more. We have 51 countries that experienced one or more civil wars, and 106 that experienced none.

(2) Independent variables

Hegre & Sambanis (2006) performed a sensitivity analysis to identify the most robust predictors of civil war and intrastate armed conflict. They found that GDP *per capita* and population size are robust predictors of both, and we therefore include these in our statistical models. However, although time at peace is generally included in statistical models of conflict, their analysis found that time at peace was a robust predictor of neither civil war nor armed conflict.

From Hegre & Sambanis' (2006) results, we identified three additional categories of predictors that were robust predictors of both intrastate armed conflict and civil war, significant on average with two-tailed probabilities: democracy, political instability, and economic growth. We therefore include these as control variables in our models.

In all cases we Z-score standardized our independent variables for ease of comparison.

(a) GDP per capita

Fearon & Laitin (2003) included in their models gross domestic product (GDP) *per capita* for each country in each year from 1945 to 1999. We use their GDP *per capita* data as a predictor for each country-year. GDP *per capita* is log-transformed to correct for skewness, with values lagged by one year in order to avoid endogeneity, as the

onset of conflict may be influenced by GDP *per capita*, but GDP *per capita* may also be influenced by the onset of conflict (Hegre & Sambanis, 2006).

In order to facilitate preliminary analysis of relationships among our independent variables, and to allow regression analysis with countries as the unit of analysis (discussed below), we also produced a mean GDP *per capita* by taking the average GDP *per capita* for each country over all available years in Fearon & Laitin's (2003) series.

(b) Population size

Models of intrastate armed conflict and civil war customarily control for a state's population size, since for a given potential for the onset of civil war, a country with a large population is more likely to pass a threshold for battle deaths for inclusion in a civil war data set (Hegre & Sambanis, 2006). Fearon & Laitin (2003) included a yearly population size for each country in their models. We use their population size series here. This variable is log-transformed to correct for skewness and lagged one year to avoid endogenous effects. As with GDP *per capita*, we produced a mean population size for each country over all available years.

(c) Democracy

Hegre & Sambanis (2006) found a robust effect of democracy on the onset of both armed conflict and civil war. We consider democratization a relevant control variable in our analysis from a theoretical standpoint, because of the negative association between infectious disease and democratization across nations (Thornhill *et al.*, 2009). In order to control for democratization, we include Polity IV's Polity2 index (Marshall & Jaggers,

2009). This is an index of countries' degree of democratization ranging from -10 to +10. The Polity IV project produces democracy scores for countries of the world based on three criteria: the existence of procedures by which citizens can express preferences about policies and leaders; institutionalized constraints on executive power; and guarantees for the civil liberties of citizens. It produces autocracy scores based on the following criteria: restriction of political participation; selection of chief executives from within the political elite; and lack of constraints on executive power. A country's Polity score is obtained by subtracting its autocracy score from its democracy score. Thus higher scores reflect greater democratization. The Polity2 index is a series based on countries' Polity scores with interpolated values to complete year-by-year data, and designed for use with time series analysis (Marshall & Jaggers, 2009). This variable is lagged by one year in order to avoid endogenous effects. We also produced a mean polity score for each country over all available years in the series.

(d) Political instability

Another variable found to be a robust predictor by Hegre & Sambanis (2006) is political instability. We use Fearon & Laitin's (2003) instability variable, which is a dummy variable coded as "1" if there was a change in a country's polity score in the previous three years, and "0" otherwise. We produced a mean political instability score for each country.

(e) GDP per capita growth

Finally, Hegre & Sambanis (2006) found that economic growth is a robust predictor of both intrastate armed conflict and civil war. This is theoretically an important control variable in our model as a measure of change in resource availability, or resource unpredictability, over time. As in Hegre & Sambanis' (2006) analysis, we calculated per cent growth in GDP *per capita* in each country from each year to the next from Fearon & Laitin's (2003) GDP *per capita* series. This variable is lagged by one year in order to avoid endogenous effects. We also produced a mean GDP *per capita* growth variable for each country over all available years in Fearon & Laitin's (2003) series.

(f) *Pathogen severity*

Our measure of the intensity of infectious disease is the Contemporary Pathogen Severity Index created by Fincher *et al.* (2008). This is a composite index of the prevalence of disease resulting from seven groups of human parasites in countries of the world, obtained from April to August, 2007, from the Global Infectious Diseases and Epidemiology Online Network (GIDEON; <http://www.gideononline.com/>). GIDEON is a regularly updated database of the distribution and severity of infectious disease. The pathogen severity index is a composite of severity scores from a total of 22 parasites from seven groups: leishmaniasis, trypanosomes, malaria, schistosomes, filariae, spirochetes and leprosy. These seven groups are those selected by Low (1989, 1990) in a series of studies that focused on the effects of pathogens on human behaviour and mate choice; she selected these groups based on satisfaction of Hamilton & Zuk's (1982) criteria for pathogens likely to have produced selection on these behaviours. These criteria are: an acute initial stage of infection; and a chronic stage with recurring acute infections, long-

term debility, or markers of past infection in those who recover from the acute stage of infection.

The severity of each pathogen in each country was recorded using GIDEON's three-point scale: (1) not endemic; (2) sporadic; (3) endemic. Because GIDEON does not map the severity of leprosy in terms of this three-point scale, leprosy was coded as such: (1) infection rate of 0 to 0.01/100,000; (2) 0.01 to 1/100,000; and (3) >1/100,000. Scores for all 22 parasites are summed for each country to produce the index (mean \pm SD=31.50 \pm 6.83, $N=225$ countries); see Fincher & Thornhill, 2008b, for data.

The validity of this index is supported by correlation with other cross-national indices of contemporary and historical pathogen severity. Low (1989, 1990) assembled an index of cross-cultural pathogen severity, based on records in disease atlases extending back to the early 1900s, for the study of the Standard Cross-Cultural Sample. Gangestad & Buss (1993) used the index assembled originally by Low (1989, 1990), to create an index of nation-level pathogen severity. Our index correlates highly with this index for the countries included in Gangestad & Buss' (1993) study ($r=0.819$, $N=27$, $P<0.001$). Our index also correlates highly with the World Health Organization's Disability Adjusted Life Years (DALY) index, a measure of mortality and morbidity across the globe. DALY scores lost years of healthy life and premature mortality as a result of various causes (World Health Organization, 2004; <http://www.who.int>). We used the DALY scores for infectious disease tracked by the WHO for the year 2002. After log-transformation of DALY for infectious disease to correct for skewness, its correlation to our index of pathogen severity is $r=.74$ ($N=192$, $P<.001$).

Because of the high degree of correlation of our measure of contemporary pathogen severity with Gangestad & Buss's (1993) results on historical cross-national pathogen severity, we include pathogen severity in our models as a time-invariant, country-specific effect (Hegre & Sambanis, 2006). The severity of infectious disease in a nation changes less dramatically over time than other variables such as *GDP per capita* and population size. Each nation's pathogen severity score is therefore entered in every country-year for that country in our models.

(3) Zero-order correlations

In order to investigate the relationships among our independent variables, we generated Pearson correlations and *P*-values for each pair of variables.

(4) Logistic regression analyses

We performed logistic regression analysis for each of the dependent variables described above: Uppsala's armed conflicts, Uppsala's civil wars, and COW civil wars. Logistic regressions were carried out using the Logit regression facility in Systat 11. We include in each model the five control variables described above, plus contemporary pathogen severity.

We first performed logistic regression analyses using country-year as our unit of analysis. This approach is standard in investigations of conflict onsets in the political science literature (e.g. Collier & Hoeffler, 2004; Fearon & Laitin, 2003; and see Hegre & Sambanis, 2006).

We performed additional logistic regression analyses using countries as our unit of analysis. Although we feel that practice in the political science literature on the onset of conflict justifies our inclusion of pathogen severity as a time-invariant, country-specific variable (Hegre & Sambanis, 2006), we were concerned that analysis at the level of country-year artificially inflates our sample size, given that we have only one measure of pathogen severity for each country in our analysis. Therefore as a check on the robustness of our results, we also report the results of logistic regression analyses in which dependent and independent variables have been collapsed to country-specific variables, and in which each N is a country, rather than a country-year.

Finally, in order to investigate the indirect effect of pathogen severity on intrastate armed conflict and civil war through its negative effect on *GDP per capita*, we follow the strategy for investigation of mediator variables described by Baron & Kenny (1986). We repeated the above analyses with *GDP per capita* removed from the model. If our hypothesis that *GDP per capita* is a mediator of the causal influence of infectious disease on conflict is correct, we will find that in models that include *GDP per capita*, it will be the dominant predictor; but that in models that exclude *GDP per capita*, the significance and magnitude of pathogen severity's predictive power will increase, indicating that pathogen severity accounts for a significant amount of the variation previously explained by *GDP per capita*.

RESULTS

(1) Zero-order correlations

The zero-order correlations among our independent variables are summarized in Table 1. We found that pathogen severity has significant, negative correlations with mean GDP *per capita* ($r=-0.68$; $P<0.001$; $N=148$) and mean polity score ($r=-0.45$; $P<0.001$; $N=153$); and a significant, positive correlation with mean political instability ($r=0.29$; $P<0.001$; $N=153$).

We also found that mean GDP *per capita* has a significant, positive correlation with mean polity score ($r=0.49$; $P<0.001$; $N=152$) and a significant, negative correlation with mean political instability ($r=-0.42$; $P<0.001$; $N=152$). In addition, mean polity and mean instability correlated negatively ($r=-0.28$; $P<0.001$; $N=157$).

We found that mean GDP growth and mean population size correlate positively ($r=0.17$; $P=0.034$; $N=152$). We found no other significant correlations for either of these variables.

(2) Logistic regression analyses

(a) Uppsala/PRIO intrastate armed conflict

Our logistic regression analysis of armed conflict onset (conflicts with more than 25 battle deaths, but which never resulted in 1000 battle deaths in one year) by country-year, including GDP *per capita* as an independent variable, found three significant predictors: population size ($b=0.47$, $P<0.001$); democracy ($b=0.39$, $P=0.008$); and pathogen severity ($b=0.50$, $p=0.002$). GDP *per capita* was not a significant predictor. After excluding GDP *per capita* from the model, the results are much the same: pathogen

severity was the strongest predictor ($b=0.59$, $P<0.001$), followed by population size ($b=0.47$, $P<0.001$) and democracy ($b=0.33$, $P=0.015$). See Table 2 for a summary of these results.

Our regression analysis by country, including GDP per capita, found that pathogen severity ($b = 0.76$; $p = 0.006$) was the only significant predictor of armed conflict. Population size ($b = 0.35$; $p = 0.068$) was a marginally significant positive predictor, and GDP per capita growth ($b = -0.34$; $p = 0.088$) was a marginally significant negative predictor. Excluding GDP per capita produced very similar results: pathogen severity was the strongest predictor of armed conflict ($b = 0.74$; $p < 0.001$). Population size ($b = 0.35$; $p = 0.068$) and GDP per capita growth ($b = -0.34$; $p = 0.086$) remained marginally significant predictors. See Table 3.

(b) Uppsala/PRIO civil wars

Our analysis of Uppsala civil wars by country-year, including GDP *per capita*, found three significant predictors of civil wars taken from the Uppsala dataset. These were GDP *per capita* ($b=-0.40$, $P = 0.002$); population size ($b=0.47$, $P<0.001$); and political instability ($b=0.16$, $P=0.036$). Democracy was a marginally significant predictor ($b=-0.20$, $P=0.088$). When we excluded GDP *per capita* from the model, population size remained a significant predictor ($b=0.47$, $P<0.001$). In addition, pathogen severity ($b=0.35$, $P<0.001$) became a significant, positive predictor, and democracy ($b=-0.32$, $P=0.004$) became a significant, negative predictor. See Table 2.

Our regression analysis by country, including GDP *per capita*, found two significant predictors: political instability ($b=0.79$; $P<0.001$) and population size ($b=0.60$;

$P=0.007$). GDP *per capita* ($b = -0.65$; $P = 0.057$) was a marginally significant predictor. After excluding GDP *per capita* from the analysis, political instability ($b=0.90$; $P<0.001$) and population size ($b=0.55$; $P=0.009$) were still the strongest predictors; in addition, pathogen severity ($b=0.51$; $P=0.026$) became a significant, positive predictor. See Table 3.

(c) Correlates of War civil wars

In our analysis of civil war onsets by country-year, including GDP *per capita*, there were three significant predictors of civil wars taken from the Correlates of War (COW) dataset. These were GDP *per capita* ($b=-0.49$, $P=0.002$); population size ($b=0.35$, $P=0.001$); and political instability ($b=0.28$, $P=0.003$). Pathogen severity was a marginally significant predictor ($b=0.26$, $P=0.086$). After excluding GDP *per capita* from the model, population size ($b=0.36$, $P=0.002$) and political instability ($b=0.31$, $P=0.001$) remained significant predictors; and pathogen severity ($b=0.46$, $P=0.001$) became the strongest, positive, significant predictor of civil war. See Table 2.

In our analysis by country, including GDP *per capita*, we found three significant predictors: GDP *per capita* ($b=-0.89$; $P=0.014$), political instability ($b=0.67$; $P=0.004$), and population size ($b=0.61$; $P=0.008$). After excluding GDP *per capita* from the model, we found that political instability ($b=0.82$; $P<0.001$) and population size ($b=0.53$; $P<0.001$) remained significant predictors; pathogen severity ($b=0.47$; $P=0.044$) was also a significant, positive predictor. See Table 3.

DISCUSSION

(1) Infectious disease predicts intrastate armed conflict

Even while controlling the effects of economic factors, population size, democracy and political instability, we found a statistically significant, positive influence of pathogen severity on the onset of small-scale intrastate armed conflicts taken from the Uppsala/PRIO dataset. Our logistic regression analysis produced a parameter estimate for pathogen severity that is larger in magnitude than that of any other predictor. Further, although GDP *per capita* is a standard control in models of armed conflict (Hegre & Sambanis, 2006), we found that GDP *per capita* was no longer a significant predictor of armed conflict in a statistical model that includes pathogen severity. This result was robust across both of our analyses, using both country and country-year as the unit of analysis.

We found a marginally significant, positive effect of pathogen severity on the onset of large-scale civil wars in the Correlates of War (COW) dataset using country-year as the unit of analysis. Although this marginally significant result in the direction we predicted might have been considered significant using one-tailed *P*-values, this result was not robust across both of our analyses of COW civil wars. However, we did find that after removing GDP *per capita* from our models of civil war, in each case pathogen severity became a significant predictor of both Uppsala/PRIO and COW civil wars. Except in the case of our model of COW civil wars by country, we found that the coefficient of pathogen severity increased to a magnitude comparable to that of GDP *per capita* in the corresponding model with GDP *per capita* included (See Tables 2 & 3). This result indicates a causal influence of pathogen severity on the occurrence of civil

war, *via* its indirect effect through the mediator GDP *per capita* (Baron & Kenny, 1986). Countries' GDP *per capita* averaged over the years 1945 to 1999 correlates with our measure of pathogen severity with $r=-0.68$; thus, the intensity of infectious disease accounts for 46% of the variation in wealth among nations. Given that GDP *per capita* is a robust, negative predictor of the onset of civil war, pathogens have an important influence on the occurrence of civil war, although this influence may not be apparent in models that include both pathogens and GDP *per capita* as independent variables.

(2) The causal role of infectious disease

These results support the direction of causality we propose here – that it is variation in intensity of infectious disease that causes variation in the frequency of intrastate armed conflict and civil war, rather than the converse. In our analysis of these conflicts by country-year, control variables such as GDP *per capita*, population size, *etc.*, were entered as yearly series and lagged by one year, while pathogen severity was entered as a time-invariant, country-specific effect. This method is standard in analysis of conflict onsets (Hegre & Sambanis, 2006) and ensures that, for example, a correlation between GDP *per capita* and onset of civil war does not reflect the endogenous effect of a civil war causing a reduction in GDP *per capita*.

Of course civil war can cause outbreaks of disease, for example among refugee populations. This results from the displacement of people by warfare, political instability, and accompanying reduction in economic growth, leading to the collapse of public health infrastructure (Ghobarah *et al.*, 2003). Thus our statistical models control for factors that mediate the causal influence of civil war on subsequent disease outbreaks; the remaining significant effects of infectious disease on the occurrence of civil war

therefore represent the causal pathway in which infectious disease stress causes conflict and civil war *via* the evocation of xenophobic and ethnocentric values.

So is it intensity of infectious disease that drives human behaviour, or *vice versa*? Certainly human behaviour with respect to social organization and mating can influence the prevalence and evolved virulence of infectious diseases, and particularly sexually transmitted diseases (Altizer *et al.*, 2003; Ewald, 1994). Nevertheless it is variation in climate, independent of factors such as human demography and wealth, that drives persistent, large-scale, geographic and cross-national variation in the distribution of human infectious diseases (Guernier *et al.*, 2004). It is this variation that is reflected in the pathogen prevalence variable used in the analysis presented here. Although the Contemporary Pathogen Severity Index used here is one composed of contemporary measures of the severity of infectious disease (Fincher *et al.*, 2008), its high correlation with Low's (1989, 1990) historical pathogen severity index, as applied to nations by Gangestad & Buss (1993), confirms that cross-national variation in these measures is relatively consistent over time. This supports our causal model, and also validates the use of contemporary measures of infectious disease in examining the historical causation of patterns in human societies and behaviours.

Letendre, Fincher, and Thornhill (in press) completed a path analysis of the Economist Intelligence Unit's Global Peace Index for 2008 (<http://www.visionofhumanity.org/gpi/results/rankings.php>) which supports the causal model proposed here. Intrastate armed conflict and civil wars produce a zero-inflated variable when collapsed into counts for each country, and are therefore problematic for purposes of path analysis. The Global Peace Index is an index of overall conflict

composed of 24 indicators including level of distrust in other citizens, political instability, numbers of external and internal conflicts, homicide rate, *etc.* The analysis by Letendre, Fincher, and Thornhill (in press) supported a causal model in which infectious disease causes the emergence of collectivist culture; infectious disease and collectivist culture together cause low GDP *per capita*; and degree of peace is caused negatively by collectivism, positively by GDP *per capita*, with an additional direct, positive causal path directly from infectious disease. Thus the causal pathway we argue for here – with infectious disease causing the emergence of collectivist cultures, and thereby leading to increased frequency of civil war – is supported theoretically and empirically.

(3) Infectious disease and conflict

We find that infectious disease has a significant, direct influence on the onset of conflicts with at least 25 battle deaths, but which never result in more than 1000 deaths in a year. Conflicts such as these bear more resemblance to the kinds of conflicts humans have engaged in for millions of years, than do large-scale conflicts that claim thousands of lives. It may be that the ethnocentrism and xenophobia that infectious disease engender are more likely to result in small-scale conflict among families and clans independent of wealth; whereas large-scale conflict involving many sub-groups more often results from broader economic grievance, opportunity for rebellion (Collier & Hoeffler, 2004), or a state's inability to suppress violent conflict (Fearon & Laitin, 2003). Despite this, we argue that intrastate armed conflict and civil war are not strictly modern phenomena. The species-typical psychology and behaviour that forms the link between parasites and violent intergroup conflict is implicated in patterns of warfare through history and in societies of all scales.

Violent conflict involves a degree of contact that increases the risk of disease transmission above that of complete isolation, particularly in conflicts in primitive societies where warfare often takes the form of hand-to-hand combat (but see Okada & Bingham, 2008, for a discussion of the importance of the advent of thrown weapons in human conflict and cooperation). If the theoretical link between infectious disease and violent intergroup conflict is the need for groups to isolate themselves from other groups that may be carrying novel pathogens, why does this result in violent conflict? Because we require the same resources to survive and reproduce, in humans, as in any other species, our most intense competitors are other conspecifics. Alexander (1979) argued that humans, especially, had become their own greatest source of mortality, and hence their own greatest source of selection, because of their elimination of the threat of predators and competitors for food sources. Unlike asocial species whose interactions are purely competitive except for mating purposes, humans also have the potential for cooperative social interaction with members of our own species. Often the benefits of cooperative social interaction dominate over the costs of our competitive interactions, and our relations are amicable and cooperative on the whole. The risk and cost of contracting novel pathogens from those outside our group increase in regions where infectious disease is intense, and this imposes a cost on intergroup cooperative interaction. As a result, people cease to regard out-group members as potential social partners, and the competitive aspects of our intraspecific interactions become dominant. Although there is a cost to engaging in violent intergroup conflict, in terms of risk of exposure to the very disease xenophobia is designed to avoid, the brief risk of exposure during this conflict is less than the protracted risk should two groups interact socially over an extended period

or share a resource such as a water source or living space. As a result, when two groups living in a region with a high intensity of infectious disease both need access to a common resource, the conflict is more likely to be solved by the use of intergroup violence than in regions where the cost of infectious disease is less. Alexander (1979, 1989) included escape from the selection pressure of disease in his description of humans' ecological dominance. However we argue that infectious disease has remained an important challenge that humans must address, even to the modern day. (Also see Thornhill *et al.*, 2009.)

(4) Disease, resources, and competition

The argument that infectious disease ultimately causes geographic and cross-national variation in the frequency of intrastate armed conflict does not diminish resource competition as a cause of this conflict. Ultimately, all conflict occurs as a result of competition for resources; thus in our model the robust negative relationship between resource availability and the frequency of intrastate armed conflict (Hegre & Sambanis, 2006) represents a real causal relationship. To this causal framework, our model adds variation in the intensity of infectious disease, and the value systems evoked under these conditions (Fincher *et al.*, 2008), which cause relative impoverishment (Price-Smith, 2001) and increasing likelihood that intergroup conflicts over resources will be resolved through warfare rather than through cooperative means.

Collier (2009) explores the characteristics of impoverished modern nations, and the “traps” that prevent the poorest nations from realizing the development and economic growth enjoyed by wealthy nations. These are: the conflict trap, in which conflict causes setbacks to economic progress, which in turn cause more conflict; the natural resource

trap, in which countries whose primary natural resources are distributed such that they can be controlled by a few, and the gains from which are therefore not invested in development and long-term growth; landlocked with bad neighbors, in which landlocked countries are impeded from trade, and particularly so if neighboring countries with access to the sea have not developed their infrastructure; bad governance in a small country, in which corrupt or abusive governments produce economic stagnation and collapse. No doubt these are important factors in understanding cross-national variation in wealth and economic development. However, focus on these factors alone neglects a more encompassing model that includes both ultimate and proximate causation, and does not explain broad, geographic patterns in the wealth of nations.

The conflict trap explains that conflict begets conflict, but the parasite-stress theory of intrastate conflict clarifies how countries at equatorial latitudes generally came to be those that fell into this trap, rather than countries at more temperate latitudes. Collier's (2009) latter three traps (the natural resources trap, landlocked with bad neighbors, bad governance in a small country) likely also play an intermediary role in the relationship between infectious disease, national wealth, and ultimately conflict. Collier (2009) addresses the squandering of natural resource wealth in patronage politics, but he argues that strong democratic institutions can prevent the typical boom and bust experienced by countries lacking these institutions, specifically contrasting the fate of Nigeria's oil wealth to that of Norway. Although whether a country is landlocked or not may come down to geographical luck of the draw, Collier (2009) argues that the obstacles to trade posed by lack of access to the sea are mitigated by having neighbors that have made investment in the infrastructure necessary to facilitate trade, specifically

contrasting Uganda and its neighbors to Switzerland and its neighbors. Degree of democratization and liberal values, including equality and willingness to invest in infrastructure and other public goods to be shared across groups, varies systematically with geography, and is accounted for in large part by variation in the intensity of infectious disease (Thornhill *et al.*, 2009). Thus, we propose a “disease trap” that feeds a diversity of factors, including those identified by Collier (2009) that lead to economic stagnation, poverty, and conflict. Similarly, such a disease trap likely contributes to the diversity of social ills, such as low life expectancy, poor health and education, and violence, attributed by Wilkinson & Pickett (2009) to inequality at the national, state, and local levels.

The influence of parasites on national wealth (Gallup & Sachs, 2001; Price-Smith, 2001) offers an explanation for the pattern of greater wealth in cooler nations, in addition to a cogent explanation that synthesizes a great deal of previous research on the differences in rates of intrastate armed conflict and civil war among nations. There is some debate over the direction of causality in the association between infectious disease and wealth (Price-Smith, 2001; Sachs & Malaney, 2002). We agree with Sachs & Malaney (2002) that causality likely runs in both directions. The morbidity caused by infectious disease incapacitates workers and depresses economic growth. In return, national wealth makes possible investment in public health infrastructure, including clean water sources and effective sewage systems, that can reduce the incidence of infectious disease. Similarly, there is debate about the direction of causality of the well-known association between individualism and national wealth (reviewed in Gelfand *et al.*, 2004). We argue that it is individualism that leads to greater wealth, through greater willingness

to invest in public welfare and infrastructure in individualist societies. We suspect there is a further indirect feedback of wealth into individualism, as investment in public health reduces the intensity of infectious disease. Although we argue that the direct causal link is individualism's effect on wealth, this indirect link may have played an important role in the liberalization and secularization of Western societies over the past century (see Norris & Inglehart, 2004, for patterns of secularization; see Thornhill *et al.*, 2009, for discussion of the role of public health on liberalization of values).

(5) Civil war in Africa

A number of scholars have specifically addressed the question of civil war in Africa. Collier & Hoeffler (2002) found that Africa fits into a global pattern of behaviour, with its poor economic performance leaving it prone to civil war. Likewise, Elbawadi & Sambanis (2000) found that Africa's high frequency of civil war results from its high levels of poverty. Our findings agree, with respect to civil war *per se*; we argue that Africa's low economic performance and high levels of poverty are the result of severity of infectious disease stress. Additionally, we find that intrastate armed conflict in Africa, and elsewhere, is a direct result of infectious disease stress, and its evocation of xenophobic and ethnocentric cultural values. The climate of much of Sub-Saharan Africa is particularly conducive to infectious diseases that lead to the development of collectivist values, and thus to widespread poverty. Additionally, as Africa is the place of origin of the human species (Templeton, 2002; Finlayson, 2005), the pathogens endemic to Africa have had a particularly long period of antagonistic coevolution with humans. As a result, the diversity of parasites, and associated intensity of human infectious disease, in Africa may be even greater than in other similarly tropical regions. The ancient ancestors of the

modern residents of those regions (e.g. southeast Asia, tropical South America) may have escaped some of their most virulent parasites when they migrated through more temperate regions that do not support high parasite richness (Guernier *et al.*, 2004).

(6) Global patterns of wealth and imperialism

Our findings raise some possibilities for the further study of international and imperialist wars. We found that relatively wealthy societies, relatively free of infectious disease, have lower frequencies of intrastate armed conflict and civil war. Although they did not examine intrastate conflict *per se*, Ember & Ember (1992a) note that democracies (which tend to be those societies with the least infectious disease; Thornhill *et al.*, 2009) do not experience less warfare overall than other countries, although they do experience less warfare in certain categories of conflict. If, as our results indicate, countries that are relatively free from infectious disease engage in less intrastate conflict, do these societies instead engage in more international wars, on their borders or overseas? In Diamond's (1997) examination of the global distribution of technology, wealth, and imperial power, he raises the possibility that societies characterized by conservatism – a value system closely related to collectivism (Oishi *et al.*, 1998; Thornhill *et al.*, 2009) – may experience slower rates of technological development. Similarly, Norris & Inglehart (2004) suggest that the technological development of Western societies may have benefited from the innovativeness of liberalism (and hence individualism). However, Diamond (1997) fails to pursue this line of reasoning, as he saw no reason that conservatism should vary systematically with geography. Diamond's (1997) thesis focuses instead on quirks of geography such as the east-west orientation of the Eurasian continent and the distribution of domesticable animals and crop plants.

The association of conservative and collectivist values with the intensity of infectious disease suggests that there is reason to believe that conservatism does vary systematically with latitude, with greatest conservatism at low latitudes where the intensity of infectious disease is greatest (Hofstede, 2001; Thornhill *et al.*, 2009). Our finding of the strong, negative correlation between pathogen severity and national wealth suggests that this relationship may have had an influence on the emergence of imperial cultures in Eurasia, and we argue that this has had a profound influence on the cultural development and economic and military histories of countries across the globe. As humans migrated from Africa into higher latitudes in Eurasia (Finlayson, 2005; Templeton, 2002), they moved into climates less hospitable to the parasites carried by humans at lower latitudes. This generated relatively individualist cultures which have increased openness to innovation and sharing of ideas and goods with neighboring groups (Thornhill *et al.*, 2009). Thus, the accumulation of wealth and technology that enabled the imperial domination of other more impoverished societies resulted not from quirks of Eurasian geography, but from escape from parasites, which allowed and promoted the development of relatively individualist cultures, and produced the large-scale geographic patterns observed today and through history, with wealthy countries concentrated in temperate latitudes, and impoverished countries wracked by internal conflict concentrated at tropical latitudes.

The model we propose offers an explanation for the global distribution of intrastate armed conflict and wealth, and may contribute to the understanding of the global history of imperialism and colonialism. Other researchers studying the frequency of civil wars have offered prescriptions for the reduction of the frequency of civil war,

including, often, economic development (e.g. Elbawadi & Sambanis, 2000). Our findings suggest that an effective approach to long-term economic assistance may be the prioritization of investment in sanitation, public health, and other means of suppressing infectious disease in impoverished nations at low latitudes. Relief from infectious disease has the potential not only to decrease the mortality and morbidity that constrains the productivity of nations with high parasite burdens, but may allow a cultural shift over time toward less xenophobic and ethnocentric cultural norms. This in turn will encourage continued domestic investment in public health and welfare, and these benefits will be perpetuated into future generations.

CONCLUSIONS

(1) Cross-national variation in the intensity of human infectious diseases produces variation in cultural norms and values. In countries with high intensity of infectious disease stress, cultures are characterized by ethnocentric and xenophobic values. This value system functions in the avoidance and management of exposure to novel pathogens which may be carried by out-group members. In addition to the relative poverty caused directly by infectious disease and the associated mortality and morbidity, xenophobic and ethnocentric cultures remain relatively impoverished because of unwillingness to make investments in public goods that will be shared across social groups within a nation. Resource deprivation, in combination with unwillingness to resolve competition and conflict between groups in a cooperative manner, leads to increased frequency of intrastate armed conflict and civil war in countries with high intensity of infectious disease.

(2) Warfare likely does cause subsequent outbreaks of infectious disease, decreases in GDP *per capita*, political instability, and other factors that are also implicated as causes of warfare themselves. Nevertheless statistical methods common to analysis of the onset of intrastate conflict allow examination of these factors prior to the outbreak of conflict, by eliminating endogenous effects from correlational analyses. Infectious disease stress makes a causal contribution to the occurrence of intrastate armed conflict.

(3) It is infectious disease stress that is the causal agent in this model. Climatic factors produce cross-national variation in the intensity of infectious disease. This variation produces cultures that are characterized by more or less ethnocentrism and xenophobia. Countries characterized by high ethnocentrism and xenophobia experience greater frequencies of intrastate armed conflict and civil war.

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REFERENCES

- Alexander, R. D. (1979). *Darwinism and Human Affairs*. University of Washington Press, Seattle.
- Alexander, R. D. (1989). Evolution of the human psyche. In *The Human Revolution: Behavioural and Biological Perspectives on the Origins of Modern Humans*, pp 455-513 (eds. P. Mellars & C. Stringer). Princeton University Press, Princeton.
- Altizer, S., Nunn, C., Thrall, P., Gittleman, J., Ezenwa, V., Pulliam, J., Pedersen, A., Dobson, A., Poss, M., Cunningham, A., Antonovics, J. & Jones, K. (2003). Social organization and disease risk in mammals: integrating theory and empirical studies. *Annual Review of Ecology, Evolution and Systematics* **34**,517-47.
- Anderson, C. A. (1989). Temperature and aggression: Ubiquitous effects of heat on occurrence of human violence. *Psychological Bulletin* **1**,74-96.
- André, C. & Platteau, J. (1998). Land relations under unbearable stress: Rwanda caught in the Malthusian trap. *Journal of Economic behaviour & Organization* **34**, 1-47.
- Baron, R. M. & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology* **51**, 1173-1182.
- Barry, H., III, Josephson, L., Lauer, E. & Marshall, C. (1976). Traits inculcated in childhood: Cross-cultural codes 5. *Ethnology* **15**, 83-114.
- Bowles, S. (2009). Did warfare among ancestral hunter-gatherers affect the evolution of human social behaviours? *Science* **324**, 1293-1298.
- Collier, P. (2009). *The Bottom Billion*. Oxford University Press, New York.

- Collier, P. & Hoeffler, A. (2002). On the incidence of civil war in Africa. *Journal of Conflict Resolution* **1**, 13-28.
- Collier, P. & Hoeffler, A. (2004). Greed and grievance in civil war. *Oxford Economic Papers* **56**, 563-595.
- Diamond, J. (1997). *Guns, Germs, and Steel*. W.W. Norton and Company, New York.
- Diamond, J. (2005). *Collapse: How Societies Choose to Fail or Succeed*. Viking, New York.
- Durham, W. H. (1976). Resource competition and human aggression, Part I: A review of primitive war. *The Quarterly Review of Biology* **51**, 385-415.
- Elbawadi, I. & Sambanis, N. (2000). Why are there so many civil wars in Africa? Understanding and preventing violent conflict. *Journal of African Economies* **9**, 244-269.
- Ember, C. R., & Ember, M. (1992a). Peace between participatory polities: A cross-cultural test of the “Democracies Rarely Fight Each Other” hypothesis. *World Politics* **44**, 573-599.
- Ember, C. R. & Ember, M. (1992b). Resource unpredictability, mistrust, and war. *Journal of Conflict Resolution* **36**, 242-262.
- Ewald, P. W. (1994). *Evolution of Infectious Disease*. Oxford University Press, New York.
- Faulkner, J., Schaller, M., Park, J. H. & Duncan, L. A. (2004). Evolved disease-avoidance mechanisms and contemporary xenophobic attitudes. *Group Processes & Intergroup Relations* **7**, 333-353.

- Fearon, J. D. & Laitin, D. D. (2003). Ethnicity, insurgency, and civil war. *American Political Science Review* **97**, 75-90.
- Field, C. B., Behrenfeld, M. J., Randerson, J. T. & Falkowski, P. (1998). Primary production of the biosphere: Integrating terrestrial and oceanic components. *Science* **281**, 237-240.
- 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 B: Biological Sciences* **275**, 2587-2594.
- Fincher, C. L., Thornhill, R., Murray, D. R. & Schaller, S. (2008). Pathogen prevalence predicts human cross-cultural variability in individualism/collectivism. *Proceedings of the Royal Society B: Biological Sciences* **275**, 1279-1285.
- Finlayson, C. (2005). Biogeography and evolution of the genus Homo. *Trends in Ecology & Evolution* **20**, 457-463.
- Gallup, J. & Sachs, J. (2001). The economic burden of malaria. *The American Journal of Tropical Medicine and Hygiene* **64**, 85-96.
- Gangestad, S. W. & Buss, D. M. (1993). Pathogen prevalence and human mate preferences. *Ethology and Sociobiology* **14**, 89-96
- Gelfand, M. J., Bhawuk, D. P. S., Nishii, L. H. & Bechtold, D. L. (2004). Individualism and collectivism. In *Culture, Leadership, and Organizations: The GLOBE Study of 62 Societies*, pp. 437-512. (eds. R. J. House, P. J. Hanges, M. Javidan, P. W. Dorfman & V. Gupta) Sage Publications, Thousand Oaks.

- Ghobarah, H. A., Huth, P. & Russett, B. (2003). Civil wars maim and kill people – long after the shooting stops. *American Political Science Review* **97**, 189-202.
- Gleditsch, N. P., Wallensteen, P., Eriksson, M., Sollenberg, M. & Strand, H. (2002). Armed conflict 1946–2001: A new dataset. *Journal of Peace Research* **39**, 615–637.
- Gleditsch, N. P., & Ward, M. D. (1999). A revised list of the independent states since the Congress of Vienna. *International Interactions* **25**, 393-413.
- Guernier, V., Hochberg, M. E., & Guegan, J. (2004). Ecology drives the worldwide distribution of human diseases. *PloS Biology* **2**, 740-746.
- Hamilton, W. D. & Zuk, M. (1982). Heritable true fitness and bright birds: a role for parasites? *Science* **218**, 384-387.
- Hegre, H. & Sambanis, N. (2006). Sensitivity analysis on empirical results on civil war onset. *Journal of Conflict Resolution* **50**, 508-535.
- Hofstede, G. (2001). *Culture's Consequences: Comparing Values, behaviours, Institutions, and Organizations Across Nations*, 2nd ed. Sage Publications, Thousand Oaks.
- Keeley, L. H. (1996). *War Before Civilization*. Oxford University Press, New York.
- Low, B.S. (1989). Pathogen stress and polygyny in humans. In *Human Reproductive Behaviour: A Darwinian Perspective*, (eds. L. Betzig, M.B. Mulder & P. Turke), pp. 115-127. Cambridge University Press, New York.
- Low, B.S. (1990). Marriage systems and pathogen stress in human societies. *American Zoologist* **30**, 325-339.

- Low, B.S. (2000). *Why Sex Matters: A Darwinian Look at Human behaviour*. Princeton University Press, Princeton.
- Manson, J. H. & Wrangham, R. W. (1991). Intergroup aggression in chimpanzees and humans. *Current Anthropology* **32**, 369-390.
- Marshall, M., & Jaggers, K. (2009). Polity IV Project: Political regime characteristics and transitions, 1800-2007. <http://www.systemicpeace.org/polity/polity4.htm>
- May, R. M., & Anderson, R. M. (1983). Parasite-host coevolution. In *Coevolution*, pp. 186-206 (eds. D.J. Futuyama & M. Slatkin). Sinauer Associates, Sunderland.
- Murdock, G. P. & White D. R. (1969). Standard cross-cultural sample. *Ethnology* **8**, 329-369.
- Navarrete, C. D. & 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.
- Norris, P. & Inglehart, R. (2004). *Sacred and Secular: Religion and Politics Worldwide*. Cambridge University Press, Cambridge.
- Oishi, S., Schimmack, U., Diener, E. & Suh, E. M. (1998). The measurement of values and individualism-collectivism. *Personality and Social Psychology Bulletin* **24**, 1177-1189.
- Okada, D. & Bingham, P. M. (2008). Human uniqueness – self-interest and social cooperation. *Journal of Theoretical Biology* **253**, 261-270.
- Price-Smith, A. T. (2001). *The Health of Nations: Infectious Disease, Environmental Change, and Their Effects on National Security and Development*. MIT Press, Cambridge.

- Sachs, J. & Malaney, P. (2002). The economic and social burden of malaria. *Nature* **415**, 680-685.
- Sagiv, L. & Schwartz, S.H. (1995). Value priorities and readiness for out-group contact. *Journal of Personality and Social Psychology* **69**, 437-448.
- Schaller, M. & Neuberg, S.L. (2008). Intergroup prejudices and intergroup conflicts. In *Foundations of Evolutionary Psychology*, pp. 399-412 (eds. C. Crawford & D. L. Krebs). Lawrence Erlbaum Associates, Mahwah.
- Schwartz, D. C. (1968). On the ecology of political violence: "The long hot summer" as a hypothesis. *American behavioural Scientist* **11**, 24-28.
- Singer, D.J. & Small, M. (1994). *Correlates of war project: international and civil war data, 1816-1992*. Data file, Inter-University Consortium for Political and Social Research, Ann Arbor, MI.
- Strand, H. (2006). Onset of Armed Conflict: A New List for the Period 1946–2004, with Applications. In Strand, H., *Reassessing the Civil Democratic Peace*, PhD thesis, Department of Political Science, University of Oslo & Centre for the Study of Civil War, PRIO.
- Sugiyama, L. S. (2004). Illness, injury, and disability among the Shiwiari forager-horticulturalists: Implications of health-risk buffering for the evolution of human life history. *American Journal of Physical Anthropology* **123**, 371-389.
- Templet, P. H. (1995). Economic scale, energy and sustainability: an international empirical analysis. *International Journal of Sustainable Development and World Ecology* **2**, 153-165.
- Templeton, A. R. (2002). Out of Africa again and again. *Nature* **416**, 45-51.

- Thornhill, R., Fincher, C. L., & Aran, D. (2009). Parasites, democratization, and the liberalization of values across contemporary countries. *Biological Reviews* **84**, 113-131.
- Van de Vliert, E., Schwartz, S. H., Huisman, S. E., Hofstede, G. & Daan, S. (1999). Temperature, cultural masculinity, and domestic political violence. *Journal of Cross-Cultural Psychology* **30**, 291-314.
- Vayda, A.P. (1967). Hypotheses about functions of war. In *The Anthropology of Armed Conflict and Aggression*, pp. 85-91 (eds. M. Fried, M. Harris & R. Murphy). Natural History Press, Garden City.
- Wilkinson, R. & Pickett, K. (2009). *The Spirit Level: Why more equal societies almost always do better*. Penguin, London.
- Williamson, P. & Moss, R. (1993). Degrees of national wealth. *Nature* **362**, 782.
- World Health Organization. (2004). Mortality and burden of disease estimates for WHO member states in 2002. Electronic file available at <http://www.who.int/healthinfo/bod/en/index.html>
- Wrangham, R. W. (1996). *Demonic Males*. Houghton Mifflin, Boston.
- Zhang, D. D., Brecke, P., Lee, H. F., He, Y. & Zhang, J. (2007). Global climate change, war, and population decline in recent human history. *Proceedings of the National Academy of Sciences* **104**, 19214-19219.

Table 1. Zero-order correlations among independent variables.

	1.	2.	3.	4.	5.	6.
1. Mean GDP <i>per capita</i>	--	152	152	152	152	148
2. Mean population	-0.02	--	157	157	152	153
3. Mean polity	0.49***	0.04	--	157	152	153
4. Mean instability	-0.42***	0.110	-0.28***	--	152	153
5. Mean GDP <i>per capita</i> growth	0.10	0.17*	0.02	-0.06	--	148
6. Pathogen severity	-0.68***	0.12	-0.45***	0.29***	0.01	--

Coefficients are below the diagonal; sample sizes are above.

† $P < .10$; * $P < .05$; ** $P < 0.01$; *** $P < 0.001$. Significance values are two-tailed probabilities.

Table 2. Logistic regression analyses of armed conflict and civil war by country-year

	Armed conflict	Armed conflict excluding GDP <i>per capita</i>	CW (Uppsala)	CW (Uppsala) excluding GDP <i>per capita</i>	CW (COW)	CW (COW) excluding GDP <i>per capita</i>
Log GDP <i>per capita</i>	-0.18 (0.17)		-0.40** (0.13)		-0.49** (0.16)	
Log population size	0.47*** (0.11)	0.47*** (0.11)	0.47*** (0.09)	0.47*** (0.09)	0.35** (0.11)	0.36** (0.11)
Democracy	0.39** (0.15)	0.33* (0.13)	-0.20† (0.12)	-0.32** (0.11)	0.01 (0.15)	-0.14 (0.14)
Political instability	0.11 (0.10)	0.13 (0.10)	0.16* (0.08)	0.20* (0.08)	0.28** (0.09)	0.31** (0.09)
GDP <i>per capita</i> growth	-0.14 (0.15)	-0.15 (0.15)	-0.11 (0.10)	-0.18 (0.11)	0.05 (0.09)	0.05 (0.09)
Pathogen severity	0.50** (0.16)	0.59*** (0.14)	0.16 (0.12)	0.35** (0.11)	0.26† (0.15)	0.46** (0.14)
Constant	-4.93*** (0.18)	-4.96*** (0.18)	-4.24*** (0.13)	-4.28*** (0.13)	-4.62*** (0.18)	-4.62*** (0.17)
<i>N</i>	5807	5807	5807	5807	4876	4876

Values are parameter estimates (standard errors) for predictors of intrastate armed conflict and civil war. † $P < .10$; * $P < .05$; ** $P < 0.01$; *** $P < 0.001$. Significance values are two-tailed probabilities. CW (Uppsala), civil war data set derived from the Uppsala/PRIO armed conflict data set; CW (COW), civil war onset data set derived from the Correlates of War (COW) intrastate conflict data set GDP, gross domestic product. See Section II for details. Armed conflict is defined as a conflict between a state government and another organized party that resulted in at least 25 battle deaths in one year. Civil wars are defined as conflicts between a state government and another organized party, and which resulted in at least 1000 battle deaths during a single year.

Table 3: Logistic regression analyses of armed conflict and civil war by country

	Armed conflict	Armed conflict excluding GDP <i>per capita</i>	CW (Uppsala)	CW (Uppsala) excluding GDP <i>per capita</i>	CW (COW)	CW (COW) excluding GDP <i>per capita</i>
Log GDP <i>per capita</i>	0.04 (0.28)		-0.65† (0.34)		-0.89* (0.36)	
Log population size	0.35† (0.19)	0.35† (0.19)	0.60** (0.22)	0.55** (0.21)	0.61** (0.23)	0.53*** (0.21)
Democracy	0.07 (0.22)	0.08 (0.21)	0.10 (0.28)	-0.07 (0.24)	0.11 (0.30)	-0.15 (0.25)
Political instability	0.01 (0.20)	-0.00 (0.19)	0.79** (0.23)	0.90*** (0.22)	0.67** (0.23)	0.82*** (0.22)
GDP <i>per capita</i> growth	-0.34† (0.20)	-0.34† (0.20)	-0.01 (0.23)	-0.07 (0.23)	0.31 (0.24)	0.25 (0.24)
Pathogen severity	0.76** (0.28)	0.74*** (0.22)	0.18 (0.29)	0.51* (0.23)	0.01 (0.96)	0.47* (0.23)
Constant	-1.12*** (0.23)	-1.11*** (0.22)	-0.98*** (0.25)	-1.03*** (0.24)	-1.14*** (0.26)	-1.20*** (0.25)
<i>N</i>	148	148	148	148	146	146

Values are parameter estimates (standard errors) for predictors of intrastate armed conflict and civil war. † $P < .10$; * $P < .05$; ** $P < 0.01$; *** $P < 0.001$. Significance values are two-tailed probabilities. CW (Uppsala), civil war data set derived from the Uppsala/PRIO armed conflict data set; CW (COW), civil war onset data set derived from the Correlates of War (COW) intrastate conflict data set. GDP, gross domestic product. See Section II for details. Armed conflict is defined as a conflict between a state government and another organized party that resulted in at least 25 battle deaths in one year. Civil wars are defined as conflicts between a state government and another organized party, and which resulted in at least 1000 battle deaths during a single year.

CHAPTER 3

PARASITE STRESS, COLLECTIVISM, AND HUMAN WARFARE

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SUMMARY

We recently proposed a new model to explain cross-national variation in the frequency of intrastate conflict based on the parasite-stress theory of sociality. In regions of high pathogen severity, cultures are characterized by xenophobia and ethnocentrism, which function in the avoidance and management of infectious disease. The xenophobia expressed in environments with high pathogen severity creates barriers to inter-group cooperation. These barriers cause greater poverty in environments with increased pathogen severity, in addition to the direct effects of disease on the human capital that is essential to economic growth. Xenophobic groups in competition for resources are unwilling to resolve this competition through cooperative means, and are more likely to resort to violent conflict. Here, we extend our findings to other categories of conflict.

We discuss the implications of our model to the understanding of human warfare in evolutionary context, and to foreign aid directed at reducing poverty and conflict.

INTRODUCTION

According to the parasite-stress model of sociality, temporal and spatial variation in parasite stresses generated past Darwinian selection that built species-typical, conditional psychological adaptations functionally designed for assessing local parasite stress. These adaptations guide the adoption and use of values (morals) pertaining to in-group and out-group behaviors that manage the risk and cost of exposure to infectious diseases. Hence, parasite stresses generated the selection that caused the evolution of this conditional psychology (ultimate causation); such stresses are ancestral cues that cause that psychology's behavioral manifestations within the lifetime of the individual (proximate causation). Host–parasite antagonistic coevolutionary races are variable and localized spatially across the range of a single human culture, yielding local co-adaptation between hosts and their local parasites (see Thompson, 2005; Fincher & Thornhill, 2008a). This creates a situation in which contact and interaction with non-group members (out-groups) is costly, because out-group members, relative to in-group members, carry parasites to which in-group members are not adapted immunologically. This can involve different parasite species or different variants of single parasite species. Xenophobia—the avoidance of and antagonism toward out-groups—appears to be an evolved solution to the problem of maladaptation to the infectious diseases parasitizing out-groups (Faulkner *et al.*, 2004). Ethnocentrism is a complementary, evolved solution to the fitness challenge imposed by parasite stress: loyalty toward, assistance of, and

interdependence with in-group members insures against the mortality and morbidity caused by local parasites (Navarrete & Fessler, 2006; Sugiyama, 2004; Sugiyama & Sugiyama, 2005). This ethnocentrism is comprised of two parts: (a) nuclear- and extended-family nepotism; and (b) cooperation with in-group, non-family members with shared values and immunology. The greater the parasite stress in a region, the greater the ethnocentrism and xenophobia; likewise, the lower the pathogen prevalence, the lower the ethnocentrism and xenophobia. Low ethnocentrism is the value of prioritizing nuclear-family-focused nepotism and with limited extended-family interactions and other in-group allegiance. Low xenophobia (= high xenophilia) is the value of attractiveness of out-group interactions and relations. Out-group interactions provide benefits to individuals of broader social networks and intergroup alliances, but such benefits will exceed costs only when parasite stresses are low. Consequently, the model proposes that parasites causally influence human values/morals, a major category of human preferences, pertaining to family life and to in-group and out-group feelings, motivations and behavior in general (Schaller & Duncan, 2007; Fincher *et al.*, 2008; Fincher & Thornhill, 2008a,b; Thornhill *et al.*, 2009).

Moreover, the model asserts that high parasite intensity leads to individuals with collectivist values/behaviors and, thus, emergent collectivist cultures, and that low levels of infectious diseases lead to individuals with individualistic values/behaviors and emergent individualistic cultures. The cross-national relationship between a country's location on the collectivism–individualism value unidimension and parasite prevalence in the country provides strong support for this aspect of the model: across many countries of the world, high parasite stress corresponds to high collectivism, whereas low parasite

stress corresponds to low collectivism, i.e., high individualism (Fincher *et al.*, 2008). Collectivism (as opposed to individualism) is a value system of out-group devaluation; in-group support; conformity to in-group norms; closed-ness to new ideas and ways; and allegiance to traditional values, hierarchy and authority. The collectivist understands self as immersed in and interdependent with the in-group, and places emphasis on distinguishing in-group from out-group members. In contrast, the ideology of individualism recognizes the validity and value of interactions with out-groups who have different norms and beliefs, and prioritizes openness to novelty, thus placing less importance on tradition and authority. The individualist understands self as relatively independent of the in-group, and in-group and out-group boundaries are dynamic and blurred (Gelfand *et al.*, 2004; Fincher *et al.*, 2008; Thornhill *et al.*, 2009).

Furthermore, parasite stress and collectivism negatively relate to democratization across the countries of the world: high parasite stress and associated high collectivism correspond to low levels of democratization, i.e., high autocracy (Thornhill *et al.*, 2009; Thornhill *et al.*, 2010). The interrelationship among parasites, collectivism, and democracy across countries is supportive of the parasite-stress model. Compared to individualistic countries, collectivist countries exhibit greater and more widespread poverty, inequality, morbidity, and mortality as a result of the reduced investment in public welfare, health, infrastructure, education and other public goods and services by the state. This reduced investment by elites stems from the collectivist ideology of devaluing out-group members, valuing in-group members and general endorsement of human inequality (Thornhill *et al.*, 2009; Thornhill *et al.*, 2010).

INTRASTATE ARMED CONFLICT

In a cross-national study of intrastate armed conflicts, a category of within-state conflict that includes civil wars (Letendre *et al.*, 2010), we proposed that in nations with relatively high intensity of infectious disease, the combination of increased resource competition (due to widespread economic dearth and inequality) and the ethnocentrism and xenophobia characteristic of collectivist societies cause increased frequency of intrastate armed conflict. In addition to the direct, negative effect of the mortality and morbidity of infectious disease on the human capital that is necessary for the generation of wealth (Bonds *et al.*, 2010; Price-Smith, 2001; Price-Smith, 2009), xenophobic groups are less willing to invest in public goods – such as infrastructure, education, and economic development – that will be shared across many groups within a nation (Thornhill *et al.*, 2009; Letendre *et al.*, 2010). In such countries, conflicts for resources are more likely to arise, and these conflicts are more likely to be resolved through armed conflict, or to escalate to civil war. In contrast, nations with relatively low intensity of infectious disease experience less severe resource competition (higher GDP *per capita* and more equitable resource distribution) and decreased ethnocentrism and xenophobia. Hence, conflicts for resources are less likely to arise in these nations; and, when they do arise, they are more likely to be reconciled through cooperative means instead of through war. This view is consistent with Hofstede’s characterization of a “high risk of domestic intergroup conflict” in collectivist societies as a key difference from individualist societies (Hofstede, 2001, p. 251; see also Price-Smith, 2009; Schaller & Neuberg, 2008).

The ideology of collectivism promotes within-state fractionation based on strong and localized preferences for in-group values and behaviors. As predicted, parasite stress

positively corresponds with both language and religion diversity across the countries of the world (Fincher & Thornhill, 2008a,b). High parasite stress leads to ideological and linguistic boundaries that cause cultural diversity within regions and within political boundaries. The within-state fractionation generated by collectivist values likely contributes to civil conflict.

Letendre *et al.* (2010) analyzed two data sets on outbreaks of intrastate armed conflicts across nations of the world: Fearon and Laitin's (2003) data on outbreaks in 157 states in the years 1945–1999, and Strand's (2006) data on outbreaks in 177 states in the years 1946–2004. Strand's (2006) data included small-scale intrastate armed conflict wars resulting in at least 25 battle deaths in one year, as well as large-scale civil wars. Fearon and Laitin's (2003) data, based on the Correlates of War Intrastate War data set (Singer & Small, 1994), tallied major civil wars—those killing at least 1,000, with a minimum yearly average of 100 dead, and at least 100 killed on both sides. Hence, data were analyzed for intrastate armed conflicts across a range of magnitude in terms of mortality.

The parasite-stress model of sociality applied to civil war was supported (Letendre *et al.*, 2010). The statistical analyses and their empirical results indicated that pathogen severity positively predicted the frequency of civil-war outbreaks across the globe, and this was found in separate analyses for small-scale conflicts with relatively low mortality, as well in large-scale civil wars with high mortality. Letendre *et al.* (2010) reviewed prior literature advocating theories of civil war based on environmental variables and the distribution and competition for resources. That review shows that the parasite-stress model of sociality integrates many diverse findings and hypotheses

reported in the political-science literature on the incidence of civil war (see also Discussion).

OTHER INTERGROUP CONFLICTS

Intrastate armed conflict is a specific category of domestic/intrastate conflict, according to standard definitions employed by political scientists (see Methods below). However, these are not the only type of such conflicts. There are additional types of these conflicts that are expected to be illuminated by the parasite-stress model. Herein, we explore extensions of the model to frequencies of (1) non-state-government wars, hereafter non-state wars, i.e., intergroup, within-state conflicts in which the state government is not a combatant; (2) political coups and revolutions; and (3) within-state terrorist events. As does civil war, all three of these additional intrastate conflicts derive from major differences in ideological preferences among groups within a nation. In non-state wars, organized groups war against one another, and the national government is not a combatant. A coup (also called a *coup d'état*) occurs when a national government is suddenly usurped and replaced by a faction (often the military) of the same government. Revolutions, like coups, involve regime changes, but over longer periods of time and involving social transformation of the old government by a considerable segment of the society. Within-state terrorism is the destruction of out-group's non-combatant civilians and/or their property because of conflicting political preferences. We hypothesize that all three types of intrastate conflicts arise, at least in part, from elevated out-group intolerance and devaluation, and in-group alliance and cooperation, and hence will be most frequent in nations with high pathogen severity and related high collectivism.

Specifically, for each of these three types of conflict, the parasite-stress model predicts that measures of parasite prevalence and of collectivism will each correlate positively with the incidence of these events across countries, and that individualism will correlate negatively with the incidence of each of the three categories of conflicts. We also reanalyze the small-scale intrastate armed conflicts we studied in Letendre *et al.* (2010), in order to test the robustness of our results when applying an additional control (see below).

Finally, we examine the application of the parasite-stress model to a measure of peace across countries. The measure combines information about the presence or absence of internal and external war across many nations. This analysis allows the extension of the parasite-stress model to international warfare, as well as to internal conflict. From the parasite-stress model, it is expected that, across nations, as parasite stress and collectivism decrease, peace will be more prevalent.

METHODS

In all cases, our analyses were of geopolitical regions (mostly countries, but some autonomous territories) that maintain a separate government – hereafter referred to as countries. Due to non-correspondence in the original data sources, sample sizes of countries vary across the analyses. We used Cohen’s (1988) guidelines that an effect size of 0.1 is a small effect, 0.3 is an intermediate/moderate effect, and 0.5 a large/strong effect.

Below, we introduce five independent variables we will analyze. The first four of these are variables indicating the incidence of four different categories of within-state

conflict. These four variables are zero-inflated: many countries experienced zero conflicts, while others experienced several. These zero-inflated variables cannot be normalized by transformation. We therefore collapsed each to a dummy variable coded “0” for countries that experienced no conflicts, or “1” for countries that experienced one or more. This dummy coding allows us to analyze these data using binary logistic regression, as in Letendre *et al.* (2010). The last independent variable we will introduce is a composite variable indicating the overall degree of peace vs. conflict within nations. As a composite of a variety of indicators of peace vs. conflict, this variable is continuous and can be normalized by log-transformation. Therefore this variable lends itself to analysis with least-squares regression and path analysis, which we describe below.

INTRASTATE ARMED CONFLICTS

We took data on the occurrence of intrastate armed conflicts from Strand's (2006) dataset of conflict onsets in 177 independent countries over the period 1946 to 2004, based on the Uppsala/PRIO Armed Conflict Dataset (ACD). The ACD defines intrastate armed conflict as: “...a contested incompatibility that concerns government or territory or both where the use of armed force between two parties results in at least 25 battle-related deaths. Of these two parties, at least one is the government of a state” (Gleditsch *et al.*, 2002, pp. 618-619).

Here, we focus on intrastate armed conflicts which occur between the state government and another internal opposition group(s), which do not reach the threshold of 1,000 battle deaths in one year to meet the definition of a civil war (Gleditsch *et al.*, 2002). These are the small-scale intrastate conflicts analyzed by Letendre *et al.* (2010),

and which we re-analyze here to test the robustness of our results while including a control for world culture region (see below).

These data contain 119 countries that experienced zero conflicts over the period 1964 to 2004, while 58 experienced one or more.

NON-STATE WARS

Data on non-state war occurrences were taken from the Uppsala Conflict Data Program (UCDP) WWW site. We used UCDP Non-State Conflict Dataset V.1.1, 2002–2005, accessible at <http://www.pcr.uu.se/research/UCDP/index.htm>. This is a cross-national (255 countries) dataset with information about armed conflict onset between two organized groups within a country, neither of which is the government of the country, resulting in at least 25 battle-related deaths in a calendar year; both military and civilian deaths are counted as battle-related deaths. Hence, these non-state wars are a different type of conflict than intrastate armed conflicts, as the latter always involve the government of a state vs. an organized warring group(s) within that state. These wars are escalated inter-ethnic or clan wars; examples are in Uganda, the Pokot clan vs. the Sabinu clan; Syria, Arabs vs. Kurds; Somalia, the Jareer subclan of the Hawiye clan vs. the Jiddo subclan of the Digil clan. This dataset lists 231 countries that experienced zero non-state war onsets, and 24 countries with one or more over the period of 2002–2005.

REVOLUTIONS AND COUPS

We used the Barro-Lee Dataset for a panel of 138 countries (Barro & Lee, 1994). The Barro-Lee variable used was REVCOU, which they define as “. . . [T]he number of

revolutions and coups per year, averaged over the period 1960–1984.” The source for these data is Banks (1979, updated). This variable was used in a recent cross-national analysis of political instability by Nettle *et al.* (2007). The events appear to reflect the standard definitions of revolutions and coups as used in political science (see above).

TERRORISM

We used all terrorist incidents listed in the Worldwide Incidents Tracking System (WITS), the National Counterterrorism Center’s (NCTC) database for the period 2005–2007 (<http://www.nctc.gov>). In all of the years in this period, the same definitions of terrorism and methodology were employed. Prior to 2005, a “limited methodology” was used that focused on international terrorism (NCTC, 2007 Report on Terrorism, April 30, 2008, p. 2; <http://wits.nctc.gov/Reports.do>). International terrorism involves citizens or property of more than one country. The dataset 2005–2007 contains both international and domestic incidents, primarily the latter. Only a very small fraction of total terrorism is international (see Abadie, 2006). The country attributed to the terrorism event is the one in which it occurred. The 2005–2007 data consistently used the following definition:

“[T]errorism occurs when groups or individuals acting on political motivation deliberately or recklessly attack civilians/non-combatants or their property and the attack does not fall into another special category of political violence, such as crime, rioting or . . . tribal violence.” (<http://wits.nctc.gov>)

Terms in this definition (e.g., political motivation, non-combatants, and so on) are defined in the NCTC 2007 Report.

PEACE

We used the Global Peace Index for 2008, collated and calculated by the Economist Intelligence Unit. The Index is available for 140 countries and is comprised of 24 qualitative and quantitative indicators, which combine factors pertaining to countries' relative peace status. Peace here prioritizes measures of an absence of violent conflicts with neighboring countries and of internal wars. The 24 indicators include: the level of distrust in other citizens; political instability; relations with neighboring countries; the number of external and internal conflicts fought between 2000–2005; the number of deaths from both external and internal conflict; military expenditures; potential for terrorist acts; homicide rate.

The Index ranges from 1 to 5, where 1 is the most peaceful and 5 the least peaceful. Iceland is the most peaceful, with a score of 1.176; Iraq is the least peaceful, with a score of 3.514. Data and descriptions of ranking methods used are at <http://www.visionofhumanity.org/gpi/results/rankings.php>. For clarity in interpreting the relationships between the Global Peace Index and other variables we examine, we reversed the coding of the Global Peace Index so that high values correspond to greatest peace, and low values correspond to least peace. Thus, for example, we predict that pathogen severity correlates negatively with the Global Peace Index after reverse coding. We log-transformed the Global Peace Index in order to obtain a normally distributed variable. We also Z-score standardized this variable for ease of comparison of results between our regression and path analyses. It is this reverse-coded, normalized, log-transformed index that we will use throughout.

PARASITE STRESS

As our measure of the variation in parasite stress across countries, we used the Contemporary Pathogen Severity Index assembled by Fincher and Thornhill (2008b). This is an index of a subset of infectious diseases for each country using data extracted from the Global Infectious Disease and Epidemiology Network (GIDEON; <http://www.gideononline.com>). We used a set of parasites similar to those used in prior research (e.g., Low, 1990; Gangestad *et al.*, 2006), using the seven classes of important human infectious disease identified by Low (1990), but expanded to include all entries in GIDEON for each class (a total of 22 human parasites). We classified the country-wide disease level of seven groups of parasites: leishmaniasis, trypanosomes, malaria, schistosomes, the filariae, spirochetes, and leprosy.

We used GIDEON's 3-point scale of parasite prevalence (3 = endemic, 2 = sporadic, 1 = not endemic) based on distribution maps and coding provided in GIDEON. Leprosy was handled differently, because GIDEON does not map the precise distribution; rather the infection rates are presented. Thus, we coded infection rates *per capita* of 0–0.01/100,000 as 1, 0.01–1/100,000 as 2, and > 1/100,000 as 3. Trypanosomiasis–African was given in GIDEON in a similar way. We coded infection rate “not endemic” as 1, > 0 to 0.25/100,000 as 2, and > 0.25/100,000 as 3. The values for the different parasites were summed and provide our index of contemporary pathogen severity (mean = 31.5 ± 6.8 , $n = 225$, range = 23–48). These data were collected from April–August, 2007 and are in Fincher and Thornhill (2008b).

The validity of this index is supported by its strong, positive correlation with other cross-national measures of the intensity of infectious disease (Fincher *et al.*, 2008;

Letendre *et al.*, 2010), including the World Health Organization's Disability Adjusted Life Years for infectious disease, which is an index of years of life lost due to the mortality and morbidity caused by infectious disease (World Health Organization, 2004; <http://www.who.int>). The Contemporary Pathogen Severity index also correlates strongly and positively with Gangestad & Buss' (1993) historical pathogen severity index ($r = 0.819, n = 27, p < 0.001$). This index is based on prevalence of pathogens and parasites in the same seven groups identified by Low (1990) as recorded in world distribution maps dating as far back as the early 1900's. This strong correlation indicates the stability of cross-national variation in infectious disease over time. The Contemporary Pathogen Severity Index also strongly correlates with the three major measures of collectivism–individualism across countries of the world (with collectivism, positively; with individualism, negatively) (see Fincher *et al.*, 2008).

COLLECTIVISM–INDIVIDUALISM

For the purposes of path analysis, we selected Family Ties as our measure of collectivism–individualism. Alesina and Giuliano (2007) reported that “family ties” – a composite variable of several values from the World Values Survey (<http://www.worldvaluessurvey.org/>) was correlated positively with collectivism (and negatively with individualism) across the many countries in the World Values Survey. Family ties is a measure of allegiance to and importance of the extended family – a major part of collectivist values (Gelfand *et al.*, 2004; Fincher *et al.*, 2008).

Family ties shows moderate to high correlations with other measures of collectivism–individualism: Hofstede individualism (Hofstede, 2001), $r = -0.481, n = 57$;

in-group collectivism practices (IGCP; Gelfand *et al.*, 2004), $r = 0.688$, $n = 44$; Suh individualism (Suh *et al.*, 1998), $r = -0.383$, $n = 52$. The highest scores on family ties correspond to high collectivism, and the lowest scores correspond to high individualism. We selected family ties as our measure of collectivism because of its relatively large sample size, $n = 81$ countries (*cf.* Hofstede: $n = 72$; IGCP: $n = 62$; Suh: $n = 61$). Because path analysis is particularly demanding of sample size (Streiner, 2005) and because the available measures of collectivism are limited relative to the other variables we will include in our analysis, we selected the measure with the largest available sample size among these.

CONTROL VARIABLES

As in Letendre *et al.* (2010) we include in our regression analyses an exhaustive battery of variables identified in a sensitivity analysis by Hegre and Sambanis (2006) as robust predictors of the onset of intrastate armed conflict. We include all categories of predictors that were found to be robust, significant (two-tailed $p < 0.05$) predictors of both small- and large-scale intrastate armed conflict: gross domestic product (GDP) *per capita*, population size, democratization, political instability, and economic growth.

GDP per capita

GDP *per capita* is relevant to the incidence of conflict, as a measure of the resources available to the people of a country. Ultimately all conflict is over reproductive resources (Manson & Wrangham, 1991; Low, 2000), and GDP *per capita* is an empirically relevant measure of the availability of such resources (Letendre *et al.*, 2010).

We predict that GDP *per capita* will correlate negatively with the frequency of the various categories of conflict, but positively with peace.

As in Letendre *et al.* (2010) we used GDP *per capita* values derived from a series covering the period 1945 to 1999 published by Fearon and Laitin (2003). We collapsed GDP *per capita* values from this series to a single value for each country for analysis at the level of the country by taking the mean over the time period.

Population Size

Population size is a standard control in the political science literature on the occurrence of intergroup conflict (Hegre & Sambanis, 2006). Many definitions for various categories of conflict include some minimum threshold for number of death (see above), and so it is to be expected that as population size increases, there is increasing potential for events that meet those definitions. We predict that population size will correlate positively with conflict; and negatively with peace, as among the indicators that comprise the composite Global Peace Index is the frequency of internal conflicts. We took population size from the series by Fearon and Laitin (2003) over the period 1945 to 1999, with values collapsed to a single value for each country, as above.

Democratization

Degree of democratization has been hypothesized to relate to the frequency of intrastate conflicts in two ways. First, democratic institutions may reduce the motivation to engage in violence against the government or other groups within the state, as democratic institutions allow greater possibility for citizens or groups to address

grievances without resorting to violence, than do autocratic institutions (Gleditsch *et al.*, 2009). Second, democratic institutions may increase the opportunity to engage in violence to address grievances relative to autocratic societies in which liberties are strictly curtailed (Gleditsch *et al.*, 2009).

These two factors have been hypothesized to produce a U-shaped relationship between intergroup conflict and democratization. However Hegre and Sambanis (2006) report only a robust effect for the linear component of democratization on the frequency of intrastate armed conflict and civil war, and therefore we include only this linear component as a control here. Degree of democratization relates to the parasite stress theory of sociality in that democratization and associated liberal values negatively vary cross-nationally with parasite stress (Thornhill *et al.*, 2009; Thornhill *et al.*, 2010).

We take our measure of democratization from the Polity IV Project's Polity 2 index (Marshall & Jaggers, 2009). Polity 2 is an index of countries democratic vs. autocratic institutions, including the existence of procedures by which citizens can express political preferences, constraints on executive power, and guarantees on the civil liberties of citizens. Higher scores indicate greater degree of democratization. Polity 2 is a yearly series, and thus as in Letendre *et al.* (2010) and as described above, we collapsed values to a single mean value for each country in our analysis.

Political Instability

Hegre and Sambanis (2006) found political instability to be a robust, positive predictor of small-scale intrastate armed conflict and civil war. As in Letendre *et al.* (2010) we take our indicator of political instability from the series covering the years

1945-1999 by Fearon and Laitin (2003), based on countries' change in score on the Polity 2 index over time. Fearon and Laitin (2003) coded their political instability series “1” for years in which countries experienced a change in Polity score, or “0” for years in which countries experienced no change. We calculated mean number of change per year from this series, in order to obtain a single value for each country.

GDP per capita growth

Finally we include *GDP per capita* growth, which is relevant to the analysis of intrastate conflict as a measure of resource unpredictability. Low *GDP per capita* growth may increase the frequency of violent conflict by producing times of scarcity (Ember & Ember, 1992; Zhang *et al.*, 2007) and anxiety about future shortages (Ember & Ember, 1992). Following Fearon and Laitin (2003) we calculated *GDP per capita* growth as percent change in countries' *GDP per capita*. We obtained a single value for each country based on Fearon and Laitin's (2003) yearly *GDP per capita* series.

World Region

Although Hegre and Sambanis (2006) did not find that variables encoding geographic regions *per se* to be robust predictors of intrastate armed conflict or civil war, critics of the research program supporting the parasite stress theory of sociality (e.g. Nettle, 2009) have questioned the validity of analyses that do not account for dependence among cultures within regions. In addition, there are other possible confounds that may vary with geographic region, such as Africa and South America's history of colonization by European nations (see discussion below). Therefore, to control for such possible

confounds, we include a categorical variable coding each countries' membership in one of Murdock's (1949) six world culture areas.

REGRESSION ANALYSES

We collapsed intrastate armed conflicts, revolutions and coups, non-state wars, and terrorism into binary dummy variables, coded “0” for countries that experienced no conflict, or “1” for countries that experienced one or more. We examined each of these using binary logistic regression analysis, with all predictors and control variables entered simultaneously.

The Global Peace Index is normally distributed when log-transformed to correct for skew. Therefore we used least squares regression to analyze this variable, with all predictors and control variables entered simultaneously.

We performed regression analyses using Systat version 11 (<http://www.systat.com>). We report one-tailed *p* values for all regression analyses, as we have unambiguous directional predictions based on the parasite-stress theory and the results in Letendre *et al.* (2010). We report standardized β s throughout.

PATH ANALYSIS

The model we propose has its causal basis in the influence of parasite stress on human behavior and cultural values, and links parasite stress to intergroup conflict via the ethnocentrism and xenophobia of collectivist cultures, and further via indirect effects such as national wealth and degree of democratization. Path analysis allows us to investigate the relative effects of all these variables in a comprehensive fashion.

For the purposes of path analysis, we chose the Global Peace Index as our measure of conflict, for two reasons. First, the Global Peace Index is a composite index which includes in its scores a diversity of factors that contribute to overall peace vs. conflict within a country. These measures include the number of internal conflicts and potential for terrorist acts; the peace index reflects a number of the other direct measures of conflict described above, and is therefore a useful summary variable for the general association between parasite stress and conflict that our model seeks to explain. Second, unlike the measures of civil war analyzed in Letendre *et al.* (2010) and the non-state wars, revolutions, coups, and terrorism described above – which, because they include many countries that experience none of these events and a few that experience many, are zero-inflated and not possible to normalize – the Global Peace Index has better fit to a normal distribution and therefore lends itself to path analysis.

Because the sample size of our total model was limited by the relatively small samples available for measures of collectivism (family ties: $n = 81$), we limited our path analysis to a relatively simple model including a small number of relevant variables. As mediators of the relationship between parasites and collectivism and their effect on peace, we included measures of GDP *per capita* and democratization. As mentioned above, these variables are theoretically relevant to the model presented here (see also Discussion). These variables' correlations with intrastate conflict are robust and widely recognized, and they are commonly regarded as important control variables in models of intrastate conflict (Hegre & Sambanis, 2006).

Finally, we included population size as a control variable in our model. Population size is a standard control variable in models of the incidence of intrastate conflict (Hegre

& Sambanis, 2006) because of the reasoning that with increasing population size there is more opportunity for the occurrence of conflict. Population size is a relevant control variable here because in our model, conflict results upon the emergence of group boundaries and inter-group hostilities; and with increasing population size there are likely to be more of these conflicting sub-groups within the political boundaries of a country. We performed path analyses with PROC CALIS, using SAS version 9.1

(<http://www.sas.com/>).

RESULTS

INTRASTATE ARMED CONFLICTS

As predicted by the parasite-stress model, pathogen severity was the strongest, positive predictor of the incidence of intrastate armed conflict ($\beta = 0.71, p = 0.032, n = 148$ countries). Intrastate armed conflict was also positively predicted by population size ($\beta = 0.38, p = 0.044$). There was a marginally significant, negative effect of GDP *per capita* growth ($\beta = -0.33, p = 0.056$). World Area did not significantly predict intrastate armed conflict, nor did GDP *per capita*, democracy, nor political instability. See Table 1 for a summary of these results.

NON-STATE WARS

As predicted by the parasite-stress model, the incidence of non-state wars was positively predicted by pathogen severity ($\beta = 1.16, p = 0.021, n = 148$ countries). Population size was also a positive predictor ($\beta = 1.17, p = 0.001$), while GDP *per capita* was a negative predictor ($\beta = -0.92, p = 0.042$). World Area did not significantly predict

non-state wars, nor did democracy, political instability, nor GDP *per capita* growth. See Table 1.

REVOLUTIONS AND COUPS

As predicted, revolutions and coups were positively predicted by pathogen severity ($\beta = 1.09, p = 0.044, n = 117$ countries). The incidence of revolutions and coups was also positively predicted by political instability ($\beta = 1.85, p = 0.001$). (The large magnitude of the effect of political instability on revolutions and coups may in part be due to endogeneity. The definition of both of these variables includes some change in the makeup or behavior of the government; thus while political instability may play a causal role in the incidence of revolutions and coups, the converse may simultaneously be true. While this may exaggerate the strength of this relationship in these results, nevertheless we found similar results with respect to the other variables included here whether political instability was included or excluded as an independent variable.) There were also marginally significant, negative effects of democratization ($\beta = -0.56, p = 0.071$) and GDP *per capita* growth ($\beta = -0.92, p = 0.082$). World Area was not a significant predictor of revolutions and coups, nor was GDP *per capita*, nor population size. See Table 1.

TERRORISM

When controlling for the other independent variables included here, terrorism is not significantly predicted by pathogen severity ($p > 0.10$). The incidence of terrorism was positively predicted by population size ($\beta = 0.95, p < 0.001, n = 148$ countries), and

negatively by GDP *per capita* ($\beta = -0.59, p = 0.047$). World Area was not a significant predictor, nor was democracy, political instability, nor GDP *per capita* growth. See Table 1.

As in our analysis of civil wars in Letendre *et al.* (2010), we investigated a possible indirect role of pathogen severity on the incidence of terrorism via pathogen severity's negative effect on GDP *per capita* (e.g., Price-Smith, 2001). In order to test GDP *per capita* as a mediator of a relationship between pathogen severity and terrorism (Baron & Kenny, 1986), we repeated the above regression analysis with GDP *per capita* excluded from the model. Still, we found that pathogen severity is not a significant predictor ($p > 0.10$). We will discuss this contrary finding further below.

PEACE

Our least squares fit of the Global Peace Index revealed that, as predicted by the parasite-stress model, pathogen severity is the strongest, negative predictor of peace across countries of the world ($\beta = -0.48, p < 0.001, n = 129$ countries). GDP *per capita* ($\beta = 0.19, p = 0.043$) and GDP *per capita* growth ($\beta = 0.19, p = 0.007$) were significant, positive predictors of peace. World area did not significantly predict peace, nor did population size, democracy, nor political instability.

PATH ANALYSIS

The hypothesized model is illustrated in Figure 1. Rectangles represent measured variables. Straight lines represent hypothesized causal pathways, with plus (+) and minus (-) signs indicating the hypothesized positive or negative direction of the relationship; the

curved line indicates a covariance relationship between the two exogenous variables, pathogen severity and population size.

We hypothesized that peace is predicted positively by GDP *per capita*, negatively by family ties, positively by polity (democratization), and negatively by population size. We hypothesized that GDP *per capita* is negatively predicted by pathogen severity and family ties. We hypothesized that democratization (Polity) is negatively predicted by family ties. Finally, we hypothesized that family ties is positively predicted by pathogen severity. This analysis thus includes 10 variables: six observed and four unobserved variables. (The four unobserved variables are error terms associated with each of the four endogenous variables. These unobserved variables must be estimated by the model, and therefore must be taken into consideration when evaluating sample size: Streiner, 2005.)

We included in our analysis only cases for which we had complete data for all six observed variables included in our model ($n = 66$ countries). Per the method described by Tabachnick and Fidell (2007), we evaluated the multivariate normality of our data by examining pairwise correlations of each pair of variables. We found that relationships among our variables are linear where relationships exist. However, the residuals of some of these relationships showed evidence of non-normality; therefore, we used the maximum likelihood method of estimation in our analyses. Among the $n = 66$ countries, we found two outliers with respect to population size (China and India); and one outlier with respect to the relationship between GDP *per capita* and democratization (Saudi Arabia). We therefore excluded these from our analysis (see Tabachnick & Fidell, 2007), leaving a final sample size of $n = 63$ countries.

We found poor support for the hypothesized model as originally specified: $\chi^2 p = 0.0078$; RMSEA = 0.1725; NFI = 0.8910 ($\chi^2 p > 0.10$, RMSEA < 0.06 and NFI > 0.95 indicate a good fitting model; Tabachnick & Fidell, 2007). We therefore performed *post hoc* model modifications to identify a better fitting model. The largest Lagrange multiplier suggested the fit of the model improves with the addition of a direct relation between pathogen severity and peace ($p = 0.0068$); this addition makes theoretical sense in that the model we proposed seeks to account for cross-national variation in conflict as the result of variation in the intensity of infectious disease. The Wald test suggested that the removal of the relation between democratization and peace would not decrease the fit of the model ($p = 0.6743$), and we therefore removed this relation.

We subsequently found that once the model takes into account a direct relationship between pathogen severity and peace, the relationships of population size and family ties with peace are no longer significant, and unnecessary for the fit of the model (Wald tests $p = 0.2459$ and $p = 0.2424$, respectively). Finally, because democratization and population size no longer play a causal role in our model of peace, and in order to produce a simpler model better served by our limited sample size, we eliminated these two variables from the final model.

Our final model fits the data well: $\chi^2 p = 0.3794$; RMSEA < 0.0001; NFI = 0.9969. It contains seven variables: four observed, and three unobserved error terms. The ratio of sample size to variables is thus 9:1. The final model is illustrated in Figure 2. Peace was predicted negatively by pathogen severity (standardized coefficient $\beta = -0.5394$, $p < 0.0001$) and positively by GDP *per capita* ($\beta = 0.2321$, $p = 0.0404$). GDP *per capita* was predicted negatively by pathogen severity ($\beta = -0.3290$, $p = 0.0174$) and family ties ($\beta = -$

0.3707, $p = 0.0074$). Family ties was predicted positively by pathogen severity ($\beta = 0.7151$, $p < 0.0001$). This model accounts for 49.66% of the variation in peace across our sample.

DISCUSSION AND CONCLUSIONS

The major empirical findings are that the predictions of the parasite-stress model, when applied to political conflicts across the countries of the world, were largely satisfied. As predicted, the incidence of intrastate armed conflict, non-state wars, and revolutions and coups was predicted by parasite stress; the predicted relationships also were seen with a measure of peacefulness, the absence of internal and external conflict. As parasite stress and its associated collectivism increase, and GDP *per capita* decreases across countries, so does the incidence of intrastate armed conflict, non-state war, and revolutions and coups. Peacefulness increases as parasite stress and collectivism decline across countries and GDP *per capita* increases. These results are robust across a variety of categories of conflict, even when controlling for a battery of demographic, economic, and political indicators.

The one category of conflict which did not satisfy the prediction of the parasite-stress theory was terrorist incidents. We failed to find support for either a direct or indirect effect of pathogen severity on the incidence of terrorism. This analysis should be viewed as preliminary, due to limitations of the available data. We could not discern confidently international from domestic terrorism events, although we are confident that the vast majority of events in the dataset we used (WITS) are domestic. We could not find the data set used by Abadie (2006), the World Market Research Center's Global Terrorism

Index. We did not use the Global Terrorism Database of the U.S. Department of Homeland Security, because it is stated on the Web site that its methods involve “no set definition . . . covering several definitions of terrorism” (<http://www.start.umd.edu/data/gtd/gtd1-and-gtd2.asp>). Given that, in some regions of the world, propaganda about terrorism and its threat are used in political competitions, it may be difficult to obtain data free from political manipulation of terrorist reports.

Another consideration is the fact that terrorism, and especially modern suicide terrorism, is extremely asymmetric: attackers can inflict damage on a multitude of undefended, civilian targets, using cheap and readily available materials that are difficult to detect (Atran, 2003). Therefore, it may be that it is generally collectivist elements within a country that are responsible for terrorist events, as we hypothesize; however, if only a few collectivist individuals can carry out terrorist attacks in countries otherwise characterized by relatively individualist cultures (e.g., the Oklahoma City bombing, carried out by right-wing extremist Timothy McVeigh in the United States in 1995; Blee & Creasap, 2010), then the regression analyses performed here at the cross-national level will not reveal this relationship. Our hypothesis with respect to terrorism is suggested by Sosis and Alcorta’s (2008) finding that terrorism is tied strongly to ethnocentric convictions about the truth of in-group religious beliefs and the falsity of out-group religious beliefs. Even secular terrorism, they suggest, is the result of strong ideological preference differences between groups. MacNeill (2004) posited that religion is an evolutionary adaptation for warfare in general – an adaptation likely to be implicated even in a relatively novel expression of inter-group violence such as terrorism. The positive association of parasite stress and religiosity has been described, tested, and

supported by Fincher and Thornhill (in press); thus, our approach places these diverse findings in the context of a general model based fundamentally on parasite stress. We hope that given relevant data, this theory may yet shed light on the causes of terrorism.

Throughout our regression analyses, we included a control for world cultural region, in order to address the issue of cultural dependence among cultures within a region that share characteristics as a result of recent common cultural descent (e.g., Nettle, 2009), and also possible confounds such as European nations' history of colonialism of African, South American, and Asian nations. In each of our analyses, we found no significant effect of world region on the incidence of conflict, or on peace. Other researchers have concluded that the high frequency of civil wars in Africa, for example, is explicable as the result of common predictors of civil war across the world, such as Africa's high levels of poverty and the characteristics of its political institutions (Collier & Hoeffler, 2002; Elbadawi & Sambanis, 2000). Likewise, we find that variation in the frequency of conflict and degree of peace within cultural regions is explained by the economic and political variables included in our analyses; and we add variation in the intensity of infectious disease as an important causal factor in the economic performance (Letendre, 2010) and political make-up (Thornhill *et al.*, 2009) of nations. Rather than the history of cultures in different regions of the world driving the association between infectious disease and conflict, we argue that ecologically determined variation in the intensity of infectious disease has caused different histories of poverty and wealth (Price-Smith, 2001), democracy and autocracy (Thornhill *et al.*, 2009), conflict and peace, imperialism and colonialism, and technological development in different regions and nations across the world (Letendre *et al.*, 2010).

These findings, combined with the detailed, longitudinal and cross-sectional analyses of civil wars and small-scale intrastate conflict by Letendre *et al.* (2010), support the application of the parasite-stress model of sociality to political conflict. Our analyses of the Global Peace Index across countries allow an initial examination of the parasite-stress model in relation to international political conflicts. Although path analysis of our model of the Global Peace Index was not supported as originally specified, we found support for a revised model that agrees with the overall argument that infectious disease plays an important causal role in variation in peace across countries. These results indicate that the parasite-stress model may be an important way to understand many major types of political conflicts.

Fundamentally, the parasite-stress model of political conflicts rests on earlier scientific findings that human behavior and its associated psychological foundations have been crafted ultimately by past biological evolutionary processes. This conclusion is based on a huge empirical base in biology and related areas of scholarship generated over the last 150 years. The parasite-stress model proceeds from this scientific conclusion—that every aspect of the human phenotype was caused ultimately by evolutionary historical processes—to a specific model of the human behavioral, emotional, motivational and deductive phenotypes that are manifested in the context of political conflicts.

In our theoretical approach to the functional organization of human political conflicts, we have focused on evolved adaptations of individuals rather than upon group-related adaptation that may hypothetically arise from group selection, because it is at the level of genes and individuals that Darwinian selection causes the evolutionary changes that

create psychological adaptations (e.g., West *et al.*, 2007; Williams, 1966). This selection favors traits of individuals that provide the bearers with the highest relative inclusive fitness—that is, traits that allow their bearer to produce the greatest number of reproductively successful genetic relatives (descendant and/or non-descendant). The behavior of groups emerges from the behavior of individuals, who are designed by evolution to maximize inclusive fitness in ancestral environments.

The parasite-stress model proposes that political conflicts can be understood as follows. They are caused by behavioral and psychological features functionally designed for (i.e., directly selected in the context of) the xenophobia that functions in the avoidance of novel pathogens that may be carried by out-group members. This out-group avoidance brings competitive interactions to the forefront of humans' relations with out-group members. In relatively xenophilic cultures, cooperative and positive social interactions may dominate; but when the increasing cost of social contact and risk of disease exposure, and the xenophobia these costs evoke, make these positive social interactions impossible, then, in humans as much as in any species, competitive intraspecific interactions dominate (Letendre *et al.*, 2010). Given the demonstrated, robust association between pathogen severity and inter-group conflict, it is not surprising that xenophobia manifests not only as fear of contagion, but also as general out-group mistrust (Ember & Ember, 1992). For any human group expressing the degree of xenophobia that is optimal given the local disease ecology, neighboring groups are experiencing similar disease stresses, and members of those groups likely express similar degrees of xenophobia and out-group hostility. Thus one's own degree of out-group

hostility is predictive of the degree of out-group hostility and proclivity to violent intergroup conflict expressed by local out-group members.

The ethnocentrism associated with collectivist cultures functions in part in the management of the morbidity and mortality of infectious disease (Navarrete & Fessler, 2006; Sugiyama, 2004; Sugiyama & Sugiyama, 2005). Additionally, given the association between pathogen severity and inter-group conflict, the ethnocentrism associated with collectivism may serve an adaptive function in within-group cohesion in defense against coercion and aggression by out-groups, and in offensive coercion and aggression against out-groups (Navarrete *et al.*, 2004). Thus, the robust association between xenophobia and conflict may account, in part, for the reliable association between xenophobia and ethnocentrism that makes up the cultural dimension of collectivism-individualism.

In this regard, the psychology of collectivism is causal, because to an important extent it is designed for (a) distinguishing group boundaries through adherence to shared in-group values and behaviors and, hence, for identifying out-groups, and (b) producing negative feelings (dislike, disgust) toward them. The other relevant part of collectivist emotions and behavior is designed for investment in, support of, and loyalty toward in-group members comprised of extended family and other group members with the same values/morals. Collectivism is interdependency on and high valuation of in-group members with simultaneous devaluation of out-group members. Collectivism is appropriately cast as in-group cooperation. Therefore, collectivism is the basis of success in both defensive and offensive out-group hostility. The degree of cooperation achieved among members of a warring group—whether a raiding party of egalitarian hunter—

gatherers or a highly hierarchical, modern army—is recognized widely as critical for effectiveness in warring (e.g., see Buss' [2004] discussion of warfare). Apparently, coalitional aggression against out-groups is pursued exclusively by men (Alexander, 1979; Wrangham & Peterson, 1996). However, the people (including women) not participating directly in warring participate by providing moral support and associated assistance, which, like the amity among members of the warring coalition, is promoted by collectivist ideology.

There is increasing evidence of psychological adaptation of men that is functionally designed for war. Its information-processing capacities include an assessment of benefits from war in the form of access to women and other resources, as well as an assessment of coalitional support and strength of own vs. enemy group (Duntley & Buss, 2009). This adaptation may have been directly sexually selected in the context of men's competition for status, women, and related resources (Buss, 2004; Low, 1993; Manson & Wrangham, 1991; Wrangham & Peterson, 1996). In the parasite-stress model, the war adaptation interacts with the psychological adaptations that function in adopting and using human values, such that xenophobia and in-group allegiance (collectivism) affect the decision that war is the appropriate means for dealing with intergroup conflict. Hence, warfare is caused partly by war adaptation in men and partly by collectivist values.

At first glance, it may seem contradictory that warfare is partially caused by the need to avoid the risks and costs of exposure to novel pathogens, given men's motivation to participate in warfare because of the opportunity to gain sexual access to out-group women through wife-capture (Chagnon, 1997; Low, 2000; Manson & Wrangham, 1991), and the ubiquity of wartime rape (e.g. Gotschall, 2004; Thornhill & Palmer, 2000).

Schaller and Murray (2008) and Thornhill *et al.* (2009) found a strong, negative correlation between infectious disease prevalence and desire for sexual variety and number of sex partners (using the Sociosexual Orientation Inventory; Simpson & Gangestad, 1991) among women cross-culturally, but a weak to nonexistent relationship among men. Thus for men, it may be that the fitness benefits of gaining sexual access to women outweigh the risk of exposure to novel diseases even in environments in which high intensity of infectious disease favors out-group avoidance. Furthermore, in environments with high pathogen prevalence, people prioritize physical attractiveness as a marker of health in their mate choices (Gangestad & Buss, 1993; Gangestad *et al.*, 2006). Thus, the women targeted for wife-capture or rape during wartime in environments with high intensity of infectious disease are predicted to be younger, more physically attractive, and healthier; and that this is a strategy on the part of men to take advantage of the fitness benefits of acquiring new mates while minimizing the cost of exposure to novel infectious diseases. In environments with reduced intensity of infectious disease, women may be targeted for wife-capture and wartime rape with less discrimination.

The parasite-stress model proposes that the psychology of collectivism and of war gives rise to political conflict when perceived benefits of intergroup conflict exceed its high costs. One benefit of pursuit of the conflict is decreased competition and greater access to resources in the event of victory over the out-group. Another benefit is the exclusion of the out-group from the region, and in some cases, out-group extermination. Although warfare may expose warriors to the risk of contracting new diseases from the enemy during combat, warfare may importantly reduce future intergroup contact and

interaction relative to the degree of intergroup contact and contagion risk that would result from sharing territory or a resource in the long-term (Letendre *et al.*, 2010).

According to the parasite-stress model, during human evolutionary history, this future reduction of intergroup contact and interaction provides inclusive fitness benefits greater than the risk and cost of contracting infectious diseases during combat. In this view, the xenophobia that motivated intergroup aggression had as its net effect avoidance of problems resulting from infectious diseases.

Thus, fundamentally, the model rests on the ultimate cause of direct selection resulting from mortality and morbidity from infectious diseases. This parasite-based selection built collectivist psychology and its capacity for evaluating, adopting, and using collectivist values and behavior, which function in avoidance of novel infectious disease (xenophobia), and management of the mortality and morbidity of disease, and resulting inter-group conflict (ethnocentrism).

The parasite-stress model also rests on the existence of moral psychological adaptation with *condition-dependent* expression ranging from high collectivism to high individualism (Thornhill *et al.*, 2009). Condition-dependence is the design of moral psychology, because in human evolutionary history, there was spatial and temporal variation in the risks and costs of infectious diseases. Accordingly, moral psychology's function is to adopt the set of values appropriate for the local ecology of human parasite prevalence. Individualism emerges in ecological settings with low infectious disease stress, and has the important benefits of intergroup trade, barter, exchange of ideas and technology, and expansion of one's pool of participants for reciprocity and mating. Its benefit-to-cost ratio increases as infectious disease problems decrease. Empirically, as

predicted, there is an increase in the degree of widespread individualism as parasite stress declines across the countries of the world. Similarly, as predicted, the degree of widespread collectivism increases as parasite stress increases across countries (Fincher *et al.*, 2008).

In the context of infectious disease, past selection created a condition-dependent moral psychology, the psychological adaptations that manifest in human cognitions and behavior as collectivism and associated xenophobia and ethnocentrism, or as individualism. The moral psychology is designed to incorporate values during development (ontogeny) by learning socially those values well suited to local parasite stress. Separately, some possible ontogenetic ancestral cues that may guide historically adaptive construction of individuals' moral repertoires have been proposed (Fincher *et al.*, 2008; Thornhill *et al.*, 2009). High parasite stress proximately causes a willingness to accept the costs of political conflict, whereas low parasite stress proximately builds pacifism and other positivism toward out-groups. The model, then, may explain much of the variation in the values affecting domestic and interstate conflicts across the globe.

In the model, the following are proximate causes of political conflict, as well as its antipole, pacifism/absence of such conflict: the war psychological adaptation, the moral psychological adaptation, the psychology of collectivism–individualism, the psychology that assesses local parasite stress, the ontogenetic events involved in the production of all this phenotypic machinery, and local parasite stress. Of course, the ontogenetic events mentioned include the important role of the evocation of values as well as the social learning of values within and across generations, which give rise to what many researchers call “cultural evolution,” referring to changes in the frequencies of ideas,

values and related behavior (Richerson & Boyd, 2006). The ontogeny of the social-learning machinery, like the ontogeny of all phenotypic features, is causally dependent on genes as a partial proximate cause.

We use “cause” here in its typical, scientific interpretation: that, without which, an effect will not occur. Each proximate cause listed above is necessary, but insufficient alone, to generate political conflict, the effect of focal interest here. Each is a partial cause; again, using the standard conception of cause in science.

By definition, proximate biological causes are those that act to generate an effect within the lifetime of the organism. Each piece of machinery in the above list of proximate causes is the product of evolutionary historical causation, i.e., ultimate causation. We have treated only the selection history of this machinery and ignored phylogenetic ultimate causation, a distinct and complementary causal framework that addresses the location on the Tree of Life where traits first appeared in the history of life. According to the parasite-stress model, the selection that built all the proximate machinery (listed above) was direct selection in the context of parasite stress, or in the case of the war adaptation, direct sexual selection for condition-dependent warring behavior.

The parasite-stress model of political conflicts predicts (i.e., requires for its support) that the frequency of political conflicts across countries will show a positive correlation with parasite stress and collectivism (and a negative correlation with individualism). If these patterns are not seen, the model is false; the findings to date reported herein and in Letendre *et al.* (2010) support the model.

The parasite-stress model is about the causes of values and their effects. A given cause-effect model specifies the predicted effects, and those effects are sought empirically to determine if the model is supported or not. The civil-conflict literature is voluminous, especially with regard to civil war (partial reviews in Hegre & Sambanis, 2006 and Nettle *et al.*, 2007 [civil war and interethnic conflict]; Alesina *et al.*, 1996 [coups]; Abadie & Gardeazabal, 2008; Sosis & Alcorta, 2008 [terrorism]). This literature proposes various causal models for these conflicts, as well as numerous tests of the models. Often, the factors of population size, GDP *per capita*, Gini (a measure of wealth inequality), time since last conflict (in the case of civil war), inconsistent democratic institutions, political instability, war-prone and undemocratic neighboring countries, ethnic diversity, and a low rate of economic growth are considered to be basic causes of such conflicts. Often in research looking at one or a few of these variables that predict conflict, other variables are considered confounds and, hence, are statistically controlled.

In the parasite-stress model, however, these variables are effects of the same underlying proximate and ultimate causation—parasite stress and its effects on values. Even increased population size, which is positively correlated with the frequency of within-country conflicts (e.g., Hegre & Sambanis, 2006), may be an effect of parasite stress in many parts of the globe, because infectious disease is correlated positively with birth rate cross-nationally, as expected from life-history theory (Guégan *et al.*, 2001). Separately, treated in detail, is how GDP/capita, economic growth, and democratization are predicted consequences of the parasite-stress model. In short, upon relative emancipation from infectious diseases, peoples' values become more individualistic or liberalized, which results in greater investment in public goods and services and the

welfare of out-groups in general (Fincher *et al.*, 2008; Thornhill *et al.*, 2009). The relationships between ethnic or cultural diversity and parasite stress are treated by Fincher and Thornhill (2008a,b). Furthermore, it has been proposed that the spatial autocorrelation in domestic conflict events among countries within a geographical region results from regional differences in the ecological conditions (e.g., rainfall and temperature) affecting parasite stress (see Fincher & Thornhill 2008a,b). We propose, too, that the variable “time since last civil conflict” is an effect of the parasite-stress model. Hence, the parasite-stress model cannot be tested appropriately by controlling statistically these variables. For example, to control for GDP *per capita* and/or democratization in an analysis of, say, parasite stress and non-state wars would reduce the ability to detect the predicted relationships, because GDP *per capita* and democratization are consequences of parasite stress: low GDP *per capita* and low democracy derives from high parasite stress (and associated collectivist values), and high GDP *per capita* and high democracy from low parasite stress (and associated individualistic values). Thus, it is noteworthy that we found a significant effect of pathogen severity on all indicators of conflict, except terrorism, even when controlling other economic and political indicators that are themselves causally dependent on the intensity of infectious disease.

Because of the above considerations, aspects of the parasite model of civil conflict do not lend themselves to analysis by multiple regression analysis, controlling for the variety of known correlates that are standard controls (which we propose are mediating variables in the causal relationship between infectious disease and conflict) in such analyses. Path analysis therefore seems to be a more appropriate method. Path analysis did not support

our hypothesized model as originally specified. However, with minor *post hoc* modification of the model, adding and removing paths according to the Lagrange multiplier and Wald test, we arrived at a model that fit our data well, and supports the theory we propose here.

Our hypothesized model did not include a direct relation between pathogen severity and the Global Peace Index, but instead linked these two indirectly via some of the intermediary effects we have discussed: family ties (collectivism), GDP *per capita*, and democratization. Analysis of our hypothesized model suggested that the model would be strongly improved by the inclusion of a direct relation of pathogen severity to peace (Lagrange multiplier: $p = 0.0068$). This addition agrees with our theory because, in order to restrict our analysis to relatively simple models, we did not include a number of other known correlates of conflict that we argue are intermediary to the relationship of infectious disease to peace (e.g., wealth disparity, political instability, etc.).

We found that the Wald test suggested that removal of the relation of democratization to peace would not decrease the fit of the model. This suggests that, while democratization is a known correlate of conflict, its relationship with conflict is spurious, and is accounted for by both variables' association with infectious disease and collectivism. We also found that removal of the direct relation of family ties to peace did not decrease the fit of the model. This may be because the family ties variable, although it correlates highly with other measures of collectivism, measures allegiance to the extended family, and may therefore be primarily a measure of ethnocentrism. While ethnocentrism is an important component of the cultural unidimension of collectivism-individualism, it does not correlate perfectly with the other component of collectivism,

xenophobia (e.g., Cashdan, 2001). Thus there is the possibility that xenophobia and ethnocentrism play distinct roles in our path model. There may be a mediating role of xenophobia between pathogen severity and peace, which is not explicit in our model because we do not have a corresponding measure of xenophobia in the analysis.

Alternatively, it may be, as our final model suggests, that collectivism leads to increased conflict (decreased peace) entirely through other intermediary effects, such as collectivism's negative impact on GDP *per capita*.

The results of our path analysis support the model we propose. Our final model fits very well to our data (e.g., RMSEA < 0.0001; models with RMSEA < 0.06 are regarded as having good fit, while RMSEA = 0.0 indicates perfect fit; Tabachnick & Fidell, 2007). Nevertheless, the results of this analysis should be regarded as preliminary. The sample size of our dataset was limited by the sample sizes for the available measures of collectivism. We selected family ties for its large sample size relative to other measures and restricted our analysis to as simple models as possible; still, its sample size is fairly small for the purposes of path analysis, and the samples with complete overlap with our other variables were even smaller. Our final 9:1 ratio of $n = 63$ sample size to seven variables (four observed and three unobserved) is somewhat less than the desired ratio in path analysis (Klein, 1998, recommends 10 samples per variable, whereas five is too few). Furthermore, although we removed univariate and multivariate outliers from our dataset, we did see evidence of multivariate non-normality in the pairwise correlations of our variables. This suggests further caution in the interpretation of these results. We hope that in the future measures of collectivism may become available for more countries of the world, perhaps in future waves of the World Values Survey, which will enable a

more conclusive analysis. Nevertheless, we report that path analysis of the data available provides preliminary support for the infectious disease model of civil conflict.

Hegre and Sambanis (2006) point out that published analyses of civil conflicts across countries are highly variable in model components and specifications of relevant statistical-control variables, and propose that this is because, “[W]e do not know the true model” (p. 513). The models of human activity that are most general and useful for scientific discovery are those based in the evolutionary science of human functional design (examples are the empirical cornucopia from Hamilton’s [1964a,b] model of nepotism; Trivers’ [1971] model of direct reciprocal altruism; Alexander’s [1987] model of indirect reciprocity and reputation). Hence, the most general and useful models of human political conflict will be those based in human psychological functional design, and models ignoring evolved psychological adaptations are of relatively limited scientific value. In the long tradition of political conflict research, there is no generally accepted model, because the research has not been inspired by evolutionary theory. This chapter is an attempt to identify a general model of political conflict that is inclusive of all the relevant values and their effects—e.g., the value of out-group dislike brings about the effect of disfranchisement of much of the citizenry of a country—that arise under high parasite stress as well as under emancipation from parasite stress. These values interact with men’s psychological war adaptation.

The parasite-stress model does not suggest that there is adaptation(s) that functions specifically in the context of the various types of political conflicts we have addressed (non-state war, revolutions and coups, terrorism, international war). Hence, there is no adaptation functionally designed for non-state war *per se*, or for coups. Rather, the model

implies that these conflicts are manifestations of moral psychological adaptation designed for historically adaptive in- and out-group relations.

Moreover, the model does not imply or require that these conflicts are adaptive currently. For example, it does not predict that intrastate conflict, on average, has a net benefit in promoting inclusive fitness of combatants in modern, large-scale civil war. In the model, the adaptive value is in terms of the moral psychological adaptation, and solely in evolutionary historical environmental settings that caused its evolution by direct selection. Modern human environments often differ greatly from the evolutionary historical settings that were responsible ultimately for the effective selection of human adaptations. Each of the types of conflicts we have treated may be currently adaptive or maladaptive at the individual level, depending on the circumstances.

Certainly, ours is not the first proposal that people have psychological adaptation that is designed to select the most relevant values for dealing with the Darwinian hostile forces in their local environment. This hypothesis is seen in the earlier writing of Alexander (1987), Wright (1994), Thornhill (1998), Hauser (2006), and others. The present chapter differs from these earlier works in proposing that important components of moral psychology are designed by selection in the context of parasite stress and function in adopting values conditionally based on local parasite stress levels.

As mentioned above, the parasite-stress model of conflict is compatible with the proposals in the scientific literature that men's sexually selected pursuit of high mate number affect positively men's decisions to engage in coalitional aggression (e.g., Low, 1993; Wrangham & Peterson, 1996; Buss, 2004; Kanazawa, 2009). Low (1990) reported that, across traditional societies, polygynous marriage systems are more frequent in

geographical regions of high parasite stress than in regions of low parasite stress. She also found that, in traditional societies, wife-capture from neighboring groups by warring men is most frequent under high parasite stress. This finding supports Low's hypothesis that high parasite stress intensifies sexual selection on males (i.e., increases the variance among men in access to mates with whom children are produced). In this case, the sexual selection intensity stems from parasites generating high phenotypic and associated genetic variance in male quality that is visible to females during mate choice. Hence, parasite stress, through its effect of enhancing polygyny and associated limitations on men's access to mates, may contribute to a net benefit of intergroup aggression to obtain out-group mates (Kanazawa, 2009).

We emphasize that the parasite-stress model of conflict is consistent with an important role for nepotistic coalitions in warring decisions (e.g., Low, 1993). Nepotistic adaptation is central to collectivism and hence, as we have explained, to cooperation in warring. Also, as we have discussed above, collectivism is more than nepotism: it includes assortative, in-group favoritism toward others who are not genetic relatives. Both of these aspects of collectivism, according to the model and its empirical tests, are related causally and positively to parasite stress.

We close by considering the implications of our findings for international aid directed at reducing the cycle of poverty and conflict in regions such as Africa, equatorial South America, and southeast Asia. We (Letendre *et al.*, 2010) and Bonds *et al.* (2010) proposed a disease trap, by which nations in regions with high levels of infectious disease become trapped in self-reinforcing poverty. Infectious disease depresses economic development through its negative effect on human capital, and this is reinforced as

poverty prevents efforts at disease reduction (Bonds *et al.*, 2010). Additionally, the xenophobia evoked by high intensity of infectious disease further diminishes the willingness of people to invest in public goods that will be shared across groups, such as economic and health infrastructure, and public goods such as municipal water sources and sanitation systems; and diminishes the willingness to engage in trade with neighboring groups for goods and technologies. Finally, the violent conflict that erupts among impoverished groups who are unwilling to seek cooperative solutions when inter-group competition arises further compounds the infectious disease problem as violent conflict causes death, disability, and disease beyond those killed directly in conflict (Ghobarah *et al.*, 2003).

Other researchers who have found that the incidence of violent conflict in war-torn regions such as Africa results from the same economic factors that predict conflict in any other region (e.g., Elbadawi & Sambanis, 2000) have recommended international aid targeted at building economic institutions in those regions. Considering the effects infectious diseases have on societies, Hotez and Thomson (2009) and we (Letendre *et al.*, 2010) instead recommended international aid targeted at control and elimination of tropical diseases. Based on our findings here and in Letendre *et al.* (2010), we advocate that this sort of targeted aid has the greatest potential to get directly to the root cause of poverty and conflict, and to disrupt the infectious disease trap that locks billions of people into poverty and conflict. Dunn *et al.* (2010) found that investment in public health can significantly negatively affect the prevalence of human pathogens; thus there is evidence that such directed investment in public health can be effective.

Further, we point out that it is in countries with high intensity of infectious disease that foreign aid directed at economic development may most frequently be misappropriated for the personal benefit of corrupt government officials. In previously unpublished analyses, we find that pathogen severity correlates strongly and positively with government corruption cross-nationally. Transparency International's Corruption Perception Index (http://www.transparency.org/policy_research/surveys_indices/cpi/) scores countries of the world based on expert assessment and opinion polls, and is scored with highest values corresponding to least corruption. Mean values for this index from 2005 to 2009 correlate strongly and negative with pathogen severity ($n = 178$ countries; $r = -.60$; $p < 0.001$), indicating that greater corruption is predicted by higher pathogen severity. It is widely recognized that foreign aid directed to corrupt governments is largely wasted (e.g., Burnside & Collar, 2004; Easterly & Pfutze, 2008); yet because the intensity of infectious disease causes both poverty and the establishment of autocratic (Thornhill *et al.*, 2009) and corrupt regimes, foreign aid directed at alleviating this poverty is necessarily directed toward countries where it is most likely to be misappropriated or otherwise squandered by corrupt officials. We suggest that direct economic aid may more readily be misappropriated, whereas aid directed at diminishing the intensity of infectious disease, such as delivery of vaccines or the construction of sewage and municipal water systems, may be more likely to provide the intended benefit to the people of these countries.

FUTURE DIRECTIONS

Given the asymmetry of terrorist acts, the logistic regression analysis that we used here may not be a valid test of the applicability of the parasite stress theory to the incidence of terrorism. The quality of available data is also a limiting factor. In the future, better quality data that more thoroughly distinguishes domestic from international terrorist incidents may facilitate the appropriate analyses. Such a dataset likely will be zero-inflated, like the other conflict datasets we examined here, and thus it may be necessary to find appropriate non-parametric methods to analyze the number of terrorist events experienced by countries. It may also be fruitful to develop a dataset that catalogues only domestic terrorist events that are supported by a terrorist organization, and which excludes events committed by individuals acting alone. Such a dataset may be more indicative of acts carried out by groups of people who share values (i.e. collectivist values) and motivations.

More generally, it may be useful to obtain temporal indicators of the intensity of infectious disease in countries of the world, to allow more thorough longitudinal analysis of the predictive power of infectious disease on conflict. The Contemporary Pathogen Severity Index used here assigns a single value to each country. We used this index in the longitudinal analyses in Letendre *et al.* (2010) based on the justification that the high correlation between this index and Gangestad and Buss' (1993) historical pathogen severity index ($r = 0.819$, $n = 27$, $p < 0.001$) indicates stability in the prevalence of infectious disease over time. Nevertheless, there is some temporal variation in the prevalence of infectious disease, with periodic outbreaks and epidemics.

According to the model we have proposed, temporal variation in the prevalence of infectious disease may temporally predict the onset of conflicts. Such a dataset of the timing of epidemics within countries of the world could be used to predict conflict onsets some number of years later. This longitudinal analysis may help tease apart the causal influence of disease on conflict from the endogenous effect of conflict increasing the severity of disease, such as with cholera outbreaks among refugee populations following civil war (although our longitudinal analyses controlled these effects to some extent; Letendre *et al.*, 2010). Additionally, such a temporal analysis may help reveal the mechanism by which infectious disease produces the associated changes in human values that cause increased frequency of conflict. Does an epidemic predict increased likelihood of conflict one or two years later? Or does an epidemic predict increased likelihood of conflict a generation later, as would be suggested by the hypothesis that prevalence of disease during childhood establishes the intergroup values that will be expressed throughout life (Fincher *et al.*, 2008; Thornhill *et al.*, 2009)?

Finally, we hope that in the future, larger cross-national samples of collectivism-individualism may become available. We selected the variable family ties for use in path analysis, because of its large sample size relative to other measures of collectivism. Even so, our sample size in our final model was marginal given the number of parameters estimated. Additionally, it appears that family ties may be primarily a measure of the ethnocentrism component of collectivism, and our final model suggests that ethnocentrism and xenophobia may play somewhat distinct roles in the relationship between infectious disease and conflict. Thus we also hope that in the future, a cross-national measure of xenophobia will become available, corresponding to the

ethnocentrism represented by family ties. These variables in combination may facilitate further path analyses that will clarify the mechanisms by which infectious disease leads ultimately to conflict.

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REFERENCES

- Abadie, A. (2006). Poverty, political freedom, and the roots of terrorism. *American Economic Review* **96**, 50-56.
- Abadie, A. & Gardeazabal, J. (2008). Terrorism and the world economy. *European Economic Review* **52**, 1-27.
- Alesina, A. & Giuliano, P. (2007). The power of the family. Discussion paper No. 2132. Harvard Institute of Economic Research, Harvard University, Cambridge, MA.
<http://post.economics.harvard.edu/hier/2007papers/2007list.html>
- Alesina, A., Ozler, S., Roubini, N. & Swagel, P. (1996). Political instability and economic growth. *Journal of Economic Growth* **1**, 189-211.

- Alexander, R.D. (1979). *Darwinism and Human Affairs*. University of Washington Press, Seattle.
- Alexander, R.D. (1987). *The Biology of Moral Systems*. Aldine de Gruyter, New York.
- Atran, S. (2003). Genesis of suicide terrorism. *Science* **299**, 1534-1539.
- Banks, A.S. (1979, updated). Cross-national Time Series Data Archive, Center for Social Analysis, State University of New York at Binghamton. Sept. 1979, updated.
- Barro, R.J. & Lee, J.W. (1994). Data set for a panel of 138 countries.
[http://www.nber.org/pub/
barro.lee/](http://www.nber.org/pub/barro.lee/)
- Baron, R.M. & Kenny, D.A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology* **51**, 1173-1182.
- Blee, K.M. & Creasap, K.A. (2010). Conservative and right-wing movements. *Annual Review of Sociology* **36**, 269-286.
- Bonds, M.H., Keenan, D.C., Rohani, P. & Sachs, J.D. (2010). Poverty trap formed by the ecology of infectious diseases. *Proceedings of the Royal Society B* **277**, 1185-1192.
- Burnside, A.C. & Collar, D. (2004). Aid, policies, and growth: Revisiting the evidence. *World Bank Policy Research*, Working Paper No. 3251.
- Buss, D.M. (2004). *Evolutionary Psychology: The New Science of the Mind*, second edition. Allyn and Bacon, Boston.
- Cashdan, E. (2001). Ethnocentrism and xenophobia: a cross-cultural study. *Current Anthropology* **42**, 760-765.
- Chagnon, N.A. (1997). *Yanomamö, Fifth Edition*. Harcourt Brace, Fort Worth.

- Cohen, J. (1988). *Statistical power analysis for the behavioral sciences*, second edition. Lawrence Erlbaum, Hillsdale.
- Collier, P. & Hoeffler, A. (2002). On the incidence of civil war in Africa. *Journal of Conflict Resolution* **1**, 13–28.
- Dunn, R.R., Harris, N.C., Davies, T.J., & Gavin, M.C. (2010). Global drivers of human pathogen richness and prevalence. *Proceedings of the Royal Society of London B* **277**, 2587-2595.
- Duntley, J.H. & Buss, D.M. (2009). The psychology of warfare. Paper at Human Behavior and Evolution Society Conference, Fullerton, CA, June 16-20, 2009.
- Easterly, W. & Pfutze, T. (2008). Where does the money go? Best and worst practices in foreign aid. *Journal of Economic Perspectives*, Working Paper No. 21.
- Elbadawi, I. & Sambanis, N. (2000). Why are there so many civil wars in Africa? Understanding and preventing violent conflict. *Journal of African Economies* **9**, 244–269.
- Ember, C. R. & Ember, M. (1992). Resource unpredictability, mistrust, and war. *Journal of Conflict Resolution* **36**, 242–262.
- Faulkner, J., Schaller, M., Park, J. H. & Duncan, L. A. (2004). Evolved disease-avoidance mechanisms and contemporary xenophobic attitudes. *Group Processes and Intergroup Relations* **7**, 333-353.
- Fearon, J.D. & Laitin, D.D. (2003). Ethnicity, insurgency, and civil war. *The American Political Science Review* **97**, 75-90.
- 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 B* **275**, 2587-2594.
- Fincher, C.L. & Thornhill, R. (In press.) Parasite stress promotes in-group assortativeness: The cases of strong family ties and heightened religiosity. *Behavioral and Brain Sciences*.
- Fincher, C.L., Thornhill, R., Murray, D.R. & Schaller, M. (2008). Pathogen prevalence predicts human cross-cultural variability in individualism/collectivism. *Proceedings of the Royal Society of London B* **275**, 1279-85.
- Gangestad, S.W. & Buss, D. M. (1993). Pathogen prevalence and human mate preferences. *Ethology and Sociobiology* **14**, 89–96.
- Gangestad, S.W., Haselton, M.G. & Buss, D.M. (2006). Evolutionary foundations of cultural variation: Evoked cultural and mate preferences. *Psychological Inquiry* **17**, 75-95.
- Gelfand, M.J., Bhawuk, D.P.S., Nishii, L.H. and Bechtold, D.J. (2004). Individualism and collectivism. In *Culture, Leadership, and Organizations: The GLOBE Study of 62 Societies*, (eds. R.J. House, P.J. Hanges, M. Javidan, P.W. Dorfman & V. Gupta), pp. 437-512. Sage Publications, Thousand Oaks.
- Ghobarah, H. A., Huth, P. & Russett, B. (2003). Civil wars maim and kill people—long after the shooting stops. *American Political Science Review* **97**, 189–202.
- Gleditsch, N.P., Hegre, H. & Strand, H. (2009). Democracy and civil war. In *Handbook of War Studies III*, (ed. M.I. Midlarsky), pp 155-192. University of Michigan Press, Ann Arbor.

- Gleditsch, N. P., Wallensteen, P., Eriksson, M., Sollenberg, M. & Strand, H. (2002).
Armed conflict 1946-2001: A new dataset. *Journal of Peace Research* **39**, 615-637.
- Gotschall, J. (2004). Explaining wartime rape. *The Journal of Sex Research* **41**, 129-136.
- Guégan, J.-F., F. Thomas, M.E. Hochberg, T. de Meeûs & F. Renaud. (2001). Disease
diversity and human fertility. *Evolution* **55**, 1308-1314.
- Hamilton, W.D. (1964a). The genetical evolution of social behaviour. I. *Journal of
Theoretical Biology* **7**, 1-16.
- Hamilton, W.D. (1964b). The genetical evolution of social behaviour. II. *Journal of
Theoretical Biology* **7**, 17-52.
- Hauser, M.D. (2006). *Moral minds: How nature designed our universal sense of right
and wrong*. Harper Collins, New York.
- Hegre, H. & Sambanis, N. (2006). Sensitivity analysis of empirical results on civil war
onset. *Journal of Conflict Resolution* **50**, 508-535.
- Hofstede, G. (2001). *Culture's consequences: Comparing values, behaviors, institutions,
and organizations across nations*, second edition. Sage Publications, Thousand Oaks.
- Hotez, P.J. & Thompson, T.G. (2009). Waging peace through neglected tropical disease
control: A US foreign policy for the bottom billion. *PloS Neglected Tropical Diseases*
3, 1-4.
- Klein, R.B. (1998). *Principles and Practice of Structural Equation Modeling*. Guilford,
New York.
- Kanazawa, S. (2009). Evolutionary psychological foundations of civil wars. *The
Journal of Politics* **71**, 25-34.

- Letendre, K., Fincher, C.L. & Thornhill, R. (2010). Does infectious disease cause global variation in the frequency of intrastate armed conflict and civil war? *Biological Reviews* **85**, 669-683.
- Low, B.S. (1990). Marriage systems and pathogen stress in human societies. *American Zoologist* **30**, 325-339.
- Low, B.S. (1993). An evolutionary perspective on war. In *Behavior, culture and conflict in world politics*, (eds. W. Zimmerman & H.K. Jacobson), pp. 13-55. University of Michigan Press, Ann Arbor.
- Low, B. S. (2000). *Why Sex Matters: A Darwinian Look at Human Behavior*. Princeton University Press, Princeton.
- MacNeill, A.D. (2004). The capacity for religious experience is an evolutionary adaptation to warfare. *Evolution and Cognition* **10**, 43-60.
- Manson, J.H. & Wrangham, R.W. (1991). Intergroup aggression in chimpanzees and humans. *Current Anthropology* **32**, 369-390.
- Marshall, M., & Jagers, K. (2009). Polity IV Project: Political regime characteristics and transitions, 1800-2007. <http://www.systemicpeace.org/polity/polity4.htm>
- Murdock, G.P. (1949). *Social Structure*. Macmillan, New York.
- Navarrete, C.D. & 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., Kurzban, R., Fessler, D.M.T, & Kirkpatrick, L. (2004). Anxiety and intergroup bias: Terror-management or coalitional psychology? *Group Processes and Intergroup Relations* **7**, 370-397.

- Nettle, D. (2009). Ecological influences on human behavioural diversity: a review of recent findings. *Trends in Ecology and Evolution* **24**, 618-624.
- Nettle, D., Grace, J.B., Choisy, M., Cornell, H.V., Guegan, J. & Hochberg, M.E. (2007). Cultural diversity, economic development and societal instability. *PLoS*, 9,1-5.
- Price-Smith, A.T. (2001). *The Health of Nations: Infectious Disease, Environmental Change, and Their Effect on National Security and Development*. MIT Press, Cambridge.
- Price-Smith, A.T. (2009). *Contagion and Chaos: Disease, Ecology, and National Security in the Era of Globalization*. MIT Press, Cambridge.
- Richerson, P.J. & Boyd, R. (2006). *Not By Genes Alone: How Culture Transformed Human Evolution*. University of Chicago Press, Chicago.
- Schaller, M. & Duncan, L.A. (2007). The behavioral immune system: Its evolution and social psychological implications. In *evolution and the social mind* (eds. J. P. Forgas, M.G. Haselton & W. von Hippel), pp. 293-307. Psychology Press, New York.
- Schaller, M. & Murray, D.R. (2008). Pathogens, personality, and culture: disease prevalence predicts worldwide variability in sociosexuality, extraversion, and openness to experience. *Journal of Personality and Social Psychology* **95**, 212-221.
- Schaller, M. & Neuberg, S. L. (2008). Intergroup prejudices and intergroup conflicts. In *Foundations of Evolutionary Psychology* (eds. C. Crawford & D. L. Krebs), pp. 399-412. Lawrence Erlbaum Associates, Mahwah.
- Simpson, J.A., & Gangestad, S.W. (1991). Individual differences in sociosexuality: Evidence for convergent and discriminant validity. *Journal of Personality and Social Psychology* **60**, 870-883.

- Singer, D.J. & Small, M. (1994). Correlates of war project: international and civil war data, 1816–1992. Data file, Inter-University Consortium for Political and Social Research, Ann Arbor.
- Sosis, R. & Alcorta, C. (2008). Militants and martyrs: Evolutionary perspectives on religion and terrorism. In *Natural security: A Darwinian approach to a dangerous world*, (eds. R. Sagarin & T. Taylor), pp. 105-24. University of California Press, Berkeley.
- Strand, H. (2006). Onset of Armed Conflict: A New List for the Period 1946–2004, with Applications. In Strand, H., *Reassessing the Civil Democratic Peace*, Ph.D. dissertation, Department of Political Science, University of Oslo & Centre of the Study of Civil War, PRIO.
- Streiner, D.L. (2005). Finding our way: An introduction to path analysis. *Research Methods in Psychiatry* **50**, 115-122.
- Suh, E., Diener, E., Oishi, S. & Triandis, H.C. (1998). The shifting basis of life satisfaction judgments across cultures: Emotions versus norms. *Journal of Personality and Social Psychology* **74**, 482–493.
- 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. (2005). Social roles, prestige, and health risk: Social niche specialization as a risk-buffering strategy. *Human Nature* **14**, 165-190.
- Tabachnick, B.G., & Fidell, L.S. (2007). *Using Multivariate Statistics, Fifth Edition*. Pearson Education, New York.

- Thompson, J.N. (2005.) *The Geographic Mosaic of Coevolution*. The University of Chicago Press, Chicago.
- Thornhill, R. (1998). Darwinian aesthetics. In *Handbook of evolutionary psychology: Ideas, issues, and applications* (eds. C. Crawford and D.L. Krebs), pp. 543-572. Lawrence Erlbaum Associates, Mahwah.
- Thornhill, R. & Palmer, C. (2000). *A Natural History of Rape: Biological Bases of Sexual Coercion*. MIT Press, Cambridge.
- Thornhill, R., Fincher, C.L. & Aran, D. (2009). Parasites, democratization, and the liberalization of values across contemporary countries. *Biological Reviews* **184**, 113-131.
- Thornhill, R., Fincher, C.L., Murray, D.R. & Schaller, M. (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.
- Trivers, R.L. (1971). The evolution of reciprocal altruism. *Quarterly Review of Biology* **46**, 35-57.
- West, S.A., Griffin, A.S. & Gardner, A. (2007). Social semantics: Altruism, cooperation, mutualism, strong reciprocity and group selection. *Journal of Evolutionary Biology* **20**,415-432.
- Williams, G.C. (1966). *Adaptation and Natural Selection*. Princeton University Press, Princeton.
- Wrangham, R. & Peterson, D. (1996). *Demonic males*. Houghton Mifflin, Boston.
- Wright, R. (1994). *The moral animal*. Vintage, New York.

Zhang, D. D., Brecke, P., Lee, H. F., He, Y. & Zhang, J. (2007). Global climate change, war, and population decline in recent human history. *Proceedings of the National Academy of Sciences* **104**, 19214–19219.

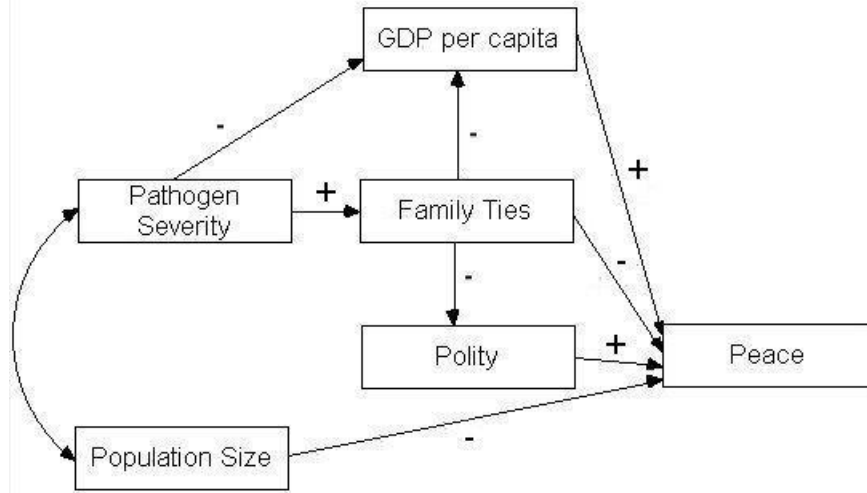
Table 1: Regression analyses of conflict by country

	Intrastate Armed Conflict	Non-State Wars	Revolutions and Coups	Terrorism	Global Peace Index
Log GDP <i>per capita</i>	0.05	-0.92*	-0.08	-0.59*	0.19*
Log population size	0.38*	1.17**	0.09	0.95***	-0.04
Democracy	0.08	.21	-0.56†	0.22	0.12
Political instability	0.02	.28	1.85**	0.34	0.0
GDP <i>per capita</i> growth	-0.33†	-0.02	-0.92†	0.27	0.19**
World Area	n.s.	n.s.	n.s.	n.s.	n.s.
Pathogen Severity	.71*	1.16*	1.09*	0.20	-0.48***
<i>N</i>	148	148	117	148	129

Standardized parameter estimates for predictors of conflict. Analyses of intrastate armed conflict, revolutions and coups, non-state wars, and terrorism are binary logistic fits. Analysis of the Global Peace Index is a least-squares fit.

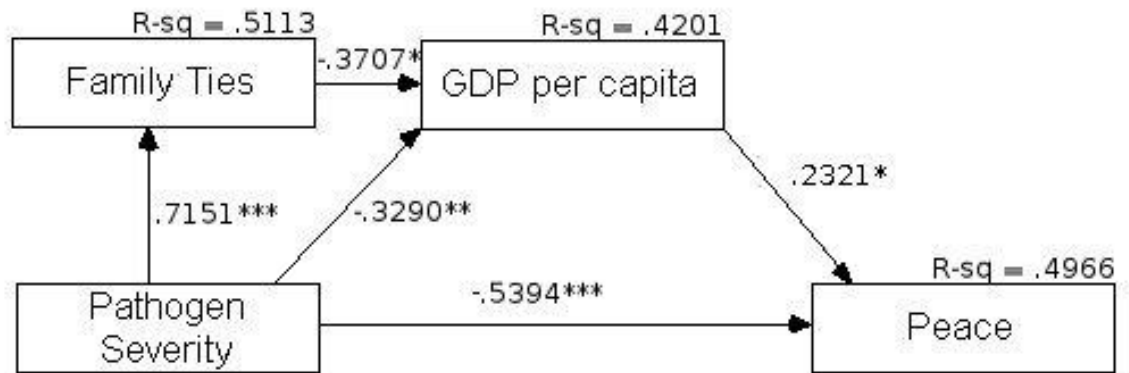
† $p < .10$; * $p < .05$; ** $p < 0.01$; *** $p < 0.001$; n.s., not significant. Significance values are one-tailed probabilities.

Figure 1: Hypothesized Structural Equation Model of the Global Peace Index



Minus signs (-) indicate hypothesized negative relationships. Plus signs (+) indicate hypothesized positive relationships.

Figure 2: Final Structural Equation Model of the Global Peace Index



Model fit statistics: $\chi^2 p = 0.3794$; RMSEA < 0.0001 ; NFI = 0.9969. Path weights are standardized coefficients. R-sq indicates proportion of variation accounted for by the model for each endogenous variable. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$

CHAPTER 4

EVOLUTION OF OPTIMAL OUT-BREEDING IN HOST-PARASITE COEVOLUTION GIVEN WITHIN-HOST COMPETITION BY PARASITES

K. Letendre

SUMMARY

A basic problem for long-lived organisms is escaping parasitism by short-lived parasite species, which rapidly adapt to avoid detection and elimination by host immune systems. I investigated the evolution of mate preferences that support optimal outbreeding as a response to the selective pressure of antagonistic host-parasite coevolution in a sexually reproducing host population. Previous studies have used stochastic computer models to investigate the evolution of sexual reproduction as a result of the fitness advantage of genetic recombination, as a mechanism for producing immune system diversity in offspring. These previous models have created systems in which maximal out-breeding is optimal, as sexually reproducing hosts seek to maximize genetic variation from one generation to the next in order to escape the fitness costs of their coevolving parasites. However, previous models have neglected the possibility that multiple strains of a given parasite species may arise due to localized coevolutionary processes, and that parasites from different strains may compete to infect a single host when a host is exposed to more than one strain of the same species of parasite. This risk of coinfection by novel strains of parasites, which may by chance be more virulent than the local strains of the same parasites, selects for optimal, rather than maximal, outbreeding to maximize the benefits

of recombination while preserving locally coadapted gene complexes and minimizing the cost of exposure to novel pathogen strains. I developed a spatially explicit model in which hosts and pathogens coevolve, while sexually reproducing hosts may select mates based on genetic difference (analogous to MHC mate preferences in humans and other mammals), but where intergroup matings allow the movement of local parasite strains into new host populations. I found that in models of both a single, well-mixed population, as well as metapopulation models, hosts expressed a preference for genetic dissimilarity in mates. Hosts in metapopulation models expressed a bias against selecting out-group mates, even when the risk of exposure to novel pathogens was eliminated, but a significantly stronger out-group bias with risk of exposure. I did not find the predicted positive relationship between parasite species richness and out-group avoidance; this may be because the pattern of increasing out-group avoidance in human populations is driven by variation in parasite virulence, rather than by the variation in species richness modeled here.

INTRODUCTION

There is a long history of stochastic computer models in the study of the evolution of sexual reproduction. Such models simulate antagonistic coevolutionary races between relatively long-lived host species and relatively short-lived parasite species. Numerous models have shown that the advantages of recombination in these coevolutionary races can outweigh the two-fold cost of sex, in which males of the species do not contribute to the production of offspring except as gene donors (e.g. Hamilton *et al.*, 1990; see review in Lively, 2010). These models have simulated communities of hosts and their

pathogens, in which host genes analogous to the major histocompatibility complex (MHC) are subject to selection for their ability to detect parasites. At the same time, the genes of pathogens are under selection to match the MHC make-up of hosts to avoid detection by the immune system and more effectively parasitize the host (e.g. Hamilton *et al.*, 1990). In studies of the evolution of sexual reproduction, alleles determining sexual or asexual reproduction in host species are also subject to selection in this coevolutionary system, and numerous studies have found that sexual reproduction becomes fixed in these populations due to the advantage of diversifying offspring MHC makeup in each generation, as recombination shuffles parental genes and produces offspring with greater ability to escape parasitism relative to asexually reproducing members of the population.

In these models, offspring genotypes that are maximally diversified are thought to be at the greatest advantage because they are maximally different from the existing genotypes in the population which the parasites have been adapting to exploit. These models have generally found that sexual reproduction is most heavily favored by selection in systems with greater numbers of parasite species. Thus one prediction of these models might be greater preference for outbreeding in environments with greater parasite species richness and greater burden of parasitism and infectious disease. That humans and other animals prefer MHC-dissimilar mates has been supported by some studies (e.g. Wedekind *et al.*, 1995; Ober *et al.*, 1997; Thornhill *et al.*, 2002) but this preference has not been found in other samples (e.g. Hedrick & Black, 1997; Ihara *et al.*, 2000; Jacob *et al.*, 2002; see review in Milinski, 2006). Recently a cross-cultural study of parasite prevalence and consanguineous marriage found that frequency of consanguineous marriage is positively predicted by parasite prevalence (Hoben *et al.*,

2011) which contradicts the prediction of mate preference for greater MHC dissimilarity in environments with greater parasite species richness. Hoben *et al.* (2011) propose that out-breeding breaks up coadapted gene complexes that confer resistance to local parasites, and that therefore optimal in-breeding must balance this cost against the benefit of out-breeding for MHC diversity. An additional mechanism, described below, may contribute to this pattern.

Another prediction derived from models of host-parasite coevolution is that parasites will on average be less virulent and less able to infect hosts to which they are not adapted (Kaltz & Shykoff, 1998). The frequency of host alleles increase and decrease cyclically as parasites adapt to new alleles so that they are disfavored; the corresponding parasite alleles follow the cyclical pattern of host alleles (*ibid*). Novel parasites have undergone an independent process of coevolution with a different population of hosts, and are unlikely to be in step with the cyclical pattern of a new population of hosts, and are therefore generally expected to be less virulent or avirulent. However, it is novel parasite associations such as ebola, bird flu, rabies, fox tapeworm, etc., that are sometimes the most virulent (Ebert & Bull, 2008).

Parasites coadapted with their hosts evolve toward an optimal level of virulence that allows the parasite to co-opt as much of the host's resources as possible without killing the host before transmission to a new one. The virulence of novel host-parasite associations is unpredictable, as it has not adapted to this optimal degree of virulence (Ebert & Bull, 2008). Despite the prediction that novel pathogens are on average less virulent, and despite the fact that the majority of novel host-parasite associations result in no infection because the parasite is not adapted to the host, occasionally they result in

extremely virulent infections. Given that increased virulence generally improves parasites' competitiveness within a host (Ebert & Bull, 2008; Mideo, 2009), exposure to the numerous sub-virulent or avirulent strains of parasite from a novel environment will not supplant the coadapted parasites a host carries, and therefore will not mitigate fitness cost of exposure to novel parasite strains in general. Meanwhile, exposure to occasional novel strains that are by chance highly virulent will impose high fitness costs. Thus, on average, exposure to novel pathogens is costly to hosts, even though these novel pathogens may only rarely be more virulent than the parasites to which the host is already exposed.

Even while mating provides fitness benefits in the form of shuffling offspring's immune system makeup to evade parasites, mating itself incurs a risk of exposure to parasites – both sexually transmitted and non-sexually transmitted – carried by potential mates. If organisms living in a metapopulation undergo independent coevolutionary processes with parasite strains within their group, then mating outside the group, although it may have advantages in terms of acquiring different MHC alleles from those available from potential mates within the group, comes with the risk of exposure to novel, unpredictably virulent parasites. Thus although genetic dissimilarity may generally be preferred in a mate, novel mates, carrying potentially virulent novel parasites, may be disfavored in environments with high parasite species richness, despite those mates' greater average genetic dissimilarity.

Many models of host-parasite coevolution have modeled well-mixed populations of hosts and parasites. A previous study (Ladle *et al*, 1996) used a metapopulation model to study the effect of both host and parasite migration rates on the evolution and

maintenance of obligate sexual reproduction. However this study modeled migration rates as a feature of the environment, rather than as a product of selection on the behavior of host organisms (such as mate choice behaviors) that produces variation in these rates. I studied how selection on preferences for in-breeding vs. outbreeding in sexually reproducing hosts influences the rate of migration of both parasites and host genes between subpopulations. The goal of this model is to reveal how host and parasite gene flow influence the evolution of out-group avoidance and the generation of genetic divergence across host subpopulations.

METHODS

Previous models of host-parasite coevolution have focused on the selection and maintenance of obligate sexual reproduction. In order to examine aspects of mate choice under host-parasite coevolution, I assume a population of obligately sexual host organisms, and a community of obligately asexual parasite organisms. As with previous models (e.g. Hamilton *et al*, 1990; Ladle *et al*, 1993; Howard & Lively, 1998), I will model host-parasite coevolution as follows:

Parasites are relatively short-lived but highly fecund, obligately asexual organisms that live on average one year. As in Hamilton *et al* (1990), parasites die at mortality rate $m_p = 0.909$ per individual per year and reproduce at the replacement reproductive rate $r_p = m_p/(1 - m_p) = 9.989$ per individual per year. Parasite genomes are composed of a string of 0's and 1's (zeros and ones) of length l , where each number represents a gene and 0 and 1 represent possible alleles for that gene. Each gene corresponds to a gene on the host genome, and a match between an allele carried by a

parasite to an allele carried by a host indicates ability of the parasite to evade the host immune system, and therefore to infect the host. Parasite fitness f_p is determined by its virulence within the host with which it is associated in a given year, i.e. the proportion of genes p at which it matches the corresponding genes of the host. There are n parasite species in an environment. I varied n from one to 20 across environments in order to model environments with variation in parasite species richness.

Hosts are relatively long-lived, obligately sexual organisms with relatively low fecundity. Each subpopulation has a carrying capacity $k = 300$ hosts. This is a slight departure from the model by Hamilton *et al* (1990), in which host and parasite populations were a constant 200 each year; this change allows for populations to fluctuate as disease epidemics pass through them (see below). Hosts have a juvenile period of $j = 13$ years, and die at mortality rate $m_h = 0.0714$ per individual per year; they therefore reproduce at replacement reproductive rate $r_h = m_h/(1 - m_h)^{j+1} = 0.2016$ per individual per year. Hamilton *et al* (1990) chose these parameter values to simulate hominid reproductive rates. Host genomes are composed of a string 0's and 1's with each digit corresponding to a digit in the genome of a parasite species in the environment. In addition, here hosts will have two genes that determine perceived value of or preference for a mate: d , which allows preference for or aversion to MHC dissimilarity; and b , an out-group bias which allows preference for or aversion to a mate based on the mate's originating subpopulation (i.e. in-group or out-group). (Mate choice decisions are described below.) Thus host genomes are of length $n*k+2$. Host fitness is determined by the proportion of genes with which it is mismatched with each parasite species i : $(1-p_i)$. Based on the reasoning that one highly virulent parasite strain can be lethal for a host

regardless of the other parasites the host carries, and thus a single highly virulent parasite can reduce a host's fitness by more than $1/n$, host fitness will be calculated as the product of these proportions for each of its parasites: for each 1 through n parasites a host carries, the host's fitness $f_h = (1-p_1)(1-p_2)\dots(1-p_{n-1})(1-p_n)$. (Many previous models (e.g. Hamilton *et al*, 1990, Gandon *et al*, 1996) have discounted host fitness based on proportion of match by parasite genomes additively rather than multiplicatively.)

At model initialization, a population of $j_h = k$ hosts, and $j_{pi} = k$ parasites of each parasite species i is created. The genomes of hosts and parasites are initially determined by randomly assigning either 0 or 1 to all immune genes. Host genes d and b are initialized by randomly selecting from a uniform distribution over the interval $[-2,2]$. A randomly selected individual from each parasite species is assigned to each host. The fitness of parasites and hosts is calculated as above, and as in Hamilton *et al*. (1990) the $m_p * j_{pi}$ least fit parasites of each species i in each subpopulation die. As in Hamilton *et al*. (1990) surviving parasites are selected at random to reproduce asexually, with each gene mutating to the alternate allele at mutation rate $m_p = 0.01$, until each species in each subpopulation is returned to a population size equal to j_h individuals (assuming the current number of hosts in a subpopulation determines the size of the parasite populations).

Hosts die probabilistically, with the probability of the death of each host k , $z_{h,k}$, determined according to the equation:

$$z_{h,k} = d_h * (j_h / k) * (f_h / f_{h,i}) \quad (1)$$

where j_h is the current number of hosts living in the population, k is the carrying capacity for the subpopulation, f_h is the mean fitness of all hosts across all

subpopulations; and $f_{h,i}$ is the fitness of host i . This causes the average probability of death for all hosts over all subpopulations to equal the host death rate d_h . The second term causes the number of hosts j_h in each subpopulation to tend toward the carrying capacity k ; as the number of hosts in a subpopulation decreases below k , the probability of further host death in that subpopulation the next year is decreased. The third term causes the probability of death to decrease with increasing host fitness relative to the total mean host fitness. Thus, equation 1 allows the number of hosts in a subpopulation to fluctuate below k when particularly virulent strains of pathogens spread through in epidemics, yet tends to prevent any subpopulation from going extinct entirely, as mortality is reduced when the number of hosts is below its carrying capacity. As mentioned above, I used $k = 300$ instead of the population size 200 used by Hamilton *et al* (1990) and this was because with $k = 200$, subpopulations occasionally go extinct due to large epidemic events; whereas with $k = 300$ – still not an unrealistic size for a primitive human group – this problem was eliminated.

Adult hosts then reproduce with probability at reproductive rate r_h . In order to express the mate preferences under selection here, each host chosen to reproduce is presented with a choice of eight potential mates: four randomly selected from within its own subpopulation, and one selected at random from each of four neighboring subpopulations. The mother will then determine the mate value v according to genetic distance from the potential mate, the proportion $(1 - p)$ of mismatched alleles across the entire genome, and its own mate preferences. Potential mates from within the same subpopulation are evaluated according to the formula:

$$v = d * (1 - p) \tag{2}$$

Potential mates from a neighboring subpopulation are evaluated according to the formula:

$$v = d * (1 - p) + b \quad (3)$$

Thus depending on the mother's particular preference as determined by d , a potential mate's perceived value may vary positively or negatively with genetic distance $(1 - p)$. Depending on the mother's degree of out-group bias b , the mother can favor or avoid mates from different subpopulations relative to mates from her own population.

The mother chooses the mate with the highest mate value v , with recombination rate $r = 0.5$ between adjacent loci to simulate the unlinked MHC loci in humans (Hamilton *et al*, 1990) with each gene mutated with probability equal to the mutation rate $m_h = 0.0001$. MHC genes are mutated by changing a value of 0 to 1, and vice versa. Mate preference genes d and b are floating point numbers, and are mutated by selecting from a normal distribution with mean equal to the current value of d or b , respectively, and a standard deviation equal to 0.1.

Following reproduction, new host-parasite associations are determined for the next year of simulation. For hosts that did not reproduce the previous year and for hosts, one individual from each species of parasite from within that subpopulation will be chosen to infect the host. For hosts that reproduced and chose a mate from within her own subpopulation, two individual parasites from each species in that subpopulation, their degree of virulence (proportion of alleles matching the hosts') is compared, and she is paired with the more virulent of the two. For hosts that chose a mate from a neighboring subpopulation, one individual from the host's subpopulation and the corresponding strain carried by the mate will be selected from each species of parasite, its

virulence is compared to the parasite of the same species carried by her mate (i.e., a parasite from the neighboring subpopulation), and she is paired with the more virulent of the two parasites. The simulation then begins a new year, randomly pairing pathogens and hosts, culling those with low fitness, and reproducing.

I ran each model for 10,000 years. This allows sufficient time for the model to establish the cyclical patterns of frequency-dependent selection on immune genes, and for mate choice preferences to equilibrate. I ran replicates of the model configured for a single subpopulation (i.e. a well-mixed model with no exposure of hosts to novel potential mates nor novel parasites.) I ran replicates with a 5 x 5 grid of subpopulations. I ran replicates with the number of parasite species varying from 1 to 20. Finally, in order to test the mechanisms underlying the evolution of out-group avoidance in this system, I ran replicates on a 5 x 5 grid where hosts may choose from potential mates from neighboring subpopulations, but even if mothers do choose these novel mates, they are not exposed to novel parasites.

I am primarily interested in how this system of host-parasite coevolution influences mate choice preferences, so I analyzed the resulting parameters determining mate choice produced at the end of the model run: the preference for genetic dissimilarity d , and out-group bias b . Because d causes mate value to vary linearly with genetic distance (equations 2 and 3) any positive d causes maximum mate value to correspond to maximal genetic distance, while any negative d causes maximum mate value to correspond to least genetic distance. Therefore in absence of b , the only important feature of d is whether it is positive or negative; the magnitude of d does not change the ordering of values for an array of potential mates. When considering b , its sign is

important, indicating a bias against (negative values), or in favor of (positive), choosing a mate from a neighboring subpopulation instead of a mate from the native group. The magnitude of b relative to that of d is also important: if the magnitude of b is small relative to d , b has little influence over the mating decision, and d dominates; on the other hand if b is relatively large, it dominates the decision and b is only a secondary concern. Therefore in my analysis of b below, I analyze a normalized $b_{norm} = b / n$.

RESULTS

The system quickly establishes the cyclical pattern of frequency-dependent selection of host immune genes and the corresponding parasite genes. In models with multiple subpopulations, host-parasite coevolution proceeds somewhat independently in neighboring subpopulations despite both host and parasite gene flow due to host interbreeding (See Figures 1a and 1b). Patterns with respect to mate preference are discussed below.

Single, well-mixed population

When the model was run with a single, well-mixed population in which hosts are exposed to neither novel mates nor novel parasites, as predicted, preference for genetic dissimilarity d , was significantly greater than zero (Binomial test: $n = 40$; $p < 0.001$). No runs produced a negative value for d ; in every run the preference was for genetic dissimilarity in mates.

In these runs, b_{norm} was not significantly different from zero ($p > 0.10$), producing equally many positive and negative values due to drift, as hosts never encountered

potential mates from outside the group, and there was therefore no selection for out-group bias.

Multiple subpopulations with parasite migration

In model runs with multiple subpopulations, with novel mates among mothers' mate choices, and with possible transfer of parasites from one group to another with intergroup mating, again, preference for genetic dissimilarity d was significantly greater than zero (Binomial test: $n = 140$; $p < 0.001$). Again, no runs produced a negative value for d .

Out-group bias b_{norm} was significantly less than zero ($p < 0.001$), indicating a significant bias against selecting novel mates. No runs produced a positive value for b_{norm} .

Contrary to the prediction that with increasing number of parasite species, out-group avoidance would increase, there was no relationship between parasite species number and out-group avoidance (Least-squares regression: $n = 140$; $p > 0.10$).

Therefore, I will restrict the remaining analyses of d and b_{norm} to runs with ten parasite species for consistency and ease of comparison.

The mean value of b_{norm} for runs with ten parasite species was -1.261 (95% CI: -1.639 to -0.883). Recall that as a normalized measure of the importance of out-group bias d , for hosts with values of $b_{norm} < -1.0$, mating with novel mates can never occur because out-group bias will always out-weigh any preference for genetic dissimilarity. In these runs, the proportion of matings between hosts from different subpopulations in the last year of the model run was 0.009 (note that although the mean value of b_{norm} was less

than -1.0, there was variation around this mean, both between runs, and among hosts within one model as a result of mutation, which allows for a small proportion of inter-group mating).

Multiple subpopulations without parasite migration

When hosts were allowed to select mates from neighboring subpopulations without risk of exposure to novel parasite strains, preference for genetic dissimilarity d was again significantly positive (Binomial test: $n = 40$; $p < 0.001$), and b_{norm} was again significantly negative ($p < 0.001$), indicating that mothers expressed a preference genetic dissimilarity and a preference for in-group mates. The mean value of b_{norm} was -0.532 (95% CI: -0.665 to -0.399). This was significantly different from the mean value of b_{norm} in runs where parasites did transfer between subpopulations (Two-sample t-test: $n = 80$; $p < 0.001$). See Figure 2 for a summary of out-group bias d_{norm} across the single-subpopulation, multiple-subpopulation, and no parasite migration model runs.

As a result of the reduced negative out-group bias in this sample relative to runs where parasites can migrate when inter-group matings occur, the proportion of pairings with mates from novel subpopulations, 0.254, was significantly greater than in runs where parasites transferred between subpopulations (Equality of proportions test: $n = 80$; $p < 0.001$).

DISCUSSION

I found that in a host-parasite coevolutionary system with a single, well-mixed population of hosts and parasites, hosts evolved an optimal mate preference for mates

with greatest genetic distance. This is consistent with predictions derived from the literature on host-parasite coevolutionary processes and the evolution of sexual reproduction in relatively long-lived hosts, which depends on the benefit of recombination of host genes to escape parasitism. This finding is also consistent with the preference for the scent of dissimilar/complementary MHC genes in potential mates that has been revealed empirically in humans and other animals.

When the model was run with a 5x5 grid of neighboring subpopulations and mothers selected from an array of potential mates both from within her subpopulation and from neighboring subpopulations, hosts evolved an optimal preference for greater genetic dissimilarity in a mate, but also a bias against selecting mates from novel subpopulations. This is despite the general increased genetic dissimilarity between two members of neighboring subpopulations as compared to two members of the same subpopulation.

Finally when mothers were able to select from both native and novel mates, but faced no risk of exposure to novel parasites when choosing novel mates, they again evolved an optimal preference for greater genetic dissimilarity, and still evolved a significant bias against selecting mates from outside the native subpopulation. This bias was significantly less in terms of magnitude relative to the preference for genetic similarity, than the bias that was optimal when mothers faced the additional risk of exposure to novel pathogens.

There are two, related costs in this system for mothers selecting a mate from outside her subpopulation. A cost that is relevant in both the multi-population models above results from the fact that to an important extent, members of the local population possess genes that are adaptive in the current parasite environment. As illustrated in

Figure 1, once the host-parasite coevolutionary process is established in a subpopulation, parasite genes cycle with host genes frequencies, but generally the cycle is shifted in time so that the parasites are always a step or two behind the host population in their pursuit of the hosts through genomic space. In this situation, the very fact that a potential mate is alive and surviving local parasite stresses is a certification that he carries genes that are reasonably well adapted to local parasites. In natural systems, females additionally evaluate potential mates by assessing other markers of health and condition, such as body symmetry (e.g. Thornhill *et al*, 2003) or colorful plumage (e.g. Hamilton & Zuk, 1983) or spots (e.g. Kodric-Brown, 1989; Milinski, 1990). In this model system, mothers have access to no such cues of health and freedom from parasites, but the fact that a mate is living in the local population indicates at least that he possesses the genes to survive the local parasites long enough to reach reproductive maturity. As a result, although there is a robust preference in this system for mate attraction to genetic dissimilarity, there is a bias against mating outside the local subpopulation, whose genes may be quite different, but are not adapted to local parasites.

The second cost to mating outside the local subpopulation in this system is the risk of exposure of the mother to novel parasite strains. This cost is indicated by the significantly higher out-group avoidance expressed by hosts when parasites can migrate between subpopulations when mothers choose a novel mate. Even if, on average, novel parasites are avirulent or sub-virulent to a novel host, the unpredictable virulence of novel pathogens makes exposure to them costly. Upon exposure to the complement of novel pathogens carried by a host coming from a different host-parasite coevolutionary history, many of those pathogens may be unable to infect the novel host; but if one of these

pathogens by chance possesses the genes to effectively evade and exploit the novel host's immune system, it will incur a high fitness cost regardless of the other parasite strains currently within the host. Even more, it is thought that in general more highly virulent parasite strains will be more effective at within-host competition with other strains (Nowak & May, 1989; reviewed in Mideo, 2009), and thus occasionally highly virulent novel strains of parasites should supplant other, native parasites within that host; following introduction, these more virulent strains should then spread throughout the population of related (and therefore genetically similar) hosts. Similar rapid spread of novel infectious agents has been observed in non-human primate social groups (Freeland, 1979).

It is impossible from this model to determine if the above two costs of breeding outside the subpopulation are additive, or whether they interact in some way. The optimal out-group bias expressed in models where no parasites migrated with out-group mating was $b_{norm} = -0.532$, compared to $b_{norm} = -1.261$ where there was the additional cost of exposure to novel pathogens. In the latter case, does the cost of acquiring genes that are not adapted to local parasites account for $\sim 4/10$ of the out-group bias, while the cost of exposure to novel parasites accounts for the remaining $\sim 6/10$? Or is there some interaction effect such that out-group bias increases non-additively? This model does not elucidate that question because there is no clear way to impose a risk of exposure to novel pathogens without host gene flow between populations – if there is a cost for selecting a novel mate but no possible benefit of acquiring mate's genes, the model can easily find an optimum b_{norm} that precludes out-group mating entirely, and the question is unanswered.

Despite finding the expression of out-group avoidance in a metapopulation model of host-parasite coevolution, I did not find the predicted relationship between increasing numbers of parasite species and out-group avoidance. This may be because it is not parasite species richness *per se*, that drives geographic and cross-cultural patterns in out-group avoidance and consanguineous marriage. In environments with climates conducive to parasite transmission, each parasite species is likely more virulent on average than in environments less conducive to transmission (May & Anderson, 1990; Ewald, 2004). More rapid transmission and resulting intense host-parasite races may then result in genetic divergence and ultimately speciation, leading to increased parasite species richness in tropical environments (Fincher & Thornhill, 2008). It may be that the increased virulence of parasites at low latitudes is the more important driver of geographical patterns in inter-group attitudes and mating preferences. An important extension to this model may be one in which time is broken into finer discrete units than a year, such that variation in host transmission rate may be modeled. Here, I assumed variation in species richness among populations, but in reality this species richness is a product of the increased transmissibility and resulting increased virulence of parasites in certain some.

This model likely over-states the out-group avoidance avoidance that results directly from parasitism in natural human or other animal populations. In this model, parasitism is the only source of variation in fitness. In natural human populations there are other benefits to out-group contact, such as trade, or exposure to new ideas or technology; on the other hand there are other costs, such as the risk of inter-group competition and violence, which may increase as increasing infectious disease pressures

impose costs on other, positive social exchange (Letendre *et al*, 2010). Nevertheless even where other possible benefits from out-group interaction and other factors are important contributors to fitness, in environments where infectious disease stress is high, the increased costs of mating outside the group demonstrated by this model, appear to shift the balance away from out-breeding and increase the rate of consanguineous marriage (Hoben *et al*, 2010).

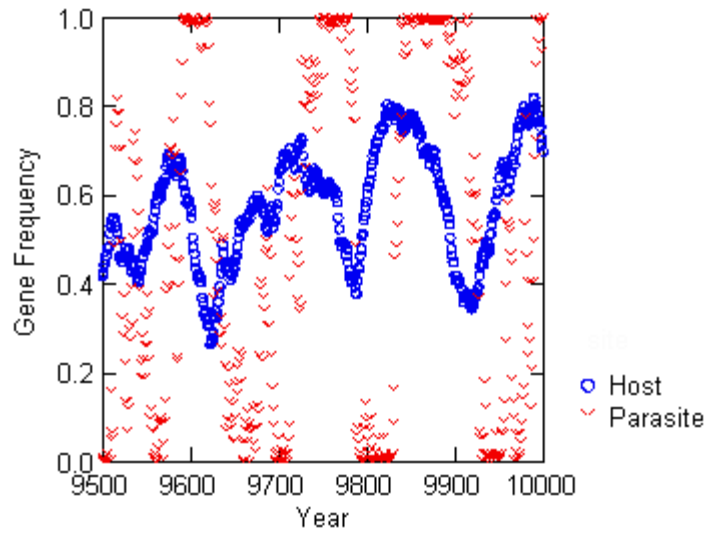
REFERENCES

- Ebert, D., Bull, J.J. (2008). The evolution and expression of virulence. In *Evolution in Health and Disease*, (eds Stearns, S.C. & Koella, J.C.), pp. 153-170. Oxford University Press, Oxford.
- Ewald, P.W. 2004. Evolution of virulence. *Infectious Disease Clinics of North America* **18**, 1-15.
- Fincher, C.L., Thornhill, R. (2008). A parasite-driven wedge: Infectious diseases may explain language and other biodiversity. *Oikos* **117**, 1289-1297.
- Hamilton, W.D., Axelrod, R. & Tanese, R. (1990). Sexual reproduction as an adaptation to resist parasites (A review). *Proceedings of the National Academy of Sciences* **87**, 3566-3573.
- Hamilton, W.D. & Zuk, M. (1983). Heritable true fitness and bright birds: a role for parasites? *Science* **22**, 384-387.
- Hedrick, P.W. & Black, F.L. (1997). HLA and mate selection: no evidence in South Amerindians. *American Journal of Human Genetics* **61**, 505-511.

- Hoben, A.D., Buunk, A.B., Fincher, C.L., Thornhill, R. & Schaller, M. (2010). On the adaptive origins and maladaptive consequences of human inbreeding: parasite prevalence, immune functioning, and consanguineous marriage. *Evolutionary Psychology* **8**, 658-676.
- Howard, R.S. & Lively, C.M. (1998). The maintenance of sex by parasitism and mutation accumulation under epistatic fitness functions. *Evolution* **52**, 604-610.
- Ihara, Y., Aoki, K., Tokumaga, K., Takahashi, K. & Juji, T. (2000). HLA and human mate choice: tests on Japanese couples. *Anthropological Science* **108**, 199–214.
- Jacob, S., McClintock, M.K., Zelano, B. & Ober, C. (2002). Paternally inherited HLA alleles are associated with women's choice of male odor. *Nature Genetics* **30**, 175-179.
- Kaltz, O. & Shykoff, J.A. (1998). Local adaptation in host-parasite systems. *Heredity* **81**, 361-370.
- Kodric-Brown, A. (1989). Dietary carotenoids and male mating success in the guppy: an environmental component to female choice. *Behavioral Ecology and Sociobiology* **25**, 393-401.
- Ladle, R.J., Johnstone, R.A. & Judson, O.P. (1993). Coevolutionary dynamics of sex in a metapopulation: escaping the Red Queen. *Proceedings of the Royal Society of London B* **253**, 155-160.
- Letendre, K., Fincher, C.L. & Thornhill, R. (2010). Does infectious disease cause global variation in the frequency of intrastate armed conflict and civil war? *Biological Reviews* **85**, 669-683.

- Lively, C.M. (2010). A review of Red Queen models for the persistence of obligate sexual reproduction. *Journal of Heredity* **101**: S13-S20.
- May, R.M., Anderson & R.M. (1990). Parasite-host coevolution. *Parasitology* **100**, S89-S101.
- Mideo, N. (2009). Parasite adaptations to within-host competition. *Trends in Parasitology* **25**, 261-268.
- Milinski, M. (1990). Female sticklebacks use male coloration in mate choice and hence avoid parasitized males. *Nature* **344**, 331-333.
- Milinski, M. (2006). The major histocompatibility complex, sexual selection, and mate choice. *Annual Review of Ecology, Evolution and Systematics* **37**, 159-186.
- Nowak, M.A. & May, R.M. (1994). Superinfection and the evolution of parasite virulence. *Proceedings of the Royal Society of London B* **22**, 81-89.
- Ober, C., Weitkamp, L.R., Cox, N., Dytch, H., Kostyu, D. & Elias, S. (1997). HLA and mate choice in humans. *American Journal of Human Genetics* **61**, 497-504.
- Thornhill, R., Gangestad, S.W., Miller, R., Scheyd, G., McCollough, J.K. & Franklin, M. (2003). Major histocompatibility genes, symmetry, and body scent attractiveness in men and women. *Behavioral Ecology* **14**, 668-678.
- Wedekind, C., Seebeck, T., Bettens, F. & Paepke, A.J. (1995). MHC-dependent mate preferences in humans. *Proceedings of the Royal Society of London B* **260**, 245-249.

1a:



1b:

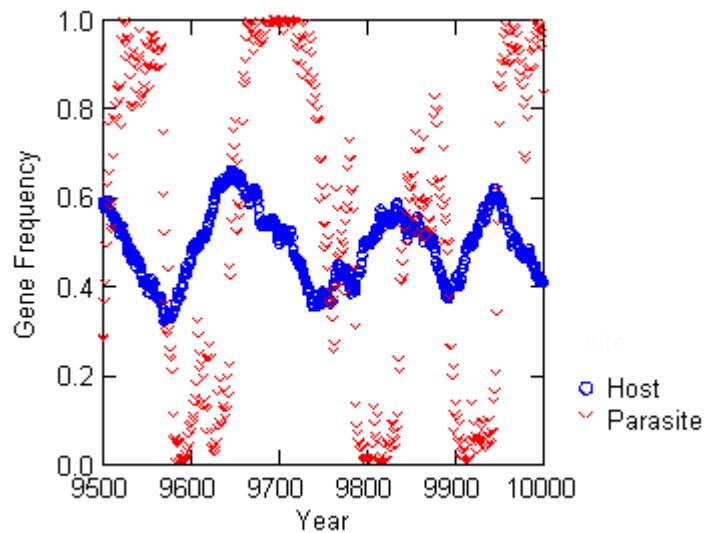


Figure 1: Parasite-host coevolution at a single gene locus. Host genes are selected for mismatch from parasites, while parasite genes are selected for match to host genes. 1a, Frequency-dependent selection causes cyclic variation over time in the frequency of host alleles, and a corresponding allele in a parasite species. 1b, independent coevolutionary process at the same gene locus in a neighboring community of hosts and parasites.

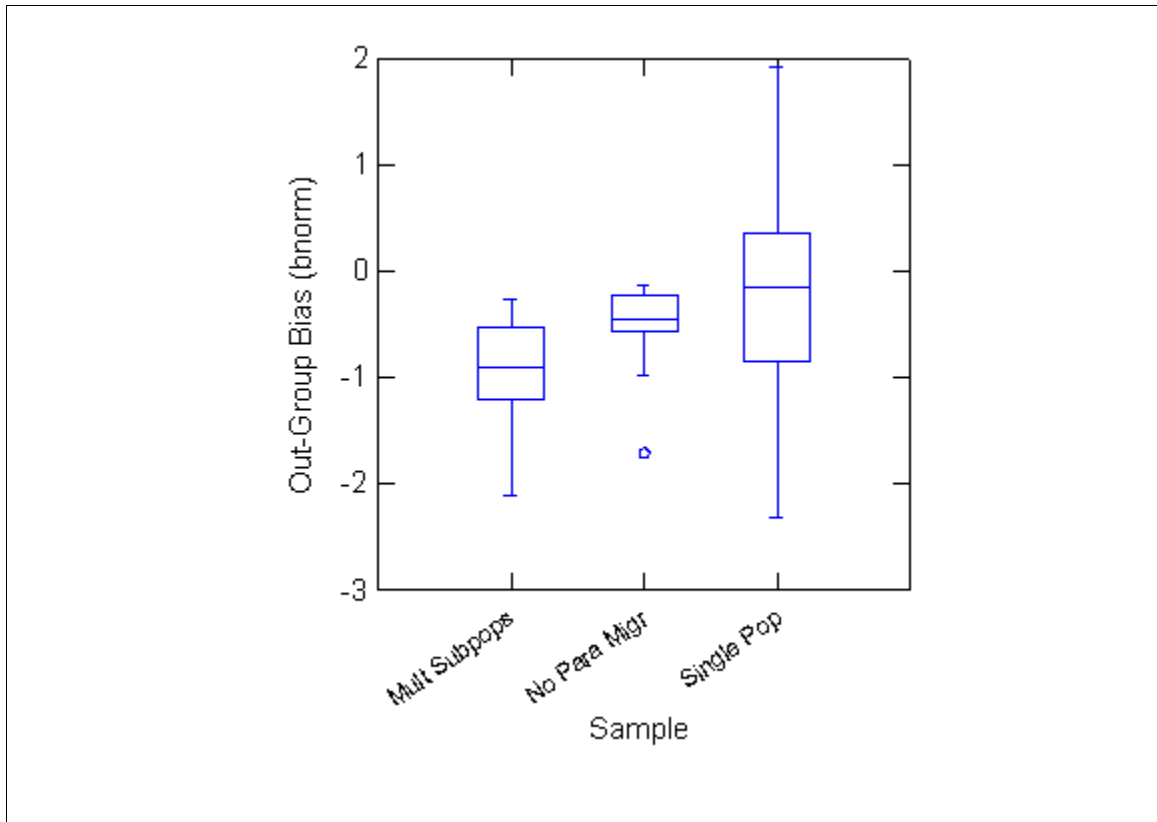


Figure 2: Summary of out-group bias, d_{norm} , produced in model runs of: 5x5 grid of subpopulations of hosts and parasites in which hosts may select mates from neighboring subpopulations, and parasites may migrate between subpopulations; 5x5 of subpopulations where hosts may select mates from neighboring subpopulations, but inter-group mating choices do not permit the migration of parasites; and a single, well-mixed population of mates and hosts, where hosts never encounter potential, novel mates, and no novel parasite species can immigrate.

CHAPTER 5

QUANTIFYING THE EFFECT OF COLONY SIZE AND FOOD DISTRIBUTION ON HARVESTER ANT FORAGING

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SUMMARY

Desert seed-harvester ants, genus *Pogonomyrmex*, are central place foragers that search for resources collectively. We studied how seed harvesters use information about their environment to improve the rate at which they collect seeds, an important fitness component, by observing foraging rates of three species of harvester ant varying over an order of magnitude in colony size. We found that foraging rates are significantly influenced by the spatial distribution of experimental seed baits: as bait sizes and densities were quadrupled, colonies collected seeds roughly doubled their foraging rate. However, the increase in foraging rate with bait density was indistinguishable across these three species, which differ substantially in forager population size, suggesting that species with larger colony sizes are no better than species with smaller colony sizes at collecting clumped seeds. These findings contradict the prediction that larger colonies are more efficient at exploiting heterogeneous resources. We discuss implications for our understanding of the evolution and maintenance of animal societies.

INTRODUCTION

According to optimal foraging theory, an animal's foraging behavior should maximize its net energy intake and minimize its costs (Stephens *et al.*, 2007; Stephens & Krebs, 1986). The distribution of food affects the optimal foraging strategy: many animals forage in groups when food is clumped, and individually when food is more dispersed (Caraco & Wolf, 1975; Hill *et al.*, 1987; Ryer & Olla, 1995). Group foraging ranges from the passive and uncoordinated, as when solitary foragers happen to congregate at scarce food patches, to the actively coordinated collection of food that either requires cooperation, because of its size (e.g. wolves preying on elk), or whose collection benefits from communication among group members.

Ants are master foragers, and studies suggest that some ant species (Formicidae) concentrate their foraging efforts on relatively clumped food, presumably because of the greater energetic returns on their foraging investments. The large-colonied seed harvester ant *Pogonomyrmex occidentalis* showed strong recruitment responses to clumped seed sources near *trunk trails* (Crist & MacMahon, 1991; Mull & MacMahon, 1997), which are semi-permanent trails that branch away from a colony toward food resources. Moving up in colony size, New World leafcutter ants (*Atta* and *Acromyrmex* spp.) create large trunk trails leading to individual trees from which they harvest massive volumes of leaves (Wilson & Osborne, 1971).

The ability of ant species with large colonies to efficiently harvest dense food sources attests to their sophisticated webs of communication (Wilson & Osborne, 1971; Beckers *et al.*, 1989; Anderson & McShea, 2001; Sumpter, 2006). For example, Beckers *et al.* (1989) found that in smaller colonies, individual foragers are less likely to

communicate to coordinate foraging, and larger colonies tend to form communication networks based on permanent trail-laying behavior. Large networks of foragers linked by chemical communication may allow large colonies to exploit food sources more efficiently because they enable more effective group decisions or because the presence of more individuals allows the mobilization of a larger, faster response (Anderson & McShea, 2001). In this study, we test two hypotheses. First, do *Pogonomyrmex* colonies collect food faster from larger, denser piles of seeds? Second, are colonies with larger forager populations more effective at exploiting large, dense seed piles relative to colonies with fewer foragers?

Seed harvester ants, *Pogonomyrmex* spp., are ideal for testing hypotheses about how food distribution and group size favor different foraging strategies. *Pogonomyrmex* are relatively large ants found mostly in arid regions of South, Central, and North America (Cole, 1968). They are well studied, monodomous, central-place foragers whose primary diet is local seeds found on the top of the soil (Hölldobler & Wilson, 2008; Carroll & Janzen, 1973; Bernstein, 1975; Davidson, 1977; Traniello, 1989; Gordon, 1991). Although all harvester ants eat seeds and often occur in sympatry, with colonies of several species often found within a few meters of each other, individual species differ in average body size, the size of seeds eaten, and average mature colony size (Bernstein, 1975; Davidson, 1977). The spatial distribution of seeds eaten by harvesters ranges from highly clumped to randomly dispersed. Reichman (1984) found extreme variability in the density of seeds eaten by *Pogonomyrmex* in the Sonoran Desert, with a 78-fold difference in seed density across space, including a 25-fold difference within microhabitats. Edeleman (2010) found

a three-fold increase in the density of seeds in soil surrounding kangaroo rat mounds in the Chihuahuan desert.

Although *Pogonomyrmex* individuals communicate and coordinate tasks in their underground nests, it is not clear whether foragers use communication networks, such as ones mediated by pheromone trails, as a foraging strategy. They are able to use pheromone trails to recruit foragers to large piles of seeds, as when supplied experimentally (Hölldobler & Wilson, 1970), but it is not clear that they commonly use pheromone recruitment under natural conditions. While some authors found evidence of recruitment (Davidson, 1977; Hölldobler, 1976; Whitford, 1978), others found that foraging is regulated by other behaviors (Gordon, 1991; Schafer *et al.*, 2006). For example, *Pogonomyrmex* foragers exhibit strong “directional fidelity,” returning to search for food in the same general direction as they successfully foraged before (Hölldobler, 1976; De Vita, 1975; Beverly *et al.*, 2009).

We predicted that harvester ant foragers would preferentially harvest dense seed patches, maximizing their efficiency by minimizing their search time within the patch. When a forager leaves its nest in search of food, it will travel in a general direction (Fewell, 1990), presumably established by a pheromone trail or by directional fidelity. The time it takes for an ant to arrive at a general area it expects to find food is the *travel time*. Once at the destination, the ant engages in a more localized search. The time it takes to find a patch of food in this general area is the *search time*. Once a patch of food is discovered, each trip to that patch requires the same travel time, but search is reduced. Beverly *et al.* (2009) showed that for harvester ants, search time has a much stronger effect on total trip duration than travel time. Thus, we hypothesize that clumps of seeds

will be collected significantly faster than randomly scattered seeds because the search time component of foraging time is reduced when an ant knows the location of a pile of seeds.

We observed colonies from three sympatric *Pogonomyrmex* species that vary substantially in forager population size and investigate how forager population size affects foraging rates for seeds in different spatial distributions. We posit that the foraging patterns we observed reflect underlying behavioral mechanisms. Understanding how foraging rates depend on food distribution and colony size may guide future studies of behavioral strategies in these and other ants.

Our two key variables were average forager population size and the spatial distribution of seeds. We estimated average forager population size in each species, experimentally manipulated seed distribution by offering colonies seed baits, and measured how these affect foraging rate.

The three species of harvester ants we studied, *Pogonomyrmex rugosus*, *P. maricopa*, and *P. desertorum*, vary substantially in total colony population size. *Forager population size* varies substantially between species, between colonies of the same species, and over time for any particular colony. Johnson (2000) estimated total colony population sizes in the Chihuahuan Desert of New Mexico as thousands for *P. rugosus*, a few hundred for *P. maricopa* and fewer than 100 for *P. desertorum*. Whitford and Ettershank (1975) estimated colony forager populations of dozens in *P. desertorum* and thousands in *P. rugosus*. Since not all members of the colony actively forage, total colony population is an upper bound on forager population size.

We manipulated the distribution of seeds by providing each colony with seeds dispersed over a broad range of pile sizes, ranging from one to 256 seeds, to establish a quantitative relationship between the distribution of seeds and the foraging rate. We then compared the relationship between food dispersion and foraging rate across these species, with their different forager numbers, to determine whether larger colonies concentrate relatively more foraging effort on clumped seeds when compared to smaller colonies.

MATERIALS AND METHODS

We studied three sympatric species of desert seed-harvester ants in the genus *Pogonomyrmex* in the high desert of central New Mexico: *P. desertorum*, *P. maricopa* and *P. rugosus*. We carried out this fieldwork in the summer of 2008 and 2009 in a mid-succession lot of approximately 13 hectares in central Albuquerque, New Mexico, in the Chihuahuan desert of the southwestern U.S. No permits were required for the described field studies.

Estimating active forager population size

We relied on data and methods from an earlier study (Moses, 2005) to estimate the average number of active foragers for each species. Preliminary estimates of forager numbers per colony and per-ant foraging distances and times characterizing each species were based on observation of colonies in the McKenzie Flats area of the Sevilleta Long Term Ecological Research site in central New Mexico. We carried out these observations in the summers of 2003 and 2004; we followed 63 individual ants from 13 colonies.

For each forager, we made the following observations. Individual foragers were followed as they left the nest, traveled to a search location, searched for and acquired a

seed, and returned to the nest. We marked some foragers either with paint (DecoColor opaque paint □ Uchida of America) or colored chalk powder; others were followed and left unmarked. For each forager we measured the time to complete a foraging trip (T_f) from nest to seed and back to the nest, the linear distance from nest to seed (d_s) and travel velocity (v_t) of each forager returning with the seed to the nest. The measurements are reported in Table 1.

One method of estimating the number of active foragers size is to multiply the average time of a foraging trip (T_f) by the rate that ants return to (and leave from) the nest when the rates of leaving and returning ants are at equilibrium. At equilibrium the number of foraging ants (F) is constant. The equilibrium rate of ants leaving (which is equal to the rate ants return) multiplied by T_f provides an estimate of active forager population at a particular time: the active forager population (F) equals the time of a foraging trip (T_f) multiplied by the rate that ants leave the nest. We estimated the number of foragers per species at the Sevilleta in 2004 using this method. We calculated the equilibrium rate by counting each ant leaving the nest for three minutes and each ant returning for 3 minutes. When these numbers differed by less than 10 percent, we considered that an equilibrium flux of ants (Table 1). We multiplied that number by average foraging trip time (T_f) to get the active forager population for that day (F , Table 1).

Manipulative Foraging Observations

In order to measure the effect of seed dispersion on foraging rates, we conducted manipulative field experiments on the three *Pogonomyrmex* species in the summers of

2008 and 2009. We began observations each morning to coincide with the start of daily foraging activity. We account for daily variations in colony activity, which may affect forager numbers (Gordon, 1991), by using the distribution of randomly scattered bait seeds to normalize our observed foraging rates (see *Data analysis* section for more details). The normalized foraging rate also allowed us to make comparisons across species and conditions that varied widely.

For each observation, we selected an active colony and baited it with dyed seeds arranged in a wide ring around the colony entrance (Figure 1). We placed dyed seeds in four distributions of different colors, equal in number of total seeds, but varying in degree of dispersion: one pile of red seeds, purple seeds divided into four piles, green seeds divided into sixteen piles, and a random scattering of blue seeds. Regardless of the pile size, we distributed the seeds in every pile evenly over a 10x10 cm² area. As soon as the first seed was placed, a starting time for the observation was marked. We immediately began observing the arrival of seeds at the nest entrance, recording the color – and thus the distribution from which each seed was collected – and the time of arrival.

We conducted 38 field observations, 11 of which we excluded because the focal colony failed to find at least two seeds from at least one experimental distribution during the observation period. This left nine observations of each species.

We dyed bait seeds using food coloring. We tested for bias in the collection or observation of bait seeds of different color by observing colonies of each species foraging in piles of bait seeds of mixed colors, with equal numbers of the four colors in a single pile.

We arranged our experimental seed distributions so as not to disadvantage small colonies. By placing fewer seeds closer to smaller colonies with smaller average foraging distances, we attempted to provide an equal opportunity for foragers from colonies from each of the three species to encounter one of our experimental seeds (Table 1). We chose a number of seeds roughly proportional to the forager population size: 1024 seeds for *P. rugosus* and 128 seeds each for *P. desertorum* and *P. maricopa*. We adjusted the distances of baits from the nest entrances in order to obtain a similar density of seed patches on the focal ant colony's territory, by placing the baits in a ring 5-7m from each *P. rugosus* colony, 2-4m from each *P. maricopa* colony and 1-3m from each *P. desertorum* colony (Table 2). Because the distance that a forager typically travels does not increase linearly with the number of foragers in a colony, it was not possible to simultaneously keep the density of seeds constant and the distance from nest to seed precisely proportional to typical forager travel distance.

P. desertorum foragers, with the smallest body size, frequently had difficulty handling the hulled millet with which we baited the other species. Because recruitment responses may be reduced with excessive handling times of large grains (Hölldobler, 1976), we baited *P. desertorum* colonies with sesame seeds. All three species readily collected experimental seeds whenever they encountered them.

After placing the experimental baits, an observer recorded the color of each seed brought into the nest with a time stamp using a computer program we created. For each experimental observation, we generated a time series for each distribution (a set of cumulative curves representing four time series in one observation is shown in Figure 2) We concluded observations when a focal colony ceased foraging or when ants had

collected all experimental baits, usually between 60 and 90 minutes after the start-time of the observation.

Data Analysis

We produced five time series from each observation, one each for seeds from each experimental seed distribution (color) and one for naturally occurring seeds. We calculated the rate each clumped distribution was collected relative to the random distribution.

We measured seed collection rates in two time periods. The first time period was measured from the placement of seeds (the start of the experiment) to the time that the first seed from each distribution was collected. This *discovery time* is the amount of time for an ant to find a seed from each distribution. The second time period was measured from the discovery of the first seed of a distribution to the last seed collected from that distribution. We call the rate at which seeds were collected once a distribution was discovered *foraging rate*. In Table 3 we report, for each species, the discovery times and the foraging rates for each distribution. We focus our analysis on the foraging rate, which measures the foraging efficiency once an ant knows the location of one seed from a distribution. We calculated the foraging rate for each distribution by dividing the number of seeds collected from that distribution by the time elapsed between the collection of the first and last seeds of that distribution.

The foraging rate for randomly scattered bait seeds served as a baseline seed collection rate for each colony during each foraging period. We calculated a *normalized rate* by dividing the foraging rate of each piled distribution by the foraging rate of

randomly scattered seeds for each observation. This ratio allowed us to quantify how much faster clumped seeds were collected relative to randomly scattered seeds and to compare foraging rates across variable colony activity levels, conditions, and colonies varying in forager number within species. In order to correct for skew in our field data, we \log_2 -transformed the normalized rates to obtain a normal distribution.

We analyzed both foraging rates and \log_2 -transformed normalized rates using repeated measures general linear model (GLM) (PASW Statistics, v. 18.0.1). Repeated measures analysis accounts for the non-independence of multiple measures taken of a single focal colony. Repeated measures also provide greater statistical power in this case by controlling for variation in activity level among our focal colonies, allowing us to distinguish within- and between-subject effects. We included species as a between-subject factor in these analyses, as a proxy for forager population size. Thus we use two independent variables, species and seed distribution, to predict our dependent variable \log_2 -transformed normalized rate

RESULTS

We estimated an active forager population (mean \pm standard error) of 71 ± 341 for *P. desertorum*, 269 ± 185 for *P. maricopa*, and 356 ± 211 *P. rugosus* in our 2009 study. The *P. maricopa* and *P. desertorum* estimates are similar to those estimated in earlier years at the Sevilleta NWR (Table 1), but the *P. rugosus* estimates are significantly lower in our experimental study in Albuquerque in 2009.

The test for bias in the collection or observation of bait seeds of different color showed no bias by color in the order of arrival of seeds at the focal nests (Kruskal-Wallis test: $n=802$ seeds; $p > 0.10$).

Figure 2 shows the cumulative number of seeds collected over time for each distribution in one field experiment. The graph depicts a single, typical observation in which more dispersed seeds are discovered faster than clumped. The x-intercept indicates the time to discovery for each distribution. These discovery times were unaffected by species identity (Repeated measures GLM: $p > 0.05$), but not surprisingly, were longer in the more clumped distributions across all species ($p = 0.002$, Table 3).

Averaged over all species, discovery times for random seeds (13.00 ± 2.02 minutes) and 16-pile distributions (14.19 ± 2.23 minutes) were indistinguishable, and discovery times for single-pile (22.83 ± 3.51 minutes) and four-pile distributions (22.59 ± 3.42 minutes) were indistinguishable.

Once an ant discovers an experimental pile, the time for a forager to search for additional seeds from that pile is negligible. Our analysis focused on the foraging rate: the rate at which ants collect seeds from a distribution once they have discovered a pile in that distribution. We measured foraging rates of naturally occurring seeds as well as rates for each colored seed distribution (Table 4). The mean foraging rates for natural seeds are similar to those for our baits, indicating that our measured rates are not an artifact of baiting the ants with extraordinary amounts of food. Figure 3 shows mean foraging rates for piled and randomly distributed bait seeds collected during our field observations.

Repeated measures analysis shows a significant difference in foraging rates between species (Repeated measures GLM: $p < 0.001$), indicating that species with larger colonies have greater absolute foraging rates, and a significant difference in foraging rates between pile sizes within species ($p < 0.001$). Within each of the three species there is a decreasing trend in foraging rate as seeds are dispersed across more piles. Paired

comparisons of foraging rates found significant differences from the four-pile, 16-pile, and random to the one-pile distribution for *P. rugosus* (Paired t-tests: $p=0.008$, 0.011 and 0.009 , respectively) and *P. desertorum* ($p = 0.004$, 0.025 and 0.012 , respectively), but no significant differences for *P. maricopa* (all $p > 0.10$).

Figure 4 shows the *normalized rates*: the foraging rate from each of the three piled distributions divided by the foraging rate for randomly distributed seeds within each observation. These data were log-transformed to obtain normal distributions (Shapiro-Wilk test: $p > 0.10$ after transformation). Note that, after \log_2 -transformation, a value of 0.0 for normalized rates indicates that seeds from a piled distribution are collected at the same rate as randomly distributed seeds, and a value of 1.0 indicates that piled seeds are collected twice as fast as random.

Repeated measures analysis of the log-transformed normalized foraging rates revealed no between-subjects effect of species (Repeated measures GLM: $p > 0.10$) indicating that species do not treat piled foods in general differently from one another; nor a within-subjects species x seed distribution interaction ($p > 0.10$), indicating that species do not treat piles of different sizes and densities differently from one another. We did find a significant within-subjects effect of distribution on normalized rates within species ($p = 0.003$). According to paired t-tests, normalized rates for four-pile and 16-pile distributions are significantly different from the one-pile distribution for *P. rugosus* (Paired t-test: $p = 0.028$ and 0.021 , respectively) and for *P. desertorum* ($p=0.001$ and 0.15 , respectively). As was the case with foraging rates, paired comparisons revealed no significant differences between distributions for *P. maricopa* (all $p > 0.10$).

Because we found no effect of species on the normalized foraging rates, we combined data from all three species and found that the normalized foraging rates declined significantly from the one-pile to the four-pile (Paired t-test: $p = 0.004$) and 16-pile ($p < 0.001$) distributions. The combined marginal means \pm standard error of \log_2 -transformed normalized rates are 1.2 ± 0.2 , 0.5 ± 0.2 and 0.3 ± 0.1 for one-pile, four-pile and 16-pile distributions of seeds, respectively. These \log_2 -transformed rates indicate that foraging rates decrease as seeds are more clumped such that seeds are collected roughly twice as fast from piles that are four times larger and denser. Table 4 summarizes the marginal means for foraging rates and normalized foraging rates for all species and distributions.

DISCUSSION

We observed foraging by three sympatric species of *Pogonomyrmex* on experimentally manipulated seed distributions, and quantified the effect of seed distribution and forager population size on foraging rate. As predicted, more densely clumped distributions were collected faster by all ant species (Figure 4), suggesting that all species reduced foraging times on clumped distributions, minimizing the cost of searching for seeds. However, given theoretical differences in foraging strategies in larger colonies (Wilson & Osborne, 1971; Beckers *et al.*, 1989; Anderson & McShea, 2001; Sumpter, 2006; Bourke, 1999), we were surprised to find no evidence that colonies with large forager populations collected clumped seeds relatively faster than smaller colonies.

Together, the three species we studied systematically increased foraging rates on seeds in more clumped distributions. Normalized foraging rates for piled distributions

increase from 0.3 to 0.5 to 1.2 as the heterogeneity of the seed distribution increases from 16 piles to four piles to one pile; seeds collection rate roughly doubles for seed piles that are 4 times larger and denser. Ants exploit more heterogeneous food distributions to improve whole colony foraging rate.

The rate at which ants collect seeds is a function of two processes — the time for the ants to discover a patch and the time it takes to collect seeds once the patch is found. For all species, the time to discover more dispersed (random and 16-pile distributions) seeds was faster than the time to discover more clumped (four- and one-pile distributions) seeds. However, once those piles were discovered, clumped seeds were collected significantly faster than the dispersed seeds. We analyzed the rate at which ants collected seeds from each piled distribution relative to randomly scattered seeds and this normalized foraging rate indicated how much faster foraging occurs once a colony knows the location of one seed from a distribution. The normalized foraging rate also accounts for differences in the number of active foragers in a given day and allowed us to make comparisons across species and conditions that varied widely.

Not surprisingly, colonies of species with more foragers collected a seeds at a greater absolute rate (Figure 3). However, repeated measures analysis showed no effect of species on normalized foraging rates. Because prior work suggests that larger colonies are more likely to use some form of group recruitment, we hypothesized that large colonies might be disproportionately good at collecting seeds from large piles. However, colonies collected seeds from large piles faster than seeds from small piles, regardless of colony size. This study suggests that large and small colonies of *Pogonomyrmex* allocate relatively similar numbers of foragers to large piles to collect them faster. Figure 4 shows

that the increase in foraging rate with pile size is indistinguishable for large and small colonies. However, these results should be interpreted in the context of our study design.

We controlled for colony territory size and for the distance that foragers travel to look for food by placing seeds closer to smaller colonies, giving large and small colonies equal opportunity to access the seed piles. However, this resulted in a higher density of piles in the territories of species with small colonies compared to the density of piles for larger colonies. In natural settings, it is possible that large colonies more often exploit large piles because their larger territories more frequently contain large piles.

This study does not reveal the specific foraging behaviors that these ants employ to collect clumped seeds faster, but we suggest that two strategies are plausible. Clumped seeds in our study could have been collected faster by group recruitment, or they may be collected faster as a result of a behavior called site fidelity.

Larger piles are collected faster with group recruitment because more ants are attracted to the site via pheromones. If the ants in our study were using group foraging, it would be surprising that larger colonies do not recruit more ants to the larger piles, since previous work on other ant species that shows that large colonies with large numbers of ants and sophisticated communication networks recruit more effectively (Beckers *et al.*, 1989; Anderson & McShea, 2001; Beverly *et al.*, 2009). It is possible that the ants in our study do use some sort of group recruitment, but allocate only a small number of additional foragers to collect from even very large piles. If large and small colonies each allocate a similar small number of foragers to collect from large piles, this could explain why large and small colonies forage equally fast on large piles.

Other researchers have hypothesized that seed harvesters rarely or never use pheromone recruitment in nature because seeds are distributed heterogeneously over time rather than over space (Gordon, 1991; Fewell, 1990). Further, *Pogonomyrmex* use a site fidelity behavior – individual foragers repeatedly return to the last location that they found a seed (Crist & MacMahon, 1991; Beverly *et al.*, 2009). This foraging behavior allows ants to exploit large piles faster because a single ant repeatedly returning to the same pile reduces its search time. Site fidelity may be sufficient to collect piles of seeds quickly (Beverly *et al.*, 2009; Buchkremer & Reinhold, 2008). For seed piles small enough that a single ant can collect all the seeds in a patch before the colony ceases foraging activity for the day, there may be no benefit in recruiting other foragers to that pile.

If ants primarily use site fidelity and not recruitment, then we would expect large and small colonies to be equally capable of collecting large piles faster, as we saw in our field study. However, in the case of a pile so large that the seeds cannot be collected by a single ant in a foraging period, or when seeds might be taken by competitors if they are not collected rapidly, recruitment of other ants to the site may be much more beneficial. In other work, Letendre and Moses (in review), use an agent-based model to show that pheromone recruitment results in increased foraging rates on more clumped distributions, although that study suggests that pheromone recruitment alone results in lower normalized foraging rates on piles than we observed in our field study. Site fidelity instead of or in addition to pheromone recruitment may provide an alternative or complementary explanation for how these seed harvesters collect large piles of seeds faster. In future modeling work we will explore how the processes of site fidelity and

pheromone recruitment may each contribute to the ants' exploitation of seeds in different distributions.

While our findings suggest no differences in foraging strategy among these species, this stands in contrast to descriptions of interspecific variation within *Pogonomyrmex* in foraging strategy in the literature (e.g. Johnson, 2000). It is possible that smaller colonies are capable of the foraging strategies that allow them to exploit more densely distributed foods when given the opportunity to do so, even though larger colonies more often have opportunity by virtue of their larger territory size, given random placement of patches of food in the environment.

Further studies specifically designed to measure foraging rate given the same distribution of seeds for all colonies are warranted, particularly since native seed distributions are not adjusted so that more small piles occur closer to small colonies. The effect of colony size might be very different given the same distribution of food for all colonies or given competition for food between colonies of different sizes. Since colony size has profound effects on colony life history (Shik, 2008; Hou *et al.*, 2010) and foraging strategy (Anderson & McShea, 2001; Jun *et al.*, 2003) this should be a fruitful area for further study.

Our study shows that ants from three *Pogonomyrmex* species systematically increase foraging rates as seeds are clumped into fewer, larger piles. The species differ substantially in colony size and forager population, but the increase in foraging rate with colony size is consistent across all three species. The increase in foraging rate on more dispersed distributions is surprisingly slow – roughly doubling as the number and density of seeds are quadrupled. Other foraging studies (e.g. Deneubourg *et al.*, 1990) suggest that

foragers of heavily recruiting ants converge very quickly on rich resources. This may suggest that seed harvesters, which forage on resources that remain relatively static over the course of a foraging period, spend more time exploring for new seeds rather than exploiting known piles of seeds. Understanding how different species of ants balance the trade-off between exploiting known resources versus exploring for new ones may improve understanding of foraging behavior in other animals that forage collectively.

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REFERENCES

- Anderson, C. & McShea, D.W. (2001). Individual versus social complexity, with particular reference to ant colonies. *Biological Reviews* **76**, 211-237.
- Beckers, R., Goss, S., Deneubourg, J.L. & Pasteels, J.M. (1989). Colony size, communication, and ant foraging strategy. *Psyche* **96**, 239-256.
- Bernstein, R.A. (1975). Foraging strategies of ants in response to variable food density. *Ecology* **56**, 213-219.

- Beverly, B.D., McLendon, H., Nacu, S., Holmes, S. & Gordon, D.M. (2009). How site fidelity leads to individual differences in the foraging activity of harvester ants. *Behavioral Ecology* **20**, 633-638.
- Bourke, A. (1999). Colony size, social complexity and reproductive conflict in social insects. *Journal of Evolutionary Biology* **12**, 245-257.
- Buchkremer, E.M. & Reinhold, K. (2008). Sector fidelity - an advantageous foraging behavior resulting from a heuristic search strategy. *Behavioral Ecology* **19**, 984.
- Caraco, T. & Wolf, L.L. (1975). Ecological determinants of group sizes of foraging lions. *American Naturalist* **109**, 343-352.
- Carroll, C. & Janzen, D. (1973). Ecology of foraging by ants. *Annual Review of Ecology and Systematics* **4**, 231-257.
- Cole, A.C. (1968). *Pogonomyrmex harvester ants: a study of the genus in North America*. University of Tennessee Press, Knoxville.
- Crist, T.O. & MacMahon, J.A. (1991). Foraging patterns of *Pogonomyrmex occidentalis* (Hymenoptera: Formicidae) in a shrubsteppe ecosystem: the roles of temperature, trunk trails, and seed resources. *Environmental Entomology* **20**, 265-275.
- Davidson, D.W. (1977). Foraging ecology and community organization in desert seed-eating ants. *Ecology* **58**, 725-737.
- De Vita, J. (1979). Mechanisms of interference and foraging among colonies of the harvester ant *Pogonomyrmex californicus* in the Mojave Desert. *Ecology* **60**, 729-737.
- Deneubourg, J.L., Aron, S., Goss, S., Pasteels, J.M. (1990). The self-organizing exploratory pattern of the Argentine ant. *Journal of Insect Behavior* **3**, 159-168.

- Edelman, A. (2010). Facilitative interactions between keystone rodents and harvester ants in the Chihuahuan Desert. In *Dispersal, facilitation, and burrow architecture in banner-tailed kangaroo rats*, PhD Dissertation, Department of Biology, University of New Mexico.
- Fewell, J. (1990). Directional fidelity as a foraging constraint in the western harvester ant, *Pogonomyrmex occidentalis*. *Oecologia* **82**, 45-51.
- Gordon, D.M. (1991). Behavioral flexibility and the foraging ecology of seed-eating ants. *American Naturalist* **138**, 379-411.
- Hill K., Kaplan, H., Hawkes, K. & Hurtado, A.M. (1987). Foraging decisions among Ache hunter-gatherers: new data and implications for optimal foraging models. *Ethology and Sociobiology* **8**, 1-36.
- Hölldobler, B. & Wilson, E. (1970). Recruitment trails in the harvester ant, *Pogonomyrmex badius*. *Psyche* **77**, 385-399.
- Hölldobler, B. & Wilson, E. (2008). *The superorganism: the beauty, elegance, and strangeness of insect societies*. WW Norton & Co Inc, New York.
- Hölldobler, B. (1976). Recruitment behavior, home range orientation and territoriality in harvester ants, *Pogonomyrmex*. *Behavioral Ecology and Sociobiology* **1**, 3-44.
- Hou, C., Kaspari, M., Vander Zanden, H. & Gillooly J (2010). Energetic basis of colonial living in social insects. *Proceedings of the National Academy of Sciences* **107**, 3634-3638.
- Johnson, R.A. (2000). Seed-harvester ants (Hymenoptera: Formicidae) of North America: an overview of ecology and biogeography. *Sociobiology* **36**, 89-122.

- Jun, J., Pepper, J.W., Savage, V.M., Gillooly, J.F. & Brown, J.H. (2003). Allometric scaling of ant foraging trail networks. *Evolutionary Ecology Research* **5**, 297-303.
- Moses, M.E. (2005). *Metabolic scaling, from insects to societies*. PhD Dissertation, Department of Biology, University of New Mexico.
- Mull, J.F. & MacMahon, J.A. (1997). Spatial variation in rates of seed removal by harvester ants (*Pogonomyrmex occidentalis*) in a shrub-steppe ecosystem. *American Midland Naturalist* **138**, 1-13.
- Reichman, O. (1984). Spatial and temporal variation of seed distributions in Sonoran Desert soils. *Journal of Biogeography* **11**, 1-11.
- Ryer, C. & Olla, B. (1995). Influences of food distribution on fish foraging behaviour. *Animal Behaviour* **49**, 411-418.
- Schafer, R.J., Holmes, S. & Gordon, D.M. (2006). Forager activation and food availability in harvester ants. *Animal Behaviour* **71**, 815-822.
- Shik, J.Z. (2008). Ant colony size and the scaling of reproductive effort. *Functional Ecology* **22**, 674-681.
- Stephens, D.W. & Krebs, J.R. (1986). *Foraging Theory*. Princeton University Press, Princeton.
- Stephens, D.W., Brown, J. & Ydenberg, R. (2007). *Foraging: Behavior and Ecology*. University of Chicago Press, Chicago.
- Sumpter, D.J.T. (2006). The principles of collective animal behaviour. *Philosophical Transactions of the Royal Society of London B* **361**, 5-22.
- Traniello, J.F.A. (1989). Foraging strategies of ants. *Annual Review of Entomology* **34**, 191-210.

Whitford, W.G. (1978). Foraging in Seed-Harvester Ants *Proconomyrmex* SPP. *Ecology* **59**, 185-189.

Whitford, W.G., Ettershank, G. (1975). Factors affecting foraging activity in Chihuahuan desert harvester ants. *Environmental Entomology* **4**, 689-696.

Wilson, E.O. & Osborne, E. (1971). *The Insect Societies*. Belknap Press, Cambridge.

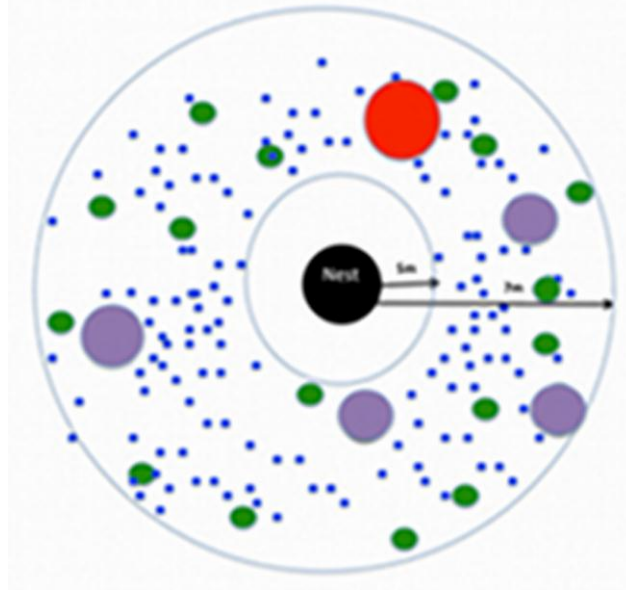


Figure 1. Experimental seed distribution around the nest entrance of a *P. rugosus* colony. Each colored circle is a pile of biat seeds dyed to that color. The size of each circle represents the relative number of seeds in that pile.

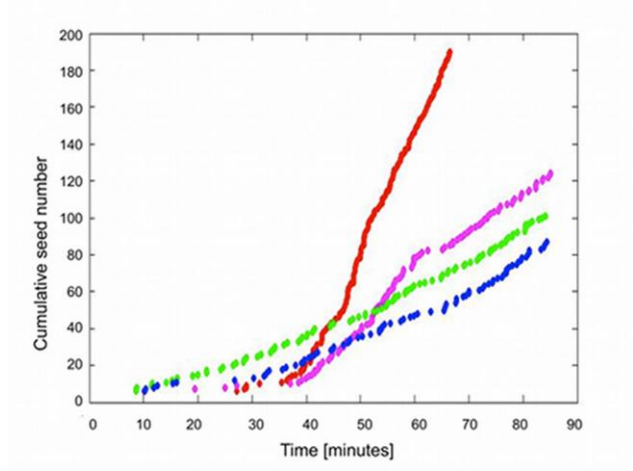


Figure 2. Seed intake curves from one field observation of a colony of *P. Rugosus*. The cumulative number of seeds collected during a 90-minute observation. Red = 1 pile of 256 seeds, purple = 4 piles of 64 seeds, green = 16 piles of 16 seeds, blue = 256 randomly scattered seeds.

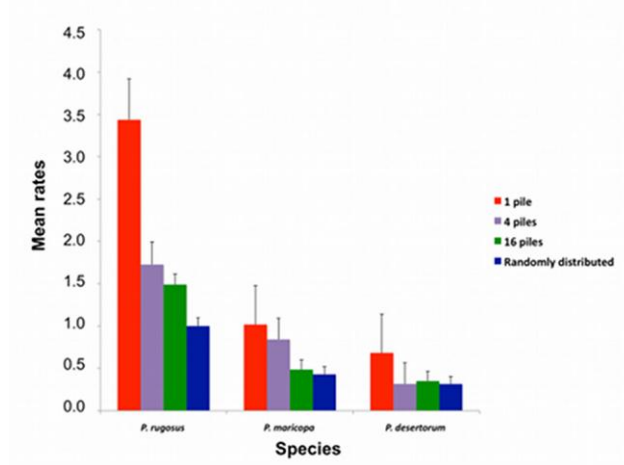


Figure 3. Mean foraging rate. Mean foraging rates of piled seeds. Rates are measured in seeds/minute per species and distribution. Error bars are standard errors.

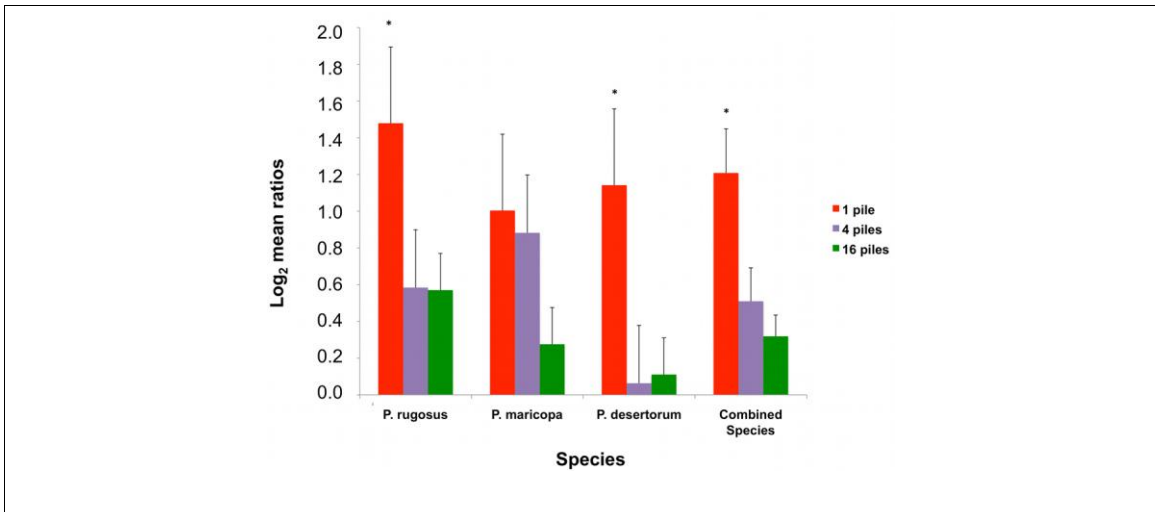


Figure 4. Normalized foraging rates. Bars indicate *normalized rates* (foraging rate of piled seeds divided by foraging rate of random seeds) for three distributions for three species depicted separately and combined. Normalized rates are log₂-transformed. Asterisks indicate significant differences of the single pile distribution rates with all other rates within the same species. Error bars are standard errors.

Table 1. Characteristic variables of each species

	Definition	Measurement Methods	Spp	N	Mean	SE	Lower Bound	Upper Bound
<i>Tf</i>	Total time of round trip [min]	Time they leave the nest to time of return with a seed	<i>P. desertorum</i>	15	7.25	2.73	1.84	12.67
			<i>P. maricopa</i>	16	17.96	2.85	12.29	23.62
			<i>P. rugosus</i>	32	13.56	2.24	9.11	18.01
<i>dt</i>	Distance from nest to seed	Measured distance between seed and nest	<i>P. desertorum</i>	15	3.86	1.04	1.8	5.92
			<i>P. maricopa</i>	16	8.18	1.09	6.02	10.34
			<i>P. rugosus</i>	32	7.83	0.85	6.14	9.53
<i>vt</i>	Travel velocity	Calculated as $vt = dt/\text{return time}$	<i>P. desertorum</i>	15	2.96	0.49	1.97	3.95
			<i>P. maricopa</i>	16	2.25	0.48	1.29	3.21
			<i>P. rugosus</i>	32	3.10	0.34	2.42	3.77
<i>R</i>	Rate of foragers returning	Measured at equilibrium (per minute)	<i>P. desertorum</i>	15	8.73	3.08	2.61	14.86
			<i>P. maricopa</i>	16	12.50	2.99	6.57	18.43
			<i>P. rugosus</i>	32	107.94	2.11	103.74	112.13
<i>F</i>	Number of active foragers	Foraging time * mean seed intake rate	<i>P. desertorum</i>	15	77.88	196.49	-312.37	468.13
			<i>P. maricopa</i>	16	208.52	190.25	-169.30	568.38
			<i>P. rugosus</i>	32	1712.6	134.53	1445.4	1979.8

Table 2. Experimental seed distributions

Species	Distance from nest	Distribution	Color	Numer of seeds in each pile
<i>P. desertorum</i>	2 - 4 m	1 pile	Red	32
		4 piles	Purple	8
		16 piles	Green	2
		Random	Blue	1
		Total		128
<i>P. maricopa</i>	2 - 4 m	1 pile	Red	32
		4 piles	Purple	8
		16 piles	Green	2
		Random	Blue	1
		Total		128
<i>P. rugosus</i> N=9	5 - 7 m	1 pile	Red	256
		4 piles	Purple	64
		16 piles	Green	16
		Random	Blue	1
		Total		1024

Table 3. Seed discovery times. Time in minutes from the start of the observation to the retrieval of the first seed for each bait distribution.

Species	Distribution	Discovery Times	
		Mean	SE
<i>P. desertorum</i> N=9	Natural	9.578	3.361
	1 pile	25.99	5.842
	4 piles	20.393	5.688
	16 piles	16.571	3.821
	Random	11.551	3.365
<i>P. maricopa</i> N=9	Natural	13.035	3.881
	1 pile	21.942	6.746
	4 piles	27.55	6.568
	16 piles	16.187	4.412
	Random	15.772	3.885
<i>P. rugosus</i> N=9	Natural	9.238	3.229
	1 pile	20.547	5.613
	4 piles	19.829	5.465
	16 piles	9.813	3.671
	Random	11.669	3.233
Combined species	Natural	10.617	2.022
	1 pile	22.827	3.514
	4 piles	22.591	3.422
	16 piles	14.190	2.299
	Random	12.998	2.024

Table 4. Seed collection rates for each distribution. Total collection time used to calculate the rates is measured from first seed to last seed of each distribution and from the time the observation starts to last seed of each distribution.

Species	Distribution	First Seed to Last Seed		Start-time to Last Seed	
		Mean	SE	Mean	SE
Measure: Seed collection rates [seeds/minute]					
<i>P. desertorum</i> N=9	Natural	0.610	1.292	0.596	0.341
	1 pile	0.683	0.485	0.304	0.179
	4 piles	0.316	0.266	0.168	0.229
	16 piles	0.350	0.123	0.204	0.118
	Random	0.314	0.095	0.234	0.132
<i>P. maricopa</i> N=9	Natural	3.286	1.218	3.448	0.331
	1 pile	1.018	0.457	0.677	0.174
	4 piles	0.842	0.251	0.369	0.223
	16 piles	0.485	0.116	0.356	0.115
	Random	0.429	0.090	0.319	0.128
<i>P. rugosus</i> N=9	Natural	3.696	1.218	3.460	0.331
	1 pile	3.436	0.457	2.450	0.174
	4 piles	1.726	0.251	1.605	0.223
	16 piles	1.490	0.116	1.353	0.115
	Random	1.005	0.090	0.867	0.128
Measure: Log-transformed normalized rates					
<i>P. desertorum</i> N=9	Natural	0.907	0.554	1.265	0.535
	1 pile	1.142	0.416	0.286	0.545
	4 piles	0.063	0.315	-0.471	0.387
	16 piles	0.111	0.200	-0.276	0.246
<i>P. maricopa</i> N=9	Natural	1.785	0.554	2.026	0.521
	1 pile	1.004	0.416	0.763	0.530
	4 piles	0.882	0.315	-0.058	0.376
	16 piles	0.276	0.200	0.258	0.239
<i>P. rugosus</i> N=9	Natural	1.735	0.554	1.828	0.521
	1 pile	1.479	0.416	1.209	0.530
	4 piles	0.584	0.315	0.607	0.376
	16 piles	0.570	0.200	0.665	0.239
Combined species N=27	Natural	1.476	0.320	1.706	0.303
	1 pile	1.208	0.240	0.753	0.309
	4 piles	0.510	0.182	0.026	0.219
	16 piles	0.319	0.115	0.215	0.139

CHAPTER 6
SIMULATING THE EVOLUTION OF RECRUITMENT
BEHAVIOR IN FORAGING ANTS

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SUMMARY

Spatial heterogeneity in the distribution of food is an important determinant of species' optimal foraging strategies, and of the dynamics of populations and communities. In order to explore the interaction of food heterogeneity and colony size in their effects on the behavior of foraging ant colonies, we built agent-based models of the foraging and recruitment behavior of harvester ants of the genus *Pogonomyrmex*. We optimized the behavior of these models using genetic algorithms over a variety of food distributions and number of foragers, and validated their behavior by comparison with data collected on harvester ants foraging for seeds in the field. We compared two models: one in which ants lay a pheromone trail each time they return to the nest with food; and another in which ants lay pheromone trails selectively, depending on the density of other food available in the area where food was found. We found that the density-dependent trail-laying model fit the field data better. We found that in this density-dependent recruitment model, colonies of all sizes evolved intense recruitment behavior, even when optimized for environments in which the majority of foods are distributed homogeneously. We discuss the implications of these models to the understanding of collective decision-making in eusocial insects, and of optimal foraging strategy and community dynamics

among ants.

INTRODUCTION

Spatial heterogeneity in the distribution of food is an important ecological determinant of the optimal foraging strategy of a species (Charnov 1976), and of the dynamics of populations and communities (Schoener 1974; Wiens 1976). More patchily distributed foods present foragers with an opportunity to take advantage of reduced search times within dense patches or clumps where food is more densely concentrated than in the environment as a whole. Foraging strategies that are best adapted to the scale and degree of heterogeneity in the environment are favored (Charnov 1976).

Heterogeneity influences the distribution of consumers (Wiens 1976) and can provide a dimension along which food niches are partitioned, allowing the coexistence of species (Schoener 1974).

Socially coordinated foraging behavior can be part of a species' strategy for exploiting patchily distributed foods (Wiens 1976). Social organisms in patchy environments can take advantage of others' knowledge of the location of food patches, either through active sharing of information or by observing others' foraging success (Ward and Zahavi 1973). Information sharing may be particularly important for eusocial insects. Because of the high relatedness among nestmates (Trivers and Hare 1967), and the fact that the reproductive success of an insect colony is mainly or entirely the result of the queen's reproductive output, workers are selected to maximize foraging success of the colony as a whole (Oster and Wilson 1979). Therefore, cooperative foraging may be

selected for among eusocial insects if it increases the foraging success of the colony, even if it comes with some cost to individual foragers.

Among many species of ants, information is communicated between foragers with the use of recruitment pheromones, which in some species are used to leave a trail along the ground as a forager makes a return trip to the nest with food (Wilson and Hölldobler 1990). When departing the nest, other foragers may follow these trails to sites where food has been found previously, and where, in environments where food is distributed heterogeneously, there may be more food available. In this way, foragers communicate information about the location of food sources, which allows reduced search times, and increased rate of food collection.

Harvester ants of the genus *Pogonomyrmex* provide an excellent model system to study the influence of heterogeneity in the distribution of food on the evolution of foraging strategies and social behavior. They feed primarily on small seeds that foragers carry individually, yet their food sources exist in a mixture of homogeneous sources and heterogeneous patches (Gordon 1993; Reichman 1984). Many seeds that harvester ants feed on are carried by the wind and are scattered dispersed at random; however depressions and other soil features may create pockets where seeds tend to fall out of the wind in greater concentrations, and some seeds may fall from plants in clumps or be harvested directly from plants' stems by the ants. Mature colonies among species in this genus range in size over more than an order of magnitude, from hundreds of foragers to more than ten thousand (Johnson 2000). The general foraging strategy of *Pogonomyrmex* varies from one species to the next, from solitary foraging to social foraging with persistent trunk trails (Johnson 2000), with a general trend toward more intense social

foraging with greater colony size (Beckers *et al.* 1989; Johnson 2000). Within a species, foragers may engage in solitary or social foraging depending on the density of available foods (Hölldobler 1976; Mull and MacMahon 1997).

In a study of the scaling of territory area with *Pogonomyrmex* forager number, Moses (2005) predicted a sub-linear scaling relationship between territory size and forager number. This results from the ants' need to strike an optimal trade-off between the cost of increased search times on a small territory, where ants distributed too densely deplete a small area of seeds and waste time searching where no more food is left to be found; and the cost of increased travel times to distant foraging patches on an increasingly large territory. However, while Moses (2005) found a sub-linear scaling relationship between territory area and forager number, and an increasing density of foragers on the territory with increasing forager number, she did not find a predicted increase in search time with forager number. This led to the hypothesis that, given some heterogeneity in the distribution of food sources, larger colonies, by virtue of having larger territories, have access to more information about this distribution, and can exploit this information to direct foraging effort to the densest, highest quality patches where foraging times are reduced.

In order to investigate the influence of forager number and heterogeneity in the distribution of food on foraging strategy, we developed agent-based models (ABMs) of foraging by harvester ant colonies, based on descriptions of their behavior in the biological literature and our own observations. We used these models to test the prediction that more intense recruitment behavior will be optimal for larger colonies and for colonies foraging in environments with more heterogeneity in the distribution of food.

ABMs simulate systems by iteratively executing rules that govern the behavior and interactions of agents within the system. On each iteration, the state of the system is updated based on the actions or interactions of the agents over each discrete time step. ABMs are particularly useful for modeling systems with spatial or temporal heterogeneity (Berec 2002; Nonaka and Holme 2007) and systems in which complex behavior emerges as the result of interactions among individual agents with relatively simple behaviors (Grimm *et al.* 2005), just as the group behavior of an ant colony emerges as the result of actions and interactions among the individual ants.

ABM's allow us to investigate the effects of forager number and food heterogeneity on the evolution of recruitment behavior by allowing perfect control over these independent variables, while controlling for other factors that are difficult or impossible to control in the field. For example, one wants to compare species that share enough similarity in their foraging habits and environment, and that vary only in the traits of interest; but it may be that traits covary among related species in the field as a result of phylogenetic dependence (Freckleton *et al.* 2002). Heterogeneity in the distribution of seeds in the field has been estimated by taking soil samples (e.g. Reichman 1984), however such estimates may not provide a relevant measure of the heterogeneity of foods for a given ant species, as ants do not collect all seeds indiscriminately. Even sympatric species may vary systematically in their preference for seeds of different sizes – and thus seeds that may be found in different spatial distributions – based on the size and morphology of the workers (Hölldobler 1976). ABM's allow us to control all confounding factors and experimental conditions, and thus provide a perfect experimental environment so that we can study only the behaviors of interest.

We are interested in the effects of forager number and food heterogeneity on the optimal foraging behavior of ant colonies, specifically colonies' use of pheromone recruitment to food sources. Therefore we optimized our ABMs using genetic algorithms (GAs), an optimization technique that simulates the process of evolution by natural selection (Forrest 1996; Mitchell 1998). GAs optimize functions or programs by repeatedly evaluating the success of a population of different possible parameter combinations, and recombining and mutating successful parameter sets to arrive at good solutions to the fitness problem over the course of generations. In the field, the behavior of *Pogonomyrmex* species is optimized by natural selection to maximize foraging success (an important component of reproductive success, among other goals and constraints) given each species' particular ecology. Therefore, GAs and other evolutionary algorithms are a particularly appealing method for selecting parameters for models of biological systems (e.g. Hamilton *et al.* 1990; Solé *et al.* 2000; Buchkremer and Reinhold 2008).

We used GAs to determine behaviors, encoded as parameters in our ABM, that maximized seed collection. The fitness function in our GA was total number of seeds collected in 10,000 ABM time steps. We executed GAs over a range of forager numbers, from colonies of ten to 1,0000 foragers, and over a range of food distributions, from fully homogeneous to fully heterogeneous, selecting for fastest rate of food collection under these varying conditions. We then used the resulting, optimized models to simulate experimental foraging observations, and compared the behavior of the resulting models to that of ants in the field.

METHODS

SIMPLE RECRUITMENT MODEL

We developed an ABM of recruitment by a colony of ants, and a GA to evolve parameters to maximize the rate of seed collection by the colony. Our model is similar in some respects to the search and recruitment behaviors in the models by Haefner and Crist (1994) and Crist and Haefner (1994); however we sought to simplify our model to reduce computational complexity and run-time, since optimizing our model for each combination of forager number and food heterogeneity requires running the model thousands of times.

Under this simple recruitment model, each time a searching ant picks up a piece of food, it lays a pheromone trail as it returns to the nest. Ants leaving the nest to begin another foraging trip follow pheromone trails to return to sites where food has previously been found. Ants that arrive in high-density food patches have lower search times on average than those in lower density patches, and reinforce the pheromone trails leading to these sites. Pheromones evaporate at an exponential decay rate, and over time the foragers are expected to converge on the highest quality patch based on its density and its distance from the nest, as more and more foragers are recruited to the highest quality sites (Beckers *et al.* 1990; Detrain & Deneubourg 2008).

At model initialization, all ants begin at the nest site located at the center of a grid of 4000 X 4000 cells. Each ant picks a direction at random and begins walking. At each time step, each ant stops walking with a constant probability determined by the parameter α , and begins to search. For high values of α , ants generally will walk short distances

from the nest; whereas for low values, ants will generally walk long distances from the nest before beginning to search.

Like *Pogonomyrmex* in the field (Crist and MacMahon 1991), searching ants move in a correlated random walk. The degree of turning in searching ants' correlated random walk allows them to search more thoroughly in a local area if turning more, or to move in a straighter line and cover more distance if turning less. We found that there may be different optimum degrees of turning at different times in an ants' search. At the end of a pheromone trail where there may be more food to be found, ants may randomly select the wrong direction to begin moving and walk away from a pile of food; however if they are able to turn more and therefore do a more thorough, local search when they begin searching (a behavior suggested by our personal observations of *Pogonomyrmex* foraging on piles of bait seeds in the field), they are more likely to find more food in a patch. If searching ants decrease their turning behavior over time, this allows ants to move off and search more widely for new food sources if they fail to find food early on.

We determined searching ants' turning behavior as follows. At each time step t , each searching ant selects a direction Θ_t to move from a normal distribution with mean equal to Θ_{t-1} and a standard deviation (SD) determined by three parameters, which allow degree of turning to change with the number of time steps since the ant began searching, t_s :

$$\text{SD} = \omega + \gamma / t_s^\delta \tag{1}$$

The parameter ω determines ants degree of turning. For lower values of ω , the direction searching ants move at each time step t is more tightly correlated with the direction the ant moved at time step $t-1$, and ants tend to turn less. For higher values, searching ants' movements are less correlated from one time step to the next, ants turn more and cover less distance. The parameter γ allows ants an extra degree of turning with low values of t_s , and to return to the baseline degree of turning ω as t_s becomes large, and therefore γ / t_s^δ becomes small; the exponent parameter δ determines how quickly this term approaches zero as t_s increases. Searching ants move at $\frac{1}{4}$ the speed of walking ants or trail-following ants, a relative rate supported by observations of ants in the field (Crist and MacMahon 1991).

Upon finding food, ants pick up the food and begin to return to the nest. *Pogonomyrmex* in the field navigate by landmarks and the polarization of sunlight, and have a keen ability to navigate even if displaced significant distances (Hölldobler 1976). Therefore, in our model, ants returning to the nest move at each time step so that their distance from the nest is non-increasing. They select an adjacent, in-bound cell to move to with probability proportional to the amount that a move to that cell would decrease the ant's distance to the nest. While returning to the nest, ants lay pheromone trails by incrementing the weight of pheromone on each cell they move through by a constant amount. Returning ants move at $\frac{1}{2}$ the speed of walking or trail-following ants (Crist and MacMahon 1991).

After returning from the first foraging trip, ants leaving the nest again follow trails if any exist, or if not (if no pheromone trails have been laid yet – see below), they begin searching at the nest entrance. Trail-following ants move at each time step so that their

distance from the nest is non-decreasing. They select an adjacent, out-bound cell with probability proportional to each cell's pheromone weight, as a fraction of the total pheromone weight on all such cells. This allows ants to bias their movement onto cells on which more pheromone has been laid, and gives ants a greater probability of arriving at sites from which more pheromone trails have recently been drawn. At each time step, trail-following ants abandon the pheromone trail and begin to search with probability determined by the parameter ε . When an ant arrives at a cell whose out-bound neighbors have no pheromone, it has reached the end of the trail, and it begins to search.

At each time step t , the weight of pheromone $\Pi_{x,y,t}$ on each cell x,y is evaporated at a rate determined by a colony-specific evaporation rate η :

$$\Pi_{x,y,t} = \Pi_{x,y,t-1} * (1 - \eta) \quad (2)$$

Over time this evaporation approximates an exponential decay rate. When the weight of pheromone on a cell falls below a threshold (pheromone weight 0.001), it is considered to have fallen below an ants' ability to perceive it, and the pheromone on that cell is set to zero.

See Table 1 for a summary of parameters used in this model.

DENSITY-DEPENDENT RECRUITMENT MODEL

During fieldwork, we observed that individual *Pogonomyrmex* foragers may sometimes travel back and forth to collect bait seeds without recruiting any other foragers to the effort (site fidelity, or patch fidelity; see Beverly *et al.* 2009; Buchkremer & Reinhold 2008; Crist & MacMahon 1991); and that this may go on indefinitely, or after some time

a number of other foragers may quickly join the effort and a foraging trail develops. Lenoir (2002) found that wood ants recruit more often to baits containing more fly larvae, and suggested that these ants may be able to measure the amount of available food in a patch; while Cerdá *et al* (2009) found that in gypsy ants, group recruitment is more common to baits with large numbers of seeds as compared to baits with small numbers of seeds. We therefore developed an alternate to the simple recruitment model above. This density-dependent recruitment model is identical to the simple recruitment model, except that instead of leaving a pheromone trail on the return trip to the nest each and every time an ant picks up food, ants make a decision to leave a pheromone trail or not. We introduced two new parameters to facilitate this.

The first parameter relevant to this decision is λ , which determines an ant's constant probability of leaving a pheromone trail each time it picks up a piece of food. Thus if λ is 1.0 or higher, the behavior of this model is identical to the simple recruitment model above; if λ is less than 0.0, the colony can abandon the use of pheromones entirely.

The second parameter μ determines ants' sensitivity to the presence of other food in neighboring cells in making the decision to lay a pheromone trail or not. Upon picking up a piece of food, an ant takes a count C of other seeds in the eight cells immediately adjacent to the cell where it found food, and decides to leave a pheromone trail on the return trip to the nest with probability p :

$$p = \lambda + C / \mu \tag{3}$$

(Note that if $p < 0$ then ants leave a trail with probability 0; if $p > 1$ then ants leave a trail with probability 1.)

The ability of ants in the field to detect other food in the neighborhood may be based on a scent of nearby seeds detectable by a foraging ant. Alternatively, Hölldobler (1976) noted that ants often handle a number of seeds before picking one up and returning to the nest; he speculated that ants may be sampling the availability of food in an area.

OPTIMIZATION BY GENETIC ALGORITHMS

As described above, the behavior of these models is subject to the selection of a variety of parameters. We are interested in differences in the behavior of ant colonies of different sizes after their behavior has been optimized by natural selection in environments with different degrees of heterogeneity in the distribution of food. Thus we are interested in the optimal behavior expressed by our models for simulated foraging environments.

An exhaustive exploration of the space of all combinations of parameters for the global optimum is not feasible. For the density-dependent recruitment model described above, we have eight floating-point parameters. If we assume a discretization of each parameter to two significant digits, and if all parameters were restricted to the interval $[0,1]$ (some are not so restricted), the search space of all possible parameters contains more than $(10^2)^8 = 10^{16}$ parameter combinations. Assuming the quarter-second run-times we experienced for our smallest and quickest simulations of ant colonies with ten foragers, an exhaustive exploration of the search space would take on the order of 10^6 years to complete. The search for optimal parameter sets for environments varying in

food heterogeneity is further complicated by the fact that the random placement of heterogeneous foods means that sometimes a colony might find a dense pile of food placed very near its nest, while at other times there may be no piles of food nearby. In order for a colony to behave optimally in such a stochastic environment, it must perform well overall given the possibility of all such eventualities, rather than being optimized to perform well in only one such configuration. Given the infeasibility of finding provable global optima for these models, we instead used genetic algorithms (GAs) to find parameters that approximate the optimal behavior possible for our models under each set of conditions.

Our GA works as follows. Each parameter is a floating point number. Parameters for each colony in the initial generation of each GA run were randomly selected from a uniform distribution. Each colony's genome is made up of one number for each parameter. The behavior of workers in our models is determined by a single set of parameters for the colony as a whole. Individual variation in the behavior of workers may be an interesting area of study; but given the high degree of relatedness among the workers in an ant colony (Trivers and Hare 1967), we expect that stochasticity in the behavior of ants modeled here will produce functionally similar variation for the purposes of this study.

Each GA run used a population of 100 colonies, over 100 generations. We ran GAs over a range of forager numbers, from 10 to 1000 foragers, and over a range of food heterogeneity. Food heterogeneity was manipulated by placing food in piles of 256 seeds, and scattering the remaining seeds, placed at random over a grid. We ran the GAs on a grid large enough so that after optimization ants of even the largest colonies

essentially never had contact with the edge, a grid of 4000x4000 cells. The grid was always set up with the same number of total seeds, but the number of seed piles ranged from zero (heterogeneity 0) to 100 (heterogeneity 1). We ran the GA four times for each combination of forager number and food heterogeneity.

All colonies were evaluated on eight food configurations per generation, with piles and seeds placed at random on the grid, but each with the particular degree of heterogeneity for that GA run. The eight food configurations were standard for each generation, such that each colony was evaluated on the eight configurations identical to those given to all other colonies in that generation.

Each colony was evaluated on each food configuration for 20,000 time steps per configuration. Because the energetic cost of foraging is a tiny fraction of the energetic value of a seed retrieved by *Pogonomyrmex* foragers (Fewell 1988, Weier and Feener, 1995), time costs dominate in selection on foraging efficiency; therefore the measure of fitness we used was the total number of seeds collected by each colony in the eight food configurations in each generation. An equal value was assigned to seeds collected from any of the distributions of food, whether piled or randomly scattered.

In each generation, tournament selection for the greatest number of seeds collected determined the parents for the next generation. Tournament selection is an efficient selection method whose selection pressure is robust to noisy fitness evaluation methods (Miller and Goldberg 1996), such as the sampling of colonies' performance on stochastically determined food distributions used here. Two colonies (parameter sets) were selected at random from the population, and their fitness (the number of seeds they had collected) were compared. The one with greater fitness was selected as a parent.

Another two colonies from the remaining 99 were then selected at random, and the one with the greater fitness from this pair was selected as a second parent. These two parental genomes were recombined with a crossover rate of 10%. Inherited parameters were mutated with probability 0.05, by selecting from a normal distribution with mean equal to the current parameter value, and standard deviation equal to the current parameter value * 0.05. Both parental parameter sets were then returned to the pool of potential parents, and this was repeated 100 times to produce the next generation of parameter sets.

COMPARISON TO FIELD DATA

We attempted to validate our models by comparison with observations of *Pogonomyrmex* foraging in the field. In a separate field study, we (Flanagan *et al.* in review) studied the foraging behavior of three *Pogonomyrmex* species that range over more than an order of magnitude in maximum colony size: *P. desertorum*, up to 500 workers; *P. maricopa*, up to 1000 workers; and *P. rugosus*, up to 10000 workers. We baited focal colonies with dyed, color-coded seeds arrayed around the nest in four different distributions. We baited with a large single pile of seeds; an equal number of seeds divided into four piles at one-quarter the density, each pile over the same area as the single large pile; seeds divided into 16 piles at one-sixteenth the density; and seeds scattered randomly. Seed baits were placed within a minimum and maximum radius, forming a donut around the nest entrance. We then observed the focal colony as it foraged, and recorded the retrieval of seeds from each of the four baits to the nest.

We simulated foraging observations using models parameterized by our GAs. We initialized models with food distributions that mimic the experimental baits we used in the field (Flanagan *et al.* in review; see Fig. 1). Based on the assumption that

Pogonomyrmex in the field have evolved to exploit a mixture of heterogeneous and homogeneous food sources, we selected parameter sets for our models that were optimized for 50% piled and 50% random food distributions (heterogeneity 0.5 in Figs. 3 and 4). We compared the behavior of models of our largest colonies of 1,000 ants, with the behavior of *P. rugosus*, a species whose colonies can grow as large as 10,000 workers, but many or most of which have fewer workers, and only a fraction of which workers forage (as opposed to engaging in other tasks or remaining idle) at any time (Moses 2005).

Following the procedure we used with our field data (Flanagan *et al.* in review), we produced cumulative intake curves from these observations, and from these calculated mean rates of seed collection from each seed distribution. We normalized the rates of collection from piled distributions by producing a ratio of the rate of collection from each piled distribution to the rate of collection from the random distribution in each observation. This allowed us to produce a measure of the effect of heterogeneity on seed collection rate that is comparable to observations of ants in the field. We analyzed these ratios using repeated measures ANOVA, a method that takes into account the non-independence between the rate of collection of food from each distribution: within a single observation, an ant retrieving a seed from one distribution is not at the same time available to collect seeds from other distributions, and therefore the rate of collection of piled foods is not independent of the rate of collection of randomly scattered foods. For ant colonies in the field, the number of active foragers may vary from colony to colony or from day to day, producing variation in the rates of collection of all foods. Repeated

measures ANOVA accounts for this dependence, and gives greater statistical power with these kinds of data.

RESULTS

GA RESULTS

We found that variation in foraging success (fitness for the purposes of our GA) from one generation to the next was greatest in the smallest colonies of ten ants, in environments where all available food is in piles. This results from a small territory size (an emergent property of parameters selected by the GA) and the chance placement of piles of food relatively close to or far from the nest. The territories of larger colonies are more likely to encompass multiple piles, and therefore these colonies experience less variation in foraging success from one generation to the next. The foraging success of colonies foraging on distributions with greater proportions of randomly scattered foods is less subject to the chance placement of dense piles of food.

Nevertheless, because we evaluated each colony on eight food configurations in each generation, we achieved sufficient stability in fitness from one generation to the next to observe optimization on the foraging task over the course of the GA runs. Fig. 2a illustrates the mean and maximum fitness (total number of seeds collected over eight evaluations foraging for 20,000 time steps) in each generation over one GA run, with a ten-ant colony foraging on a fully heterogeneous food distribution. It was with this combination of small forager number and high degree of heterogeneity in the food distribution that we saw the greatest variation about the mean in number of seeds collected from one generation to the next. For contrast, Fig. 2b illustrates the mean and

maximum fitness during one GA run for a 1000-ant colony foraging on a fully heterogeneous food distribution, where foraging success from one generation to the next is reduced. We tested this difference in variance for the ten- and 1000-ant GA runs illustrated in Fig. 2, selecting generations 50 and greater, after all or most optimization was complete. After normalizing mean fitness for each colony to 1, we found that the variance in foraging success was significantly greater for the ten-ant colony (F-test for equality of two variances: ten-ant variance = 0.053, 1000-ant variance = 0.009; $F = 6.136$, $df = 49,49$, $p < 0.001$).

We are interested in the degree to which forager number and heterogeneity in the distribution of food select for the use of pheromone trails to direct the foraging activity of the colony. Two parameters are indicative of the intensity of recruitment behavior: with decreasing rate of pheromone evaporation η , pheromone trails are less ephemeral, and it is possible for ants to follow trails longer distances from the nest; and with decreasing probability that a trail-following ant will abandon a pheromone trail at each time step, ϵ , ants are more likely to follow pheromone trails to their end. To some extent, these parameters may be traded off against one another as GAs converge on an optimal degree of trail-following behavior. Below, we use as a measure of the intensity of recruitment a derived Recruitment Factor, which is the geometric mean of trail persistence $(1 - \eta)$ and ants' trail fidelity $(1 - \epsilon)$:

$$\text{Recruitment Factor} = \sqrt{(1 - \eta)(1 - \epsilon)} \quad (4)$$

Thus, ants of a colony with Recruitment Factor equal to 1 could theoretically follow pheromone trails an infinite distance, as trails would be permanent, and ants following trails would unfailingly follow them to their end. Ants of a colony with Recruitment Factor equal to 0 would be unable to follow pheromone trails any distance, because either pheromone trails would evaporate instantly, or because ants would have no tendency to follow pheromone trails leading from the nest.

(a) *Simple Recruitment Model*

In the simple recruitment model, we found significant, positive, main effects of both food heterogeneity and forager number on recruitment factor (GLM, standardized β coefficients throughout. Heterogeneity: $\beta = 0.624$, $p < 0.001$; Forager Number: $\beta = 0.373$, $p < 0.001$. $N = 133$ GA runs.) and a significant, negative interaction effect of forager number with food heterogeneity (GLM: $\beta = -.295$, $p < 0.001$). Recruitment behavior increased with forager number, and in environments with more heterogeneously distributed foods. The relationship of recruitment behavior with forager number and food heterogeneity is illustrated in Fig. 3a.

(b) *Density-Dependent Recruitment Model*

In the density-dependent recruitment model, similar to the results of the simple recruitment model, we found significant, positive, main effects of both food heterogeneity and forager number on recruitment (GLM: Heterogeneity: $\beta = 0.507$, $p < 0.001$; Forager Number: $\beta = 0.166$, $p = 0.009$. $N = 137$ GA runs.) and a significant, negative interaction effect of forager number with food heterogeneity (GLM: $\beta = -.211$, $p = 0.003$).

Relative to the simple recruitment model (Fig. 3a), the intensity of recruitment behavior in the density-dependent recruitment model is more robust to decreasing heterogeneity in the distribution of food (Fig. 3b). Recruitment behavior remained relatively high even in the smallest colonies with as little as one quarter of the food distributed in dense piles; whereas in the simple recruitment model, recruitment behavior declined steadily with decreases in food heterogeneity below heterogeneity 1. This is reflected in the relatively weak effect of forager number on recruitment behavior in the density-dependent recruitment model ($\beta = 0.166$, 95% CI 0.042 to 0.290; cf. $\beta = 0.373$, 95% CI 0.229 to 0.517 in the simple recruitment model).

In order to understand why and when ants use pheromone trails, we investigated the relationship between forager number, food heterogeneity, and the tendency of ants to leave pheromone trails on their return trip to the nest. The parameter λ , which describes ants' baseline probability of leaving a pheromone trail on the return trip with food, provides the clearest indicator of a colony's use of pheromone trails. We found no effect of forager number on λ (GLM: $p > 0.10$), but a significant quadratic effect of food heterogeneity (GLM: heterogeneity $\beta = -1.138$, $p < 0.001$; heterogeneity² $\beta = 0.947$, $p < 0.001$. $N = 137$.) These relationships are illustrated in Fig. 4.

Thus, colonies optimized on mixed distributions of food were most selective in their trail-laying behavior. We found that colonies evolved the most liberal trail-laying behavior when optimized on fully homogeneous food distributions; and that colonies evolved significantly more selective trail-laying behavior on fully heterogeneous distributions (two-sample t-test: heterogeneous mean $\lambda = -0.272$, SD = -0.674; homogeneous mean $\lambda = 0.448$, SD = -0.464. $p < 0.001$. $N = 53$ GA runs.) Therefore ants

are significantly more likely to leave a pheromone trail each time they pick up food from a fully homogeneous food distribution than ants foraging on fully heterogeneous distributions. We believe this counter-intuitive result indicates selection for patch-switching behavior in heterogeneous environments, but is in part an artifact of our model. This is a subject we will return to in Discussion below.

COMPARISON TO FIELD DATA

Fig. 5 illustrates the cumulative collection of bait seeds placed in four distributions: one large pile of 256 seeds, represented by the red cumulative curve; four piles of 64 seeds each, represented by the purple curve; 16 piles of 16 seeds each, represented by the green curve; and 256 randomly scattered seeds, represented by the blue curve. Fig. 5a represents a typical field observation of *P. rugosus* foraging over an hour period. Fig. 5b represents a simulated observation of foraging by the simple recruitment model. Fig. 5c represents a simulated observation of foraging by the density-dependent recruitment model.

Qualitatively, the density-dependent model (Fig. 5c) provides a better match to the field data than the simple recruitment model. The field observation shows slow initial discovery of the single-pile and four-pile distributions, but the rate of collection of seeds from these distributions increases over the course of the observation. On the other hand, the simple recruitment model (Fig. 5b) produces a less satisfying match to the field observation: it is always the more homogeneous distributions that are collected more rapidly, and the rate of collection of each distribution falls off over time as the remaining seeds become fewer and harder to find. Because we found better fit of the density-

dependent recruitment model to the field data, and because the density-dependent recruitment models had the capacity to evolve behavior identical to the simple recruitment models if that behavior were favored, we will focus the quantitative analysis below on the behavior of the density-dependent recruitment model.

We compared mean rate of collection of seeds from heterogeneous distributions relative to that of homogeneously distributed seeds, by ants in the field and by our models. As in Flanagan *et al.* (In review), we analyzed the results of our simulated foraging observations using repeated measures ANOVA. In our field work, we (Flanagan *et al.* in review) found a significant effect of source seed distribution on the ratio of seeds collected from piled distributions relative to random seeds, but we found no effect of forager number. Similarly, our analysis (Repeated measures ANOVA. $N = 27$ simulated foraging observations) of the behavior of the density-dependent recruitment model found a significant within-subjects effect of seed distribution ($p < 0.001$), but no between-subjects effect of colony size ($p > .10$), nor a within-subjects distribution X colony size interaction ($p > .10$). These relative rates are illustrated for the field data collected by Flanagan *et al.* (In review) (Fig. 6a) and simulated foraging by the density-dependent recruitment model (Fig. 6b). Values for these ratios are \log_2 -transformed, so that a value of zero indicates that seeds were collected at the same rate as randomly distributed seeds, while a value of one indicates that seeds were collected twice as fast.

We analyzed field and simulation data together, including both species and data source as factors (Repeated measures ANOVA. $N = 54$, 27 field and 27 simulated foraging observations.) We found that ratios for the density-dependent recruitment model are generally lower than those for ants in the field (Between-subjects effect of data

source: $p = 0.017$), perhaps indicating that ants in the field have a keener ability to exploit piled foods using additional behaviors to those we have modeled here, e.g. the ability to smell or otherwise sense and move to nearby seeds when searching; or using individual memory to return to a site where food was previously found (site fidelity), which behavior may be of value in exploiting patchily distributed foods even for ant species that use other methods to share social information (Grüter *et al.* 2010). However the relative treatment of piles of different sizes follows much the same pattern in the model and in the field (Within-subjects effects of seed distribution X species and seed distribution X data source, both $p > 0.10$; Fig. 6).

DISCUSSION

We found a satisfying fit between the behavior of our density-dependent recruitment model and the foraging behavior of *Pogonomyrmex* harvester ants in the field. The simple recruitment model we developed based on common models of ant recruitment in the literature (Detrain and Deneubourg 2008) produced a less satisfying fit, as a result of its inability to converge foraging effort on high-quality patches in environments where seeds exist in a mixture of homogeneous and heterogeneous sources. We found that when optimized by GA, the density-dependent recruitment model tended to evolve relatively selective trail-laying behavior, instead of the behavior of the simple recruitment model. This allowed the model to decrease the noise in the pheromone system that resulted from trails leading to low quality patches in the simple recruitment model, and allowed colonies to adaptively converge their foraging effort on high quality patches given any degree of heterogeneity in the distribution of food. The density-dependent

recruitment model allowed the evolution of intense recruiting behavior in colonies of all sizes, and in all environments except those completely devoid of heterogeneous food sources. Density-dependent recruitment behavior allows ants to exploit heterogeneity in the distribution of food when they encounter it, even if they encounter it only rarely.

Contrary to the expectation of the simple recruitment model, in which ants leave pheromone trails each and every time they pick up food, we found that for all forager numbers and for all degrees of heterogeneity in the distribution of food, the density-dependent recruitment model evolved mean values for λ less than 1; colonies always evolved a condition-dependent trail-laying behavior. The lowest values of λ tended to evolve in environments with mixed heterogeneous and homogeneous food sources, where there is greatest advantage in basing the decision to leave a pheromone trail on the presence of other nearby foods, in order to distinguish randomly scattered foods from piled foods. We found a robust effect of increasing food heterogeneity on the evolution of increasing recruitment behavior in the simple recruitment model. In the density-dependent recruitment models, recruitment behavior remained high until essentially all heterogeneity was removed from the food distribution.

Although the results of our GA runs with the simple recruitment model revealed the hypothesized positive effect of forager number on recruitment behavior, this result was supported less strongly in our density-dependent recruitment model. Like the simple recruitment model, the density-dependent recruitment model evolved increasing recruitment behavior with increasing forager number in fully homogeneous food distributions. It is in these completely homogeneous environments, however, where we least expect to see the evolution of recruitment behavior. Therefore, this result indicates

colonies' use of the pheromone trails to direct foraging effort an optimal distance away from the nest, rather than toward a particular food source. This is similar to the use of foragers' use of trunk trails to travel some distance from the nest before beginning to search. It is likely, however, that individual ants have the capacity to walk a distance from the nest entrance before beginning to search, without relying on pheromone trails to do so. While it may be that ants will often drop off a pheromone trail before reaching its end in order to explore for additional, nearby food sources, we think it unlikely that pheromone trails would be used solely for directing foragers away from the nest, given that, for *Pogonomyrmex* and other ant taxa that produce trail pheromone with a specialized gland (Hölldobler *et al.* 2004), producing pheromone presumably has some physiological cost.

In our models, we forced ants to follow pheromone trails from the nest, or else to begin searching immediately at the nest entrance, in order to force optimization on the use of the pheromone trails. The evolution of increased recruitment behavior with forager number in environments with completely homogeneously distributed foods may be an artifact of this aspect of our model. There may be other reasons that ants will use trunk trails to travel a distance from the nest before beginning to forage, e.g. avoidance of predators or management of conflict with neighboring nests; but we suspect it is unlikely that ants in nature use pheromone trails solely for the purpose of directing foraging effort away from the nest.

With the introduction of any piled foods to the environment, we found that the density-dependent recruitment model evolved relatively intense recruitment behavior even in the smallest colonies. Given some degree of heterogeneity in the environment,

colonies that can exploit this heterogeneity when they encounter it are at a selective advantage over those that do not, even in species with small colonies that encounter piled foods relatively rarely. The density-dependent recruitment behavior allows colonies to exploit heterogeneity when and where they find it, even if they encounter piles of food infrequently, as for the small colonies modeled here (note the great variance in foraging success for the ten-ant colony in Fig. 2a).

We observed that the behavior of the simple recruitment model was similar to the behavior of the density-dependent recruitment model when evolved on fully heterogeneous food distributions. This is because in these environments, all available food is found in dense patches. Therefore if an ant finds a piece of food, that piece of food is necessarily coming from a dense patch and information about that location is of as much value as for that of food found anywhere else. In these environments both models tend to produce well-defined pheromone trails and converge their foraging effort on nearby piles, as predicted by common models of ant recruitment and collective decision-making (Detrain and Deneubourg 2008). In addition, we found that the density-dependent recruitment model evolved significantly more selective use of the pheromones on fully heterogeneous food distributions than in fully homogeneous distributions. More selective trail-laying behavior allows ants to be sensitive to the depletion of dense piles, and allows the colony to more rapidly and adaptively switch to a new pile as the remaining seeds become fewer and harder to find. Wilson (1961) described the way in which the number of *Solenopsis* workers at a food source may be regulated by unsuccessful foragers returning to the nest without laying a trail. Similarly, Mailleux *et al.* (2005) found that *Lasius* workers require a threshold volume of nectar in their crops in

order to lay a trail. This negative feedback has a lag of several minutes, however, resulting in an “overshoot” of the optimal number of workers arriving at a site (Wilson 1961). On the other hand, if even successful foragers are able to return from a dwindling food source without laying a trail, as we observed here, this “overshoot” may be minimized. Thus, even for species whose foods occur only in patches too large to be collected by a single forager, e.g. army ants specializing on raiding other social insect colonies (Franks *et al.* 1991; Solé *et al.* 2000) it may be adaptive to make relatively selective use of pheromone trails. Given that producing pheromone may bear some physiological cost – a cost we did not impose on the simulated colonies in our GA runs – we expect that for ants in the field, there is even greater advantage in using pheromone trails selectively.

When the simple recruitment model was evolved on increasingly homogeneous food distributions, recruitment behavior steadily fell off. We found that the addition of homogeneously distributed foods prevented the convergence of foraging effort on high quality patches. This was a surprising outcome, given the assumption that colonies will converge on high quality patches because of the increased ease of finding food in those patches (Detrain and Deneubourg 2008). We observed that pheromone trails being drawn back to the nest by ants that picked up homogeneously distributed foods created so much noise in the system that the colonies were unable to converge (see Fig. 1a); many ants that set out from the nest followed trails that led back to a site where no food was to be found, and therefore did not arrive in the high quality patches.

Instead of evolving less intense recruitment behavior with an increasing proportion of homogeneously distributed foods in the environment, the density-dependent

recruitment models evolved increasingly selective use of the pheromones (see Fig. 1b). By becoming increasingly selective about drawing a pheromone trail on the return trip to the nest, the information value of the pheromone trails remained high enough that colonies continued to recruit heavily even when as little as one quarter of the available food was distributed in piles.

The binary decision making process surrounding the laying of a pheromone trail is analogous to the decision-making process by scouts of *Temnothorax albipennis* in nest-site selection (Dornhaus *et al.* 2004; Visscher 2007). Scouts of these species evaluate potential nest sites and selectively recruit nestmates to preferred sites by tandem running, and a variety of nest-site properties relevant to this decision have been identified (Visscher 2007). Presumably each desired property is factored into a decision-making process such that each contributes to the probability that a scout will begin recruiting to the site. Similarly, *Pogonomyrmex* foragers may incorporate a variety of additional factors into the decision to lay a pheromone trail, including, for example, the presence of other ants which may compete for a food source if it is not collected quickly. Over time, other foragers making the same evaluation cause reinforcement of paths to high value patches and convergence on the optimal colony-level behavior, without distraction by the noise of paths leading to sites of little or no value. This model of recruitment differs from that described by Beckers *et al.* (1993) for the black garden ant *Lasius niger*, in which workers modulate the weight of pheromone trails according to the concentration of sugar solutions they discover. Compared to *Lasius niger* which forages for nectars that may vary substantially in their concentration and value, there may be less variation in the quality and value of seeds returned to the nest by *Pogonomyrmex* foragers. Harvester

ants select seeds within a range of sizes that are easy enough to handle given forager size and morphology of a species (Hölldobler 1976); within this range there may be less variation in nutrient value than that encountered by *Lasius niger* foraging on nectar. Therefore for *Pogonomyrmex* foragers, the qualities of individual seeds discovered may be less important to the colonies' foraging success than the presence of other nearby seeds, and this may be an important determinant of the optimal recruitment behavior in these taxa.

Johnson (2000) categorizes the foraging behavior of *Pogonomyrmex* species, ranging from solitary foraging to recruitment using persistent trunk trails. Here we have explored via simulation the benefits of social foraging via pheromone recruitment across a range of colony sizes. Our results and those of Flanagan *et al.* (In review) suggest that the behavioral components of recruitment may not differ categorically across species. Rather, the tendency for harvester ant species to engage primarily in solitary foraging vs. trunk trail recruitment may depend on the likelihood from one day to the next that a small vs. a large colony finds a high-density patch of seeds somewhere on its territory.

We wondered if differences in the ability to recruit to high quality food patches might cause the niche partitioning that allows the co-occurrence of the three sympatric *Pogonomyrmex* species examined in Flanagan *et al.* (In review). Our results suggest such a differential ability to recruit is not the answer to that question. Species with larger colony size may dominate high quality patches, however, by recruiting large numbers of foragers to these sites and overwhelming and excluding smaller colonies foraging there, while these smaller colonies are then forced to forage on randomly or less densely distributed foods. This suggests further research into interspecific competitive

interactions may be fruitful in understanding how heterogeneity in the distribution of food causes niche partitioning among these ant species.

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REFERENCES

- Beckers R., Goss S., Deneubourg J.L. & Pasteels J.M. (1989). Colony size, communication, and ant foraging strategy. *Psyche: A Journal of Entomology* **96**:239-256.
- Beckers R., Goss S., Deneubourg J.L. & Pasteels, J.M. (1990). Collective decision making through food recruitment. *Insectes Sociaux* **37**: 258-267.
- Beckers R., Goss S. & Deneubourg J.L. (1993). Modulation of trail laying in the ant *Lasius niger* (Hymenoptera: Formicidae) and its role in the collective selection of a food source. *Journal of Insect Behavior* **6**: 751-759.

- Beverly B.D., McLendon H., Nacu S., Holmes S. & Gordon, D.M. (2009). How site fidelity leads to individual differences in the foraging activity of harvester ants. *Behavioral Ecology* **20**: 633-638.
- Bonabeau E., Dorigo M. & Theraulaz G. (2000). Inspiration for optimization from social insect behavior. *Nature* **406**: 39-42.
- Buchkremer E.M. & Reinhold K. (2008). Sector fidelity—an advantageous foraging behavior resulting from a heuristic search strategy. *Behavioral Ecology* **19**: 984-989.
- Cerdá, X., Angulo, E. & Boulay R. (2009). Individual and collective foraging decisions: a field study of worker recruitment in the gypsy ant *Aphaenogaster senilis*. *Behavioral Ecology and Sociobiology* **63**: 551-562.
- Charnov, E.L. (1976). Optimal foraging: the marginal value theorem. *Theoretical Population Biology* **9**:129-136.
- Crist, T.O. & McMahon, J.A. (1991). Individual foraging components of harvester ants: movement patterns and seed patch fidelity. *Insectes Sociaux* **38**:379-396.
- Crist, T.O. & Haefner, J.W. (1994). Spatial model of movement and foraging in harvester ants (*Pogonomyrmex*) (II): The roles of environment and seed dispersion. *Journal of Theoretical Biology* **166**: 315-323.
- Detrain C. & Deneubourg, J.L. (2008). Collective decision-making and foraging patterns in ants and honeybees. *Advances in Insect Physiology* **35**: 123-173.
- Dornhaus, A., Franks, N.R., Hawkins, R.M. & Shere H.N.S. (2004). Ants move to improve: colonies of *Leptothorax albipennis* emigrate whenever they find a superior nest site. *Animal Behavior* **67**: 959-963.

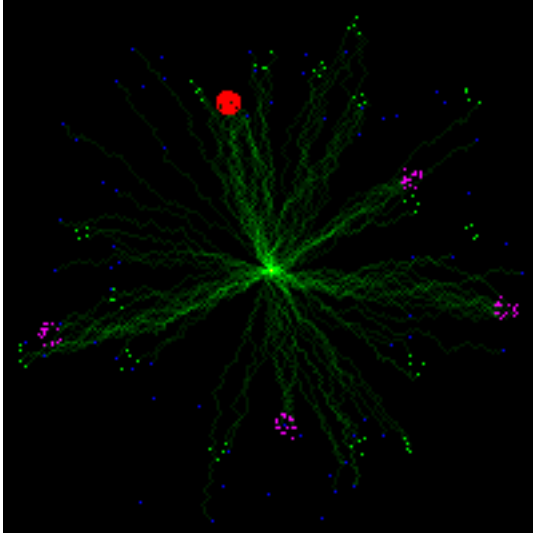
- Fewell, J.H. (1988). Energetic and time costs of foraging in harvester ants, *Pogonomyrmex occidentalis*. *Behavioral Ecology and Sociobiology* **22**: 401-408.
- Flanagan, T.P., Letendre, K., Burnside, W.R., Fricke, M. & Moses, M.E. (In review). Quantifying the effect of seed dispersion and colony size on foraging rates in three species of desert seed harvesting ants.
- Forrest, S. (1996) Genetic algorithms. *ACM Conference on Computer and Communications Security* **28**: 77-80.
- Franks, N.R., Gomez, N., Goss, S. & Deneubourg, J.L. (1991). The blind leading the blind in army ant raid patterns: Testing a model of self-organization (Hymenoptera: Formicidae). *Journal of Insect Behavior* **4**: 583-607.
- Grüter, C., Czaczkes, T.J. & Ratnieks, F.L.W. (2010). Decision-making in ant foragers (*Lasius niger*) facing conflicting private and social information. *Behavioral Ecology and Sociobiology* **65**: 141-148.
- Haefner, J.W. & Crist, T.O. (1994). Spatial model of movement and foraging in harvester ants (*Pogonomyrmex*) (I): The roles of memory and communication. *Journal Theoretical Biology* **166**: 299-313.
- Hölldobler, B., Morgan, E.D., Oldham, N.J., Liebig, J. & Liu, Y. (2004). Dufour gland secretion in the harvester ant genus *Pogonomyrmex*. *Chemoecology* **14**: 101-106.
- Gordon, D.M. (1993). The spatial scale of seed collection by harvester ants. *Oecologia* **95**: 479-487.
- Hamilton, W.D., Axelrod, R. & Tanese, R. (1990). Sexual reproduction as an adaptation to resist parasites (A review). *Proceedings of the National Academy of Sciences* **87**: 3566-3573.

- Hölldobler, B. (1976). Recruitment behavior, home range orientation and territoriality in harvester ants, *Pogonomyrmex*. *Behavioral Ecology and Sociobiology* **1**: 3-44.
- Johnson, R.A. (2000). Seed-harvester ants (Hymenoptera formicidae) of North America: An overview of ecology and biogeography. *Sociobiology* **36**: 83-122.
- Lenoir, L. (2002). Can wood ants distinguish between good and bad food patches on the forest floor? *European Journal of Soil Biology* **38**: 97-102.
- Mailleux, A.C., Detrain, C. & Deneubourg, J.L. (2005). Triggering and persistence of trail-laying in foragers of the ant *Lasius niger*. *Journal of Insect Physiology* **51**: 297-304.
- Miller, B.L., Goldberg, D.E. (1995). Genetic algorithms, tournament selection, and the effects of noise. *Complex Systems* **9**: 193-212.
- Mitchell, M. (1998). *An Introduction to Genetic Algorithms*. MIT Press, Cambridge.
- Moses, M.E. (2005). *Metabolic Scaling in Individuals and Societies*. Dissertation, University of New Mexico.
- Mull, J.F. & MacMahon, J.A. (1997). Spatial variation in rates of seed removal by harvester ants (*Pogonomyrmex occidentalis*) in a shrub-steppe ecosystem. *American Midland Naturalist* **138**: 1-13.
- Oster, G. & Wilson, E.O. (1979) *Caste and Ecology in the Social Insects*. Princeton University Press, Princeton.
- Reichmann, O.J. (1984). Spatial and temporal variation of seed distributions in Sonoran Desert soils. *Journal of Biogeography* **11**: 1-11.

- Ruxton, G.D. & Beauchamp, G. (2008). The application of genetic algorithms in behavioural ecology, illustrated with a model of anti-predator vigilance. *Journal of Theoretical Biology* **250**: 435-448.
- Schoener, T.W. (1974). Resource partitioning in ecological communities. *Science* **185**: 27-39.
- Solé, R.V., Bonabeau, E., Delgado, J., Fernández, P. & Marín J. (2000). Pattern formation and optimization in army ant raids. *Artificial Life* **6**:219-226.
- Trivers, R.L. & Hare, H. (1967) Haplodiploidy and the evolution of the social insects. *Science* **191**: 249-263.
- Visscher, P.K. (2007). Group decision making in nest-site selection among social insects. *Annual Review of Entomology* **52**: 255-75.
- Ward, P. & Zahavi, A. (1973). The importance of certain assemblages of birds as “information-centres” for food-finding. *Ibis* **115**: 517-534.
- Weier, J.A. & Feener, D.H. (1995). Foraging in the seed-harvesting ant genus *Pogonomyrmex*: are energy costs important? *Behavioral Ecology and Sociobiology* **36**: 291-300.
- Wiens, J.A. (1976). Population responses to patchy environments. *Annual Reiview of Ecology and Systematics* **7**: 81-120.
- Wilson, E.O. (1962). Chemical communication among workers of the fire ant *Solenopsis saevissima* (Fr. Smith) 1. The organization of mass foraging. *Animal Behavior* **10**: 134-147.
- Wilson, E.O., Hölldobler, B. (1991) *The Ants*. Belknap Press, Cambridge.

Table 1: Summary of parameters that influence the behavior of the models, and which are selected by GAs	
Parameter	Function
α	At model initialization, determines the probability each time step that an ant walking from the nest will stop walking and begin to search. For lower values, ants tend to walk farther from nest before beginning to search.
ω	For searching ants moving in a correlated random walk, determines the baseline degree of deviation in the direction an ant will move from one time step to the next. For low values, ants turn less, move in a straighter line, and cover more distance; for high values, ants movements are more random, they turn more, search more thoroughly in a local area, but cover less distance.
γ	For searching ants, determines the additional degree of deviation in turning early on in an ant's search, allows for more thorough, local searching at the end of a pheromone trail.
δ	For searching ants, this exponent term determines how quickly turning behavior approaches the baseline turning behavior determined by ω as time spent searching increases.
ε	For ants following a pheromone trail, determines the probability each time step that an ant will abandon the trail and begin searching before reaching its end. For lower values, ants tend to follow pheromone trails greater distances, and or more likely to follow trails to their end, where food was previously discovered.
η	Determines the rate at which pheromones evaporate. Higher values produce faster exponential decay of the pheromones from the grid.
λ	Determines the baseline probability that ants will leave a pheromone trail each time they pick up a piece of food. For values greater than or equal to one, ants leave pheromone trails each time they pick up food. Lower values correspond to decreased probability. For values below zero, the presence of other nearby food is required for ants to leave a pheromone trail. Density-dependent recruitment model only.
μ	Determines ants' sensitivity to the presence of other food when making a decision to leave a pheromone trail or not. With higher values, the presence of each additional piece of food in the neighborhood increases the probability of leaving a pheromone trail less. Density-dependent recruitment model only.

1a: Simple Recruitment Model



1b: Density-Dependent Recruitment Model

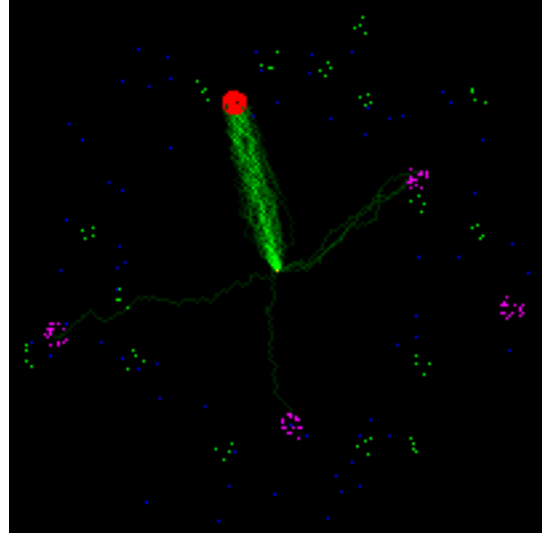
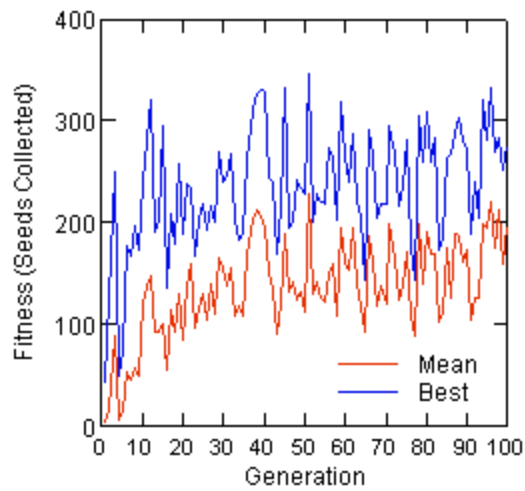


Figure 1: 1a) Simple recruitment model running a simulated foraging observation. 1b) Density-dependent recruitment model running a simulated foraging observation on an identical bait distribution as in 1a. Pheromone trails radiate from the centrally located nest, overlaid on top of baits as they appeared at the beginning of the simulation before foraging began. For the sake of clarity, ants are not displayed, and bait piles have smaller numbers of seeds than reported in text.

2a: Ten-ant colony on fully heterogeneous food



2b: 1000-ant colony on fully homogeneous food

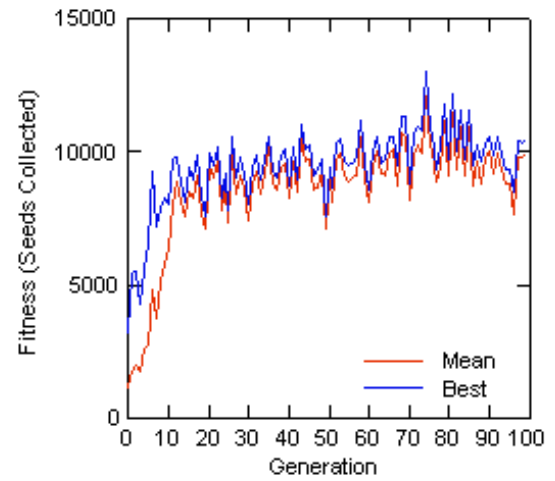
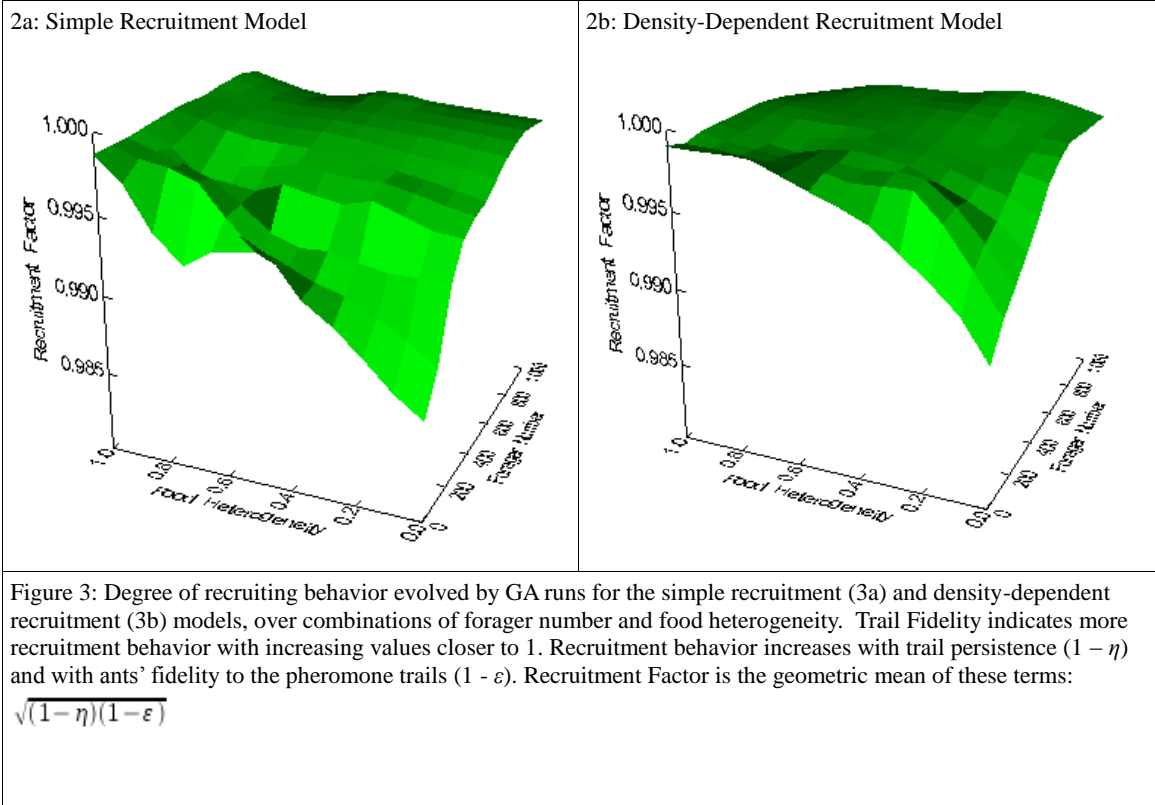


Figure 2: Sample Fitness Curves for GA runs. a) One hundred generation GA run for a ten-ant colony foraging on a fully heterogeneous food distribution. b) One hundred generation GA run for a 1000-ant colony foraging on a fully homogeneous food distribution. Fitness is the total number of seeds collected over eight simulations lasting 20,000 time steps each. The best and mean fitness in each generation are shown.



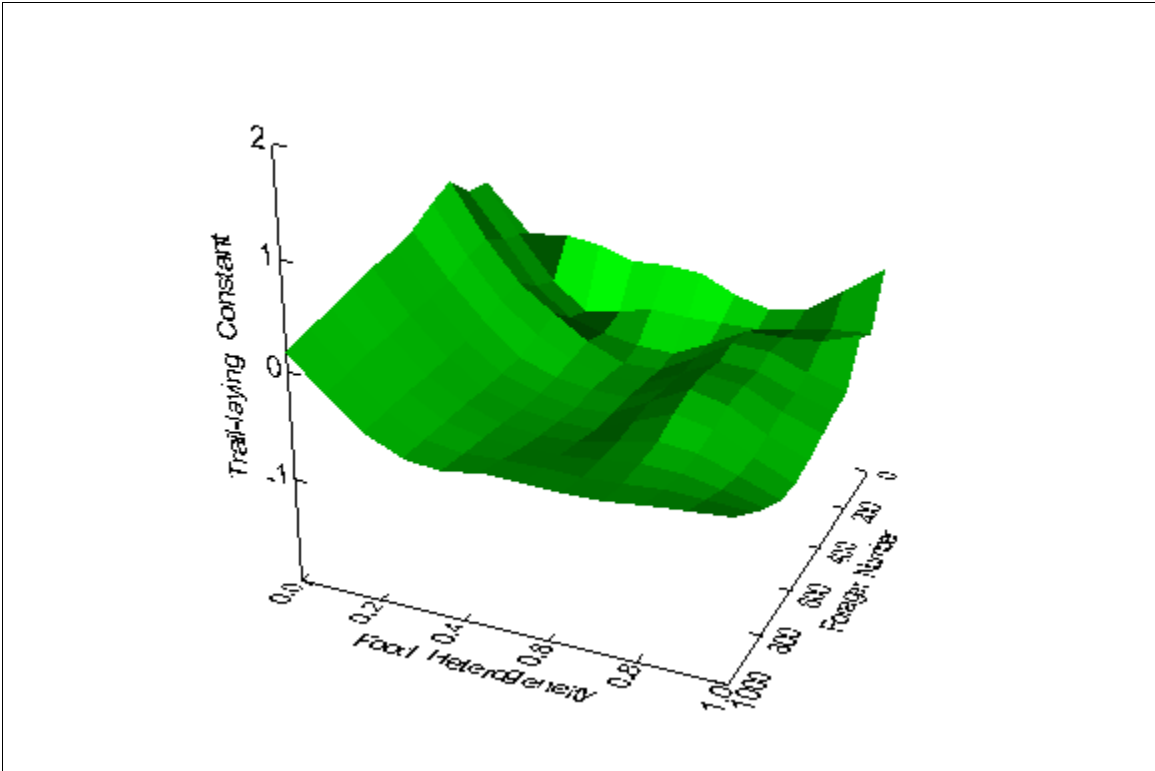


Figure 4: The relationship between colony size, food heterogeneity, and λ , the baseline probability that ants will leave a pheromone trail on the return trip to the nest with food. Greater values for λ indicate greater probability of leaving a pheromone trail each time a piece of food is picked up. Note that this figure is rotated to allow a clearer view of the surface, such that the X and Y axes are reversed relative to Figure 3.

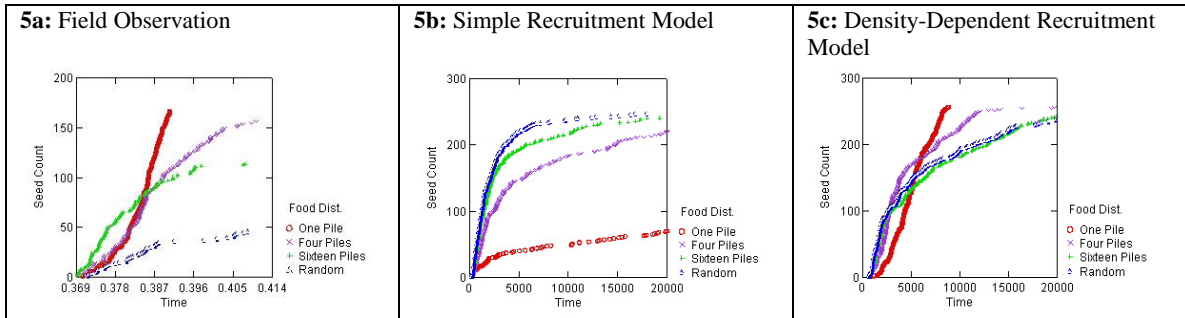
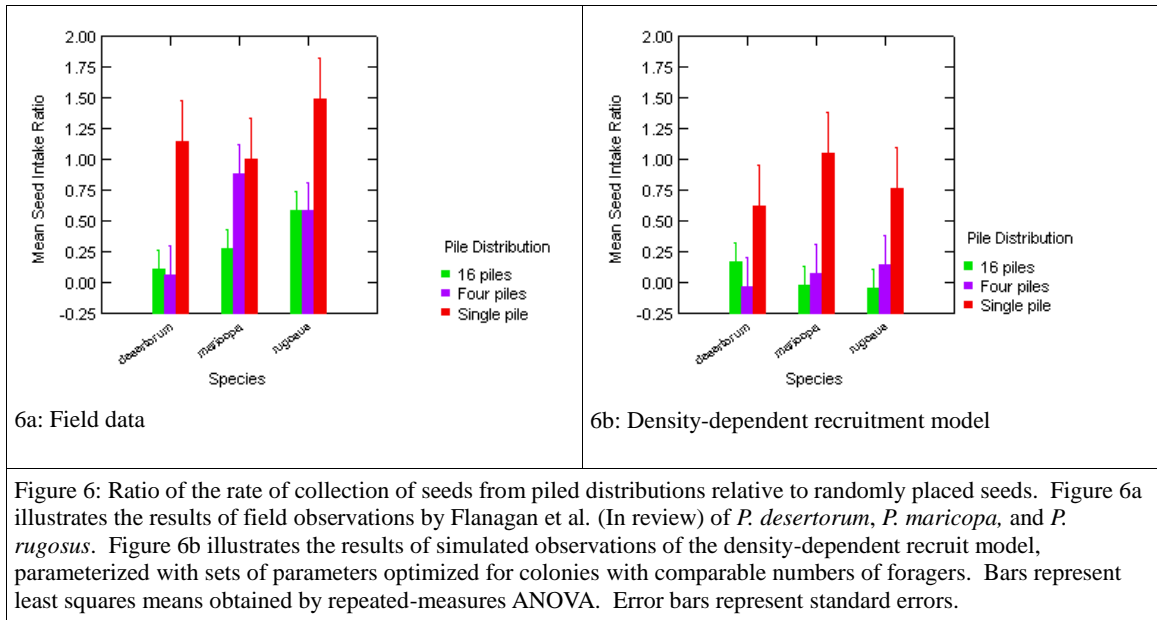


Figure 5: Foraging observations of ants given seed baits in four distributions. Figure 5a represents a field observation of a *P. rugosus* colony taken from Flanagan *et al* (In Review). Figures 5b and 5c represent a simulated foraging observation of the simple recruitment model and density-dependent recruitment models, each parameterized with values optimized for a 50% homogeneous and 50% heterogeneous food distribution.



CHAPTER 7

CONCLUSIONS

Despite the fact that violent conflict is a universal, species-typical behavior of humans, and feature of human societies, there is important variation in the frequency and intensity of conflict. The long-recognized pattern of geographical variation in conflict, with conflict most frequent and intense in tropical latitudes, especially in sub-Saharan Africa, equatorial South America, and southeast Asia, has been a subject of intense interest to political scientists. However, although researchers have identified many factors that predict this variation, such as resource scarcity and intergroup attitudes, until now the ultimate source of this broad geographical pattern has not been explained.

The findings of my research on variation in the frequency and intensity of conflict agrees with previous findings, that economic factors such as resource availability or uncertainty, and political systems and instability, are important causes of conflict. Previous research has concluded that, for example, relative poverty causes greater frequency of conflict (e.g. Fearon & Laitin, 2003), but has not gone on to explain why it is that countries at tropical latitudes are generally poorest and thus most conflict-prone. I have found that variation in the intensity of infectious disease, resulting from climatic variation favoring or disfavoring the transmission of infectious disease agents, causes variation in wealth among nations (Price-Smith, 2001) as well as in the intergroup attitudes of the people and cultures of those nations, ultimately driving the broad-scale geographic pattern of the frequency and intensity of conflict around the globe.

Selection by host-parasite coevolutionary processes for the expression of xenophobia, and the adaptive avoidance of out-group contact it encourages, is a critical link between infectious disease and intergroup conflict. Computer modeling of these coevolutionary processes, and resulting optimal mate choice preferences, reveals that, despite confirming the prediction that preference for genetic dissimilarity in mates should be optimal, out-group avoidance is favored because of the risks and costs of producing offspring with very novel genetic makeup that may not be well-adapted to local parasites, and also because of the risk of exposure to potentially highly virulent, novel parasites. The evolution of out-group avoidance in such mate choice decisions is foundational to intergroup relations, because these decisions establish patterns of between-group relatedness, shared or divergent fitness interests between the members of neighboring groups, and interest in cooperative or hostile intergroup relations. These set the stage for the pattern of variation in violent conflict among human groups that is apparent around the globe.

The behavior of harvester ants is designed by natural selection to maximize the foraging success of the colony (among other goals and constraints). The organization of foraging effort in an ant colony results from interactions of individual ants who behave in ways that have maximized colony reproductive success in the past. Harvester ants forage primarily on seeds, and these can be scattered randomly by the wind, but are often found heterogeneously deposited in depressions in the soil or under seeding plants. Thus each seed is a unit of food that is carried by an individual forager, yet these may be found in more or less dense patches on the ants' territory. Harvester ants in the field are able to exploit this heterogeneity to increase their seed collection rate when foraging in dense

patches. However, despite my prediction that larger colonies are able to exploit denser piles more effectively than small colonies, I found no difference in large colonies' treatment of piled foods relative to small colonies.

Computer modeling indicates that a recruitment behavior in which successful foragers leave a pheromone trail each time they return to the nest works poorly in this system, despite the prediction that trail reinforcement to sites where food is dense and search times are reduced should lead to convergence of foraging effort on these sites. I find that in this system, a conditional trail-laying behavior is optimal, in which successful foragers do not leave a pheromone trail on each return trip to the nest, but instead decide to lay a pheromone trail or not based on the presence of other locally available seeds. Following optimization by genetic algorithm for a mix of homogeneously and heterogeneously distributed seeds, I find that these models replicate the foraging behavior of ants in the field reasonably well. As I found with ants in the field, optimized models of large colonies showed no difference in their treatment of piled foods from optimized models of small colonies. This is because, although large colonies are expected to encounter piled foods more often, by virtue of their larger territories, the conditional recruitment behavior allows small colonies to reserve recruitment for the occasions when they do find densely piled foods. This allows them to exploit heterogeneity similarly to large colonies when given the opportunity to do so.

Social behavior has evolved independently numerous times on the tree of life. Distantly related taxa like humans and ants owe their incredible success to complex social behavior. The complex behavior of social groups emerges as the result of expression of adaptive social behavior by the individuals that make up these groups, and

is shaped by natural selection acting on individuals' behavior over the course of evolutionary time. Social behavior by any organism, and the resulting, emergent behavior of the groups those organisms compose, is shaped by natural selection to maximize inclusive fitness of the individual expressing that behavior, in environments typical of the evolutionary past. Thus, for social organisms whose environments vary, whether variation in infectious disease prevalence or in type or distribution of food, the optimal social behavior favored by natural selection varies as well, ultimately producing important variation in the behavior of social groups. This variation is apparent in broad-scale geographic patterns such as variation in the frequency and intensity of human violent conflict, and in variation among species of harvester ant living in sympatry.