Selection to outsmart the germs: The evolution of disease recognition and social cognition

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Abstract

The emergence of providing care to diseased conspecifics must have been a turning point during the evolution of hominin sociality. On a population level, such care may have minimized the costs of socially transmitted diseases at a time of increasing social complexity, although individual care-givers would have potentially incurred increased transmission risks while providing care. We propose that care-giving likely originated within kin networks where the costs of providing care may have been balanced by fitness increases obtained through caring for ill kin. We test a novel theory of hominin cognitive evolution in which disease may have selected for the cognitive ability to recognize when a conspecific is infected. Moreover, because diseases may produce symptoms that are likely detectable via the perceptual-cognitive pathways integral to social cognition, we suggest that disease recognition and social cognition may have evolved together. We use agent-based modeling to test 1) under what conditions disease can select for increasing disease recognition and care-giving among kin, 2) whether the strength of selection varies according to the disease's characteristics, 3) whether providing care produces greater selection for cognition than an avoidance strategy, and 4) whether care-giving alters the progression of the disease through the population. We compare the selection created by diseases with different fatality rates (i.e., similar to Ebola, Crimean-Congo hemorrhagic fever, measles, and scabies) under conditions where agents provide care to kin and under conditions where they avoid infected kin. The greatest selection was produced by the measles-like disease which had lower risks to the care-giver and a prevalence that was low enough that it did not disrupt the population's kin networks. When care-giving and avoidance strategies were compared, we found that care-giving reduced the severity of the disease outbreaks and subsequent population crashes. The greatest selection for increased cognitive abilities occurred early in the model runs when the

outbreaks and population crashes were most severe. Therefore, we conclude that over the course of human evolution, repeated introductions of novel diseases into naïve populations could have produced sustained selection for increased disease recognition and care-giving behavior, leading to the evolution of increased cognition, social complexity, and, eventually, medical care in humans. Finally, we lay out predictions derived from our disease recognition hypothesis of hominin cognitive evolution that we encourage paleoanthropologists, bioarchaeologists, primatologists, and paleogeneticists to test.

Key words: agent-based model, disease transmission, cooperation, hominin evolution, social complexity, kin selection

Introduction

Exposure to disease is a major cost of sociality (McCabe et al. 2015; Nunn and Altizer 2006; Rifkin et al. 2012). Despite this, hominins have evolved extraordinary social complexity (Tomasello 2014), including a strikingly social way of mitigating the effects of socially transmitted diseases—we provide care to diseased individuals. Such care hinges on the ability to recognize disease in others. Currently, the cognitive basis of this ability is not well understood. In this paper, we present the novel hypothesis that the ability to recognize disease may have evolved together with social cognition in hominins.

A synthesis of paleoanthropological, ethnographic, and host-parasite research suggests that increasing social complexity during the origin of *Homo* dramatically increased disease risk, i.e., (Harper and Armelagos 2013; McCabe et al. 2015; Rifkin et al. 2012; Sugiyama 2004). Thus, part of the selection for increasing cognitive abilities in *Homo* may have been selection to accurately assess the disease risk presented by interaction partners. We integrate findings from the literature on hominin social structure, hominin disease ecology, disease recognition in nonhuman animals, and human social cognition. Based on these data, we create an agent-based model to examine under what conditions increased cognition and care-giving could have evolved in the hominin lineage. Using our results, we create predictions deriving from our novel disease recognition hypothesis of hominin cognitive evolution that can be tested by paleoanthropologists, paleogenticists, bioarchaeologists, and primatologists.

Broadening social networks between hominin subgroups

Across birds and mammals, larger communities show greater levels of contagious parasites, environmentally transmitted parasites, and vector-borne parasites (Rifkin et al. 2012). Though

network modularity (sub-grouping) may reduce the transmission risks in large communities where many dyads do not interact (Griffin and Nunn 2012), hominin networks appear to have connected spatially distant subgroups, facilitating transmission within a fission-fusion, multi-level society (Grove et al. 2012; Hill et al. 2011).

Hominin community sizes have been reconstructed as having expanded over time, from ~50 in apes and small-brained australopiths to 100-120 in late *H. erectus* and *H. heidelbergensis* to 120-150 in *H. neandertalensis* and *H. sapiens* (Aiello and Dunbar 1993; Dunbar 1998; Gamble et al. 2011; Grove et al. 2012; Layton et al. 2012). This is believed to have produced an increase, not only in social network size, but also in complexity (Grove et al. 2012). As hominins dispersed towards northern latitudes and community sizes increased, the home-range requirements for sustaining them would have also increased (Grove et al. 2012). This produced communities whose daily nutritional needs were too large to be fulfilled in the amount of space a cohesive group could cover each day (Grove et al. 2012). The result is thought to have been the evolution of a multi-level fission-fusion system in which larger communities subdivide, rather than foraging cohesively (Grove et al. 2012). This would have enabled large communities of hominins to forage across greater areas and expand into new habitats, yet still obtain the benefits of a large social network, such as information transfer, social learning, and cooperation (Grove et al. 2012; Layton et al. 2012). Thus, even though mean population density decreased over time as hominins dispersed northward, overall community size and social network size likely increased (Grove et al. 2012; Layton et al. 2012).

Community size estimates for modern hunter-gatherers range from 125 to a few thousand (Layton et al. 2012). The extensiveness of human social networks was documented in a study showing that while chimpanzee males typically only interact with about 20 other males, a

113 modern male hunter-gather may watch over 300 other men make tools (Hill et al. 2014). The
114 evolution of such long-distance social networks linking different subgroups (Hill et al. 2014)
115 may have prevented the reduction in disease risk that might otherwise be expected to have
116 occurred as hominin density decreased, i.e., (Armelagos et al. 2005). Hominins' extensive,
117 community-wide social networks would have facilitated widespread pathogen transmission,
118 including any novel pathogens acquired as hominins spread into new habitats (McCabe et al.
119 2015).
120
121 **Increasing connectedness within groups**
122 Simultaneously with the expansion of networks connecting subgroups, the complexity of
123 networks within the subgroups also likely increased with the evolution of cooperative breeding
124 during the origin of *Homo.* Early *Homo* fossil assemblages show an increased number of
125 immature relative to mature individuals compared to australopith assemblages (Tobias 2006),
126 suggesting shortened interbirth intervals, increasing energetic demands on reproducing females,
127 and a shift towards cooperative breeding (Aiello and Key 2002). Ethnographic work supports
128 this view of humans as cooperative breeders, revealing greatly expanded social networks that
129 include multiple providers (hunting males, post-reproductive females) for females and young
130 (Hawkes 2003; Hill et al. 2009; Hrdy 2009). This contrasts with chimpanzees in which the young
131 are solely dependent upon their mothers (Burkart et al. 2009). Collectively, these studies suggest
132 that as community size increased during the origin of *Homo,* so did the complexity of the social
133 networks linking both greater numbers of individuals and different demographics (e.g., young
134 dependents, post-reproductive females, hunting males). The close cooperation, interdependence,

and density of social networks within cooperatively breeding hominin groups would have facilitated the spread of diseases within these groups (McCabe et al. 2015).

Hominin Disease Ecology

The shift to larger networks linking subgroups within a larger community and greater connectedness within cooperatively breeding groups is believed to have selected for enhanced social cognition (e.g., prosociality, shared-intentionality, theory of mind) which facilitated prolonged, close interactions among individuals and promoted social learning, cooperation, technological advances and cumulative culture (Burkart et al. 2014; Byrne and Bates 2007; Herrmann et al. 2007; Tomasello et al. 2005; van Schaik et al. 2012; Whiten 2000). However, such intense, close proximity interactions would have also facilitated disease transmission (McCabe et al. 2015). Recent work in genetics and evolutionary medicine indicates that hominins harbored numerous pathogens before the advent of agriculture and animal domestication (Harper and Armelagos 2013). This includes endoparasitic worms (Hoberg et al. 2001; Hurtado et al. 2008), lice (Harper and Armelagos 2013), tuberculosis (Stone et al. 2009), typhoid fever (Harper and Armelagos 2013), whooping cough (Harper and Armelagos 2013), and viruses, e.g., herpes viruses, Epstein Barr virus (Harper and Armelagos 2013). Thus, hominins were likely under strong selection to assess the disease status of others.

Disease recognition in animals and humans

Comparative evidence suggests that disease recognition may have been present in early hominins (citations below). Several species with relatively low social complexity have been documented to recognize disease, often either avoiding diseased conspecifics or taking advantage

158 of sick and weakened competitors, e.g., social lobsters (Behringer et al. 2006), pipefish
159 (Rosenqvist and Johansson 1995), bullfrog tadpoles (Kiesecker et al. 1999), rodents (Kavaliers et
160 al. 1997), house finches (Bouwman and Hawley 2010; Zylberberg et al. 2012), but see (Nunn
161 2003). While the underlying cognitive processes are not well understood, these studies suggest
162 that recognition is based on diverse symptoms including olfactory/chemical cues (Kavaliers et al.
163 1997; Kiesecker et al. 1999), visual detection of spots (Rosenqvist and Johansson 1995), and
164 behavioral changes including lethargy and feather fluffing (Bouwman and Hawley 2010;
165 Zylberberg et al. 2012). Though the amount of cognitive processing required to detect disease
166 may differ by symptom type, the wide array of cues and recognition in multiple species suggests
167 that some simple form of disease recognition could have been basal in hominins.
168 Infectious pathogens can cause noticeable symptoms that could potentially be detected via
169 the perceptual-cognitive pathways that are integral to social cognition in primates. Subtle
170 differences perceived in conspecific faces (Leopold and Rhodes 2010; Sartori et al. 2011), voices
171 (Belin 2006; Belin et al. 2004), and movement/gait (Loula et al. 2005; Peterman et al. 2014;
172 Sartori et al. 2011) may enable, not only the decoding of conspecifics' identities, emotions, and
173 intentions, but also facilitate the detection of disease. This could include changes in facial
174 coloration and texture due to fever, rashes, or nasal discharge, changes in vocalizations due to
175 coughing, nasal discharge or reduced lung capacity, and changes in movement/gait due to
176 weakness, lethargy, or signs of pain (Chapman et al. 2005; Fink and Matts 2008; Hart 1988).
177 Thus, if the detection of social information and disease involve the same perceptual-cognitive
178 pathways, then disease circulating within hominin populations may have selected for increased
179 cognitive capacities and care-giving.

Importantly, such disease recognition would *not* require individuals to have an abstract concept of disease. Following the well-accepted definition of cognition as information processing, e.g., seminal book: (Neisser 1967), recent publications: (Byrne and Bates 2007; Deaner et al. 2006; Fernandes et al. 2014; Herrmann et al. 2007; Lee 2007; Reader et al. 2011; Woodley et al. 2015), the cognitive aspect would be processing the proximate cues that distinguish healthy individuals from diseased individuals (changes in appearance, behavior, etc.). Selection for such disease recognition would operate at the ultimate level of causation (Sherman 1988; Tinbergen 1963), favoring individuals who were able to discriminate who was healthy and who was not. Those who avoided infectious individuals or provided care to ill kin would increase their reproductive fitness. Similarly to how kin recognition can operate without individuals having an abstract concept of kin (Rendall 2004), disease recognition could operate without a concept of disease.

Care-giving among animals and humans

The literature contains numerous reports of striking cases of social care given by animals, including dolphins that cooperatively supported a dying conspecific who could no longer swim (Park et al. 2013), an elephant that attempted to lift a collapsed and dying conspecific to her feet (Douglas-Hamilton et al. 2006), primates that groom, stand watch over, and/or chase others away from dying group members (Anderson et al. 2010; Bezerra et al. 2014; Nakamichi et al. 1996), and an otter group that provisioned an elderly female (Davenport 2010). Though very interesting, these reports do not provide evidence of widespread long-term care which would be expected to have a more significant selective influence on a species' evolution.

Some of the best opportunities for systematically investigating care-giving in animals have come from studies of populations with high prevalences of severe injuries (Beamish and O'Riain 2014; Byrne and Stokes 2002; Stokes and Byrne 2006) or congenital disabilities (Turner et al. 2014). These studies generally suggest that, instead of relying on social care, severely injured or disabled individuals survive by adapting and making adjustments themselves, rather than receiving accommodation or assistance (Beamish and O'Riain 2014; Byrne and Stokes 2002; Stokes and Byrne 2006; Turner et al. 2014). The exception to this is social grooming (Dittus and Ratnayeke 1989). Wound cleaning has been shown to be an important mechanism for avoiding infections and it is widespread in animals (Dittus and Ratnayeke 1989; Hart 2011). Thus wound cleaning may have been a basal form of social care in hominins.

In addition, evidence from modern foraging, hunting, and horticultural peoples, suggests that provisioning people who are ill or injured is important in reducing the mortality rate (Sugiyama 2004). For example, Sugiyama (2004) found that over 50% of individuals reported at least one time in their lives when they were incapacitated and could not forage for at least a month. During such times, provisioning was critical to their survival (Sugiyama 2004). Based on this evidence, we expect that hominins could have significantly reduced the mortality arising from disease and infection-related injuries through provisioning (Sugiyama 2004) and wound cleaning (Dittus and Ratnayeke 1989). Additionally, food sharing networks of hunting males also served as provisioning networks during times of illness (Gurven et al. 2000; Sugiyama 2004; Sugiyama and Chacon 2000), suggesting that the evolution of social care may have co-evolved with cooperative breeding.

Care-giving in the fossil record

225 Fossil evidence of hominins surviving illness, injuries, and disabilities goes back nearly 2
226 million years to include fossils from *H. erectus, H. heidelbergensis, H. neandertalensis,* and *H.*
227 *sapiens*. While the following discussion is not exhaustive, it does illustrate the variety of
228 conditions hominins survived, the time depth of the fossil record, and the taxa included. Below
229 we follow, when possible, the taxonomic classifications provided in Grove et al. (2012). In *H.*
230 *erectus* this includes: premortem loss of all but one tooth in the 1.77 mya cranium and mandible
231 from Dmanisi (D3444 and D3900 (Lordkipanidze et al. 2005; Lordkipanidze et al. 2006)),
232 possible hypervitaminosis A in the 1.6 mya KNM-ER 1808 (Walker et al. 1982), evidence of a
233 herniated disc in the 1.5-1.6 mya Nariokotome boy KNM-WT 15000 (Grove et al. 2012;
234 Haeusler et al. 2013; Schiess et al. 2014), and a healed cranial lesion caused by trauma or
235 burning in the 0.6 mya Hulu 1 cranuim, also called Nanjing 1 and Tangshan 1 (Shang and
236 Trinkaus 2008; Wu et al. 2011). Among *H. heidelbergensis* this includes craniosynostosis and
237 neurocranial deformities in a 0.53 mya immature, cranium 14, who survived for at least
238 approximately 5 years (Gracia et al. 2009), a 0.53 mya adult male pelvis and lumbar spine, SH
239 Pelvis 1, showing lesions and degeneration possibly resulting from lumbar kyphotic deformity,
240 spondylolisthesis, and Baastrup disease (Bonmati et al. 2010), and a squamous temporal lesion
241 that shows healing on the 0.35 mya Broken Hill cranium Kabwe 1 (Grove et al. 2012; McBrearty
242 and Brooks 2000; Montgomery et al. 1994). For Neandertals this includes Aubesier 11, dated to
243 at least 0.17 mya, which shows significant tooth loss and alveolar lesions (Lebel and Trinkaus
244 2002; Lebel et al. 2001) and Shanidar 1 dated at 73-40 kya who lost much of his right arm, may
245 have been blind on one side, and suffered from hyperostotic disease (Crubezy and Trinkaus
246 1992; Hublin 2009). *H. sapiens* individuals that survived severe conditions include: a child,
247 Qafzeh 12 dated to approximate 0.095 mya, who showed signs of hydrocephaly and survived

until about 3 years old (Tillier et al. 2001), an older child Qafzeh 11, also dated to 0.95 mya, that had a healed cranial fracture (Coqueugniot et al. 2014), and an adult female, Dolní Věstonice 3, dated to approximately 0.027 mya, who sustained a severe injury to her face that might have interfered with eating (Trinkaus et al. 2006; Trinkaus and Jelinek 1997).

While all of these individuals *might* have benefited from care, comparative evidence with nonhuman primates suggests that care is not necessary (DeGusta 2002, 2003; Dettwyler 1991). Studies of wild baboons and great apes show that primates frequently survive even when a hand or foot is maimed or severed, e.g., in snares (Beamish and O'Riain 2014; Byrne and Stokes 2002; Munn 2006; Stokes and Byrne 2006). Though these animals may show changes to their activity budgets (Beamish and O'Riain 2014), altered locomotion patterns (Munn 2006), and reduced feeding efficiency (Byrne and Stokes 2002; Stokes and Byrne 2006), survival appears to be high, with some groups having as many as ~20% of their members permanently disabled (Munn 2006). Extensive tooth loss also appears to be survivable. Apes and other primates have been observed to survive antemortem tooth loss comparable to that observed in the fossil record (Cuozzo and Sauther 2004; DeGusta 2002). Degusta (2002) provides a review of cases in which chimpanzees were observed to survive with tooth loss similar to Aubersier 11 and Cuozzo and Sauther (2004) reported that tooth loss is common among ring-tailed lemurs, with one individual surviving with 80% tooth loss. Overall the evidence from the fossil record and animal studies indicate that while various fossils have clearly survived severe health conditions, it is very difficult to rule out the possibility that they may have survived without care (DeGusta 2002, 2003; Dettwyler 1991).

The modeling approach

It is currently not possible to determine when extensive social care evolved in the human lineage, but it is possible to consider *how* it might have evolved and what conditions might have selected for it. We expect that, because kinship is a fundamental property of primate (including human) social networks (Silk 2009), providing care to the diseased may have originated along kin networks. Hamilton's rule of inclusive fitness (Hamilton 1964) predicts that individuals will act altruistically when: (benefit to the recipient)*(relatedness to recipient) > (costs to the altruist). Thus, individuals could increase their own reproductive fitness in two ways: 1) by avoiding ill individuals, particularly nonkin, and 2) by providing care to ill kin who, upon recovery, would reproduce. Whether the fitness benefits are greater when individuals avoid ill conspecifics or provide care (thus risking becoming infected) will depend upon the benefits, the degree of relatedness, and the costs.

We use agent-based modeling to test a varying intensity of disease scenarios and quantify selection pressures for increased cognition and care-giving. Agent-based models provide powerful, quantitative insights into disease transmission, including predicting the impact of current/future outbreaks and planning intervention/prevention strategies, e.g., influenza (Guo et al. 2015), Ebola (Merler et al. 2015). We take the innovative approach of applying these techniques to reconstruct the potential impact of disease on hominin evolution.

A modeling approach is valuable because, while our knowledge is increasing, i.e.,(Harper and Armelagos 2013), we do not have sufficiently detailed data concerning how/when disease load changed during hominin evolution to be able to test whether the evolution of care-giving co-occurred with increasing cognitive abilities, social complexity and disease risk. Therefore, we use agent-based modeling to examine under which conditions disease could select for increased cognition and care-giving. We hypothesize that 1) disease will produce care-giving among kin

294 and an increase in average population intelligence, that 2) varying disease characteristics will
295 produce variation in the strength of selection, and that 3) care-giving will produce greater
296 selection for cognition than an avoidance strategy.
297
298 **Material and methods**
299 *Study design*
300 We created two models for comparison. The first (Model 1: Care-giving model) simulates
301 disease transmission in a population of hominins who give care (The ODD description is in
302 Appendix A at the end of the paper. The code is available in supplementary file 1). In order to
303 more fully explore the model and how care-giving may alter the progression of disease through
304 the population, we then created a control model (Model 2: Avoidance only) similar to the first
305 except that agents avoid diseased kin and provide no care. (The ODD description is in Appendix
306 B at the end of the paper. The code is available in supplementary file 2).
307
308 ***Model 1: Care-giving model***
309 *Disease characteristics*
310 We programmed an SIS model (susceptible – infected – susceptible) in Netlogo 5.0.5 (Railsback
311 and Grimm 2011; Wilensky 1999). We created four hypothetical diseases with case fatality rates
312 modeled after Ebola [2014 outbreak: 70% (Aylward et al. 2014; WHO 2014a), Crimean-Congo
313 hemorrhagic fever (40% (WHO 2013), CCHF, hereafter), measles (~10% (WHO 2014b)), and a
314 low risk comparison, such as scabies (fatality rate set at 1%, though scabies is generally not fatal
315 (WHO 2015)]. We did not attempt to precisely simulate the natural history of these diseases.

316 Rather, these diseases were chosen to represent a range of fatality rates occurring in socially
317 transmitted diseases.
318
319 *Optimizing the disease transmission rates*
320 Because transmission rates have complex relationships with virulence and host density (e.g.,
321 trade-off hypothesis (Alizon et al. 2009)), we screened possible transmission rates to determine
322 what would be optimal for persistence of these diseases in this population. For the Ebola-like,
323 CCHF-like, and measles-like diseases, we ran the model 1000 times in Netlogo's
324 BehaviorSpace, varying the probability of transmission from 10-100% by increments of 10. For
325 the scabies-like disease, we ran the model 1000 times varying the probability of transmission
326 from 1% to 98.5% by increments of 2.5. The inclusion of lower transmission values for the
327 scabies-like disease is based on literature showing that less virulent diseases tend propagate
328 slower, e.g., (Alizon et al. 2009; Ewald 1993). Then, for each disease, we selected the runs which
329 had both healthy and diseased individuals after 100 time steps. We averaged the probability of
330 transmission across those successful runs to obtain a transmission rate that is optimal for each
331 respective disease: Ebola-like 78%, CCHF-like 33%, measles-like 10%, scabies-like 2%. The
332 higher transmission rates in the diseases with higher fatality rates is consistent with the
333 relationship between virulence and transmission documented in the literature (Alizon et al.
334 2009).
335
336 *Determining the probability of recovery after care*
337 We expect that the earliest forms of social care given by hominins would have been
338 assistance with hygiene, including keeping wounds, sores, and topical infections clean as in

nonhuman primates (Dittus and Ratnayeke 1989), provisioning those who are too ill to forage with food and water (Sugiyama 2004), and watching over individuals who may be too ill to themselves be vigilant against predators (Anderson et al. 2010; Bezerra et al. 2014; Nakamichi et al. 1996). None of these forms of care requires medical knowledge, yet evidence from nonhuman primates (Dittus and Ratnayeke 1989) and human foraging groups (Sugiyama 2004) suggests that they are effective at reducing mortality rates.

It is difficult to estimate how effective each of these care-giving techniques would be for each of our hypothetical diseases. In nature, the more incapacitated the individual is and the longer the recovery takes, the greater the chances that the individual would succumb to dehydration, starvation, or predation unless care is given. Because we did not wish to bias the effectiveness of the care towards the more severe diseases, we set the probability of recovery after care at 0.5 for all diseases. This reflects an equal chance of recovery and failure to recover.

The population

The landscape is a 40 x 40 cell grid that wraps horizontally and vertically. Each cell represents 5 km^2, making the landscape 200 km^2. This is within the confidence intervals of the space requirements calculated for a community of *H. erectus, H. heidelbergensis, H. neandertalensis,* and *H. sapiens* using a gas model in Grove et al. (2012). Table 1 summarizes the group sizes, densities, and space requirements presented in Grove et al. (2012).

[Table 1]

The carrying capacity of the landscape is set at 200. Two hundred was chosen because it is large enough to encompass the group sizes predicted for hominins based on cranial capacities, brain volumes, and neocortex ratios of fossil hominins [Table 1, (Aiello and Dunbar 1993;

Gamble et al. 2011; Grove et al. 2012)], but is generally smaller than community sizes reported for modern humans, e.g., (Hill et al. 2014; Layton et al. 2012). We set the carrying capacity above the calculated community sizes for hominins, e.g., ~150 or smaller (Aiello and Dunbar 1993; Dunbar 1998; Gamble et al. 2011; Grove et al. 2012), to allow for the event that the actual community sizes of the model populations would likely be lower than the carrying capacity.

Initialization

The program is initialized with 10 agents randomly placed on the landscape. Each agent is randomly assigned an intelligence score (0-1). In the model the intelligence score is the likelihood of an agent correctly identifying the disease status of another agent. We refer to it as intelligence because we expect that the ability to recognize disease is related to a more general ability for efficient information processing, including social information, e.g., (Byrne and Bates 2007; Deaner et al. 2006; Fernandes et al. 2014; Herrmann et al. 2007; Lee 2007; Reader et al. 2011; Woodley et al. 2015). As the population grows, each offspring's intelligence is drawn from a normal distribution with the parent's intelligence as the mean and a standard deviation of 0.15.

Population growth and genetic structure

The population grows at each time step of the model when healthy agents reproduce according to the formula: [(1 - (number of agents / carrying-capacity)) * number of healthy agents]. Reproduction occurs asexually. Offspring are placed within a radius of 3 of the parent, producing spatial clustering of kin as is consistent with human and nonhuman primate groups (Chapais and Berman 2004; Hatchwell 2010; Hill et al. 2011; Silk 2009).

Relatedness is tracked by links between agents with the links containing the relatedness value. Parent-offspring relationships receive relatedness values of 0.5 and offspring inherit the links of the parent but with ½ the relatedness value. Because offspring inherit the links of the parent, sibling relationships are included in the model with a relatedness value 0.25. To prevent the model from becoming too computationally intensive, patrilineal relationships and matrilineal relationships beyond a relatedness of 0.25 were not modeled. This decision is supported by findings showing that kin recognition occurs most reliably for close matrilineal kin identified via familiarity, e.g., (Chapais and Berman 2004; Chapais et al. 1997). The population represents a single, kin structured community with multiple matrilines. Space displays the contact structure between agents and random movement simulates mixing within the population.

Space

With a carrying capacity of 200 individuals and a landscape of 200 km^2, our model has a maximum population density of 1 individual / km^2, which is within the confidence intervals calculated for *H. habilis* and *H. erectus* [Table 1, (Grove et al. 2012)]. However, the purpose of our model is not to attempt to reconstruct a particular hominin species or population. We made this decision because the population densities and number of levels of fissioning have been reconstructed to vary dramatically even within species, depending upon the habitat quality and latitude (Atkinson et al. 2008; Grove et al. 2012; Powell et al. 2009). Instead, hominin societies are conceptualized as more generic fission-fusion communities in which subsets of individuals are out of contact with other subsets of individuals (Grove et al. 2012; Layton et al. 2012). This is represented in our model by the restrictions created by the movement, care-giving, and infection radii. The care-giving radius (5) and infection radius (5) are equal to reflect that agents

who are close enough to give care are also close enough to become infected. Similarly, agents who avoid infectious kin by moving away will also be moving away from potential care-givers should they themselves become infected. These radii of 5 represent 25 km^2 and are in the upper range of the distance that modern hunter-gatherers travel from camp when they will return to camp later the same day (Grove et al. 2012; Layton et al. 2012).

Disease and care-giving

After four time steps of the model, 25 agents are randomly infected with one of the diseases. This is approximately 16% of the population and reliably seeded the disease into the population without increasing to 100% prevalence.

Healthy agents evaluate the relatedness and disease status of other agents within a radius equivalent to 5 grid cells. The infection radius is also set at 5, thus any healthy agent that can provide care, is also close enough to be infected.

Kin are accurately recognized and the accuracy of disease recognition is a function of the agent's intelligence. A random number between 0-1 is drawn. If the number is below the agent's intelligence value, the disease status is correctly recognized. Otherwise, the agent's disease status is incorrectly recognized (healthy kin are classified as diseased or diseased kin are classified as healthy). These individuals make up the group the agent *perceives* to be its diseased kin (perceived diseased kin). Whether the error is a false positive (healthy kin classified as diseased) or a false negative (diseased kin classified as healthy) is determined by the disease status of the kin agent. Thus, the likelihoods of false positive and false negative errors are functions of disease prevalence. As the proportion of diseased agents increases, false positives decrease and false negatives increase.

Agents randomly select one of their perceived diseased kin and decide whether to provide care based on a modification of Hamilton's rule, which predicts altruism when: (relatedness between the recipient and altruist)*(benefit to the recipient)>(cost to the altruist) (Hamilton 1964). We adapted this formula so that agents provide care when: (relatedness between the care-giver and the recipient)*(probability of recovery after care) > (probability of transmission to care-giver)*(probability of infection being fatal). If the inequality is fulfilled (thus care is given) and the recipient was in fact diseased (not just *perceived* to be diseased), a random number between 0 and 1 is generated and if it is below the probability of recovery, the diseased individual recovers. If the random number was above the probability of recovery, the recipient remains diseased. A new random number is drawn for the care-giver and if it is below the probability of transmission to the care-giver, then the care-giver is infected. If the recipient was erroneously categorized as diseased, but is actually healthy (a false positive error), there is no change in the disease statuses of the recipient or the care-giver. It is worth noting that when a false negative error occurs (diseased kin are classified as healthy), the agent that made the error does not incur a cost that is explicitly coded into the model. However, the agent does potentially incur emergent costs through the interactions between agents. This may occur in two ways: a) if that diseased kin agent dies (later in the model run), this reduces the kin network available to give care, simulating a loss of inclusive fitness to the agent that failed to recognize the disease in its kin, and b) the presence of diseased kin in the population increases the risk that others will become infected, including the agent that failed to recognize the disease in its kin.

If healthy agents have no perceived diseased kin, they move to a grid cell with no other agents on it within a radius of 8. If no empty cells are available, the agent does nothing. A movement radius of 8 represents 40 km^2. This is the median daily *total* travel distance used by

Grove et al. (2012) to calculate hominin area requirements and it is based on data compiled from modern hunter-gathers, e.g., (Layton et al. 2012).

Avoidance of infectious individuals

If the randomly selected recipient (from the agent's perceived diseased kin) does not fulfill the inequality for receiving care, the agent moves to a grid cell with no other agents on it within a radius of 8. This can occur due to a low relatedness with the recipient of care, high costs of exposure to the disease, or a low likelihood of recipient recovery. Under these conditions, the agent avoids the diseased individual instead of providing care. Note that nonkin do not receive care, thus if no perceived diseased kin are within the care-giving radius, the agent moves.

Because the care-giving radius and the infection radius are set at 5 and this is less than the movement radius (8), agents that do not provide care can move out of the infection radius. The effectiveness of movement as a disease avoidance strategy is based on chance and the density of infected individuals. By chance the healthy agent may move to a grid cell that is outside of the infection radius of the diseased agent. However, as the density of infected agents increases, so does the likelihood that the healthy agent will move to a grid cell that is within the infection radius of another diseased agent. This reflects the difficulties of avoiding exposure at when there is a high density of infectious individuals in the population.

If no empty cells are available, the agent does nothing.

Mortality and disease transmission

The model generates a random number for each diseased agent. If the number is below the probability of fatality, that agent dies. All healthy agents have a probability of becoming infected

from any infected agent within a radius of 5 grid cells, based on the probability of transmission. Five grid cells represent the upper range of the daily travel radius for modern hunter-gatherers (25 km^2) (Grove et al. 2012; Layton et al. 2012). A random number (0-1) is drawn for each healthy agent in danger of infection. If the number is below the probability of transmission, the agent is infected. If an agent is in danger of infection from more than one diseased agent, the process is repeated for each infectious agent in 5 grid cells.

Model analysis

We ran the model 2000 times for 100 time steps for each disease. We considered runs to be successfully completed when both the disease and population had persisted (defined as ≥ 1 diseased agent and ≥ 1 healthy agent at 100 time steps). The first 1000 successfully completed runs were divided into 10 groups of 100. We calculated average population size, average disease prevalence, average percentage of diseased individuals who received care (percent care), and average population intelligence at each time step across the 100 runs. This created an n of 10 average runs for which we made curves depicting the changes in each of these output variables for the four diseases we considered. We used the 10 averages in the subsequent statistical tests instead of the original 1000 runs to avoid inflating our sample size, and thus the power of our tests (Railsback and Grimm 2011).

Statistics

We compared the endpoints of the curves by comparing the output variables (average population size, average disease prevalence, and average percent care) across the diseases at time step 100 using one-way ANOVAs (n=10 average runs/disease). We calculated the change in

500 average population intelligence between the first and 100th time step, tested whether the
501 differences were different from zero using one-sample T-tests, and whether these differences
502 varied across disease types using a one-way ANOVA. We calculated maximum slopes for the
503 curves of the average percent care and the average population intelligence using grofit (Kahm et
504 al. 2010) in R 2.13.1 (RCoreTeam 2011) and RStudio 0.98.1062 (RStudio 2014). We tested
505 whether the slopes differed across disease types using a one-way ANOVA. Some violations of
506 normality and equal variances existed (Supplementary files 3 and 4). One-way t-tests were
507 bootstrapped with 1000 samples for robusticity to non-normality and 95% bias corrected
508 accelerated confidence intervals were calculated (Field 2013). Though one-way ANOVAs are
509 generally robust to such violations when groups have equal sample sizes, when variances were
510 unequal, we used the Brown-Forsythe F-ratio. Alpha was set at 0.05 and multiple comparisons
511 across disease types were Bonferroni corrected when variances were equal and Tamhane T2
512 corrected when they were unequal. Statistical tests were run in SPSS Statistics 22 or 23 unless
513 otherwise stated.
514
515 ***Model 2: Control model – Avoidance only***
516 Following the initial analysis of the care-giving model (Model 1), we programmed a control
517 (Model 2: Avoidance only) to further explore how care-giving may have altered the progression
518 of disease through hominin populations. This model used the same population and diseases, but
519 differed in two ways. First, agents who have perceived diseased kin avoid them instead of
520 providing care. All agents with perceived diseased kin move randomly to an empty grid cell
521 within a radius of 8. Second, if the agent has no perceived diseased kin or there are no empty
522 grid cells within a radius of 8, the agent does not move. This differs from the care-giving model

in which agents with no diseased kin also move to an empty grid cell within 8. (Because agents that give care do not move, this was necessary in the care-giving model to ensure movement within the population.) We made this second change to the avoidance model to be conservative with respect to our expectation that only care-giving will produce intelligence changes. This second change increased selection on avoidance behavior because in Model 2 (Avoidance only) the only opportunity agents have to move is when they are avoiding diseased kin.

Model analysis and statistics

We used the same procedure as above to create 10 average runs for each output variable for each disease. We conducted one-sample T-tests to determine whether the difference in average population intelligence between the first and 100^{th} time steps were significantly different from zero for the scabies-like, measles-like, CCHF-like, and Ebola-like diseases. We used two sample T-tests to determine whether the population size, prevalence, and intelligence at the 100^{th} time steps differed between models 1 and 2. Some violations of normality and equal variances existed (Supplementary files 3 and 4). T-tests were bootstrapped with 1000 samples for robusticity to non-normality and 95% bias corrected accelerated confidence intervals were calculated (Field 2013). When Levene's test showed violations of the assumptions of equal variances, we report results calculated without assuming equal variances (Field 2013). Alpha was set at 0.05.

Analysis of the intelligence curves produced by Model 1 (Care-giving)

We analyzed the trajectories of the intelligence curves of the 10 average runs for each disease using linear mixed-models run in R 3.2.4 (RCoreTeam 2015) using the nlme package. We use this approach to relate infection prevalence to changes in mean intelligence, while taking

into account population size. We test for an interaction between prevalence and population size on changes to mean intelligence by including interaction term in the model: prevalence * population size. As the data are longitudinal (i.e., time series) we allow for autocorrelated errors using an ARMA process, incorporate time as a fixed effect, and use the averaged simulation run as the random effect. We check for issues of multicollinearity using variation inflation factor, and check the residuals of the models for non-normality, heteroscedasticity, and autocorrelation. (model: change in mean intelligence ~ time + prevalence*population size + random intercept). In order to keep the paper focused on the evolution of increasing average population intelligence, we did not conduct this analysis on the Model 2 curves, which showed either no increase or a decrease in average intelligence.

Results

Model 1: Care-giving model

After 100 time steps the four diseases produced significantly different population sizes, disease prevalence, percentages of the diseased who received care, and average population intelligences (Tables 2-3, Fig. 1).

[Table 2]

[Table 3]

[Figure 1]

The Ebola-like disease, unlike the other three, produced no care-giving and no change in average population intelligence (Table 4, Fig. 1).

[Table 4]

Both the Crimean-Congo hemorrhagic fever-like (CCHF-like) and measles-like diseases show initial increases in both care-giving and intelligence followed by a plateau (Fig. 1). The CCHF-like disease produced a care-giving rate of 4.7%, a final intelligence level of 0.62, and a 12% net change in intelligence. Of the four diseases, the measles-like disease produced the highest rate of care-giving (6.7%) and the highest average population intelligence (0.71) at the final time step. This was generated by the greatest maximum slopes for care-giving and intelligence changes and the greatest net change in intelligence over time (21%). The scabies-like disease showed a strikingly different pattern. As prevalence steadily increased, because the fatality rate was low, care-giving decreased. Infected individuals did not provide care and rarely died, meaning that the number of healthy individuals able to provide care decreased. This produced a negative slope for care-giving, though low increases in average population intelligence were still observed (care-giving rate: 1.4%, final average population intelligence: 0.53, net intelligence change: 3%, Tables 2-3).

Model 2: Control model – Avoidance only

The model two results revealed two important findings. First, an avoidance strategy did not result in an increase in average population intelligence (Tables 5 and 6). The net change in intelligence overtime was not significantly different from zero under the scabies-like and measles-like conditions (Table 5). Under the CCHF-like and Ebola-like conditions the average population intelligence decreased significantly (Table 5).

[Table 5]

[Table 6]

Second, a visual inspection of Figures 2-4 shows that the progression of the diseases through the population differed under Model 1 (care-giving) and Model 2 (avoidance only). Descriptive statistics are provided in Supplementary file 5. For the scabies-like and measles-like diseases, when agents gave care the final population sizes were higher and the final prevalences were lower (Fig. 2 & 3, Table 6). A visual inspection of Fig. 3b reveals that when agents give care, the "boom and bust" cycle of disease outbreaks in the population was reduced with prevalence increasing and decreasing less dramatically. For the CCHF-like disease, the final population sizes differed however prevalence did not differ (Table 6). An inspection of Fig. 3c shows that the cycle of outbreaks was very similar in the care-giving and avoidance conditions. For the Ebola-like disease, final population size and final prevalence did not differ in the care-giving and avoidance conditions.

[Fig. 2]

[Fig. 3]

[Fig. 4]

Analysis of the intelligence curves produced by Model 1 (care-giving)

For each of the scabies-like, measles-like, and CCHF-like diseases, time was negatively related to changes in intelligence (Table 7). Thus, the largest increases occurred early in the run with smaller increases occurring later. In the case of the Ebola-like disease, intelligence did not change, thus there was no relationship between time and changes in mean intelligence.

For the scabies-like disease, VIF scores indicated high collinearity between dependent variables (VIF scores >100). When we dropped population size from the analysis, VIF scores fell below 7. In this reduced analysis, changes in intelligence were positively related with prevalence (Table 7, Fig. 5).

For the measles-like disease, changes in intelligence were positively related with both prevalence and population size with the greatest increases in intelligence occurring at larger population sizes and high prevalences (Fig. 6). For the CCHF-like disease, the proportion of the variation explained by the analysis (marginal $R^2 = 0.15$) was reduced compared to the measles-like (marginal $R^2 = 0.57$) and scabies-like (marginal $R^2 = 0.47$) diseases. However, similar to the measles-like disease, an interaction effect between prevalence and population size was present, indicating that at low prevalences, changes in intelligence were negatively related to population size, but at higher prevalences, they were positively related with population size (Fig. 7). Thus the greatest changes in intelligence occurred at low prevalences and low population sizes or high prevalences and high population sizes.

No relationships between time, prevalence or population size were found for the Ebola-like disease because the Ebola-like disease produced no changes in intelligence (Tables 5 and 7, Fig. 8).

[Table 7]

[Fig. 5]

[Fig. 6]

[Fig. 7]

[Fig. 8]

Discussion

General discussion

Our findings suggest that the evolution of care-giving may have created a profound shift in how hominins evolved in the presence of their pathogens. The avoidance approach (Model 2) likely represents the basal condition, under which disease either does not select for or against increasing cognitive abilities (high prevalence, low fatality diseases) or selects against it (low prevalence, high fatality diseases). In contrast, under the care-giving condition (Model 1), care-giving not only selected for increasing cognitive abilities, but also altered and controlled the progression of some of the diseases throughout the population. We discuss both models and their implications in detail below.

Model 1

Our results from Model 1 suggest that disease circulating among kin can select for care-giving among kin and greater cognitive abilities. Furthermore, the diseases produced selection of varying strengths, with higher care-giving rates producing greater increases in average population intelligence.

The findings are relevant to the evolution of care-giving in hominins as they suggest that not all diseases produce care-giving behavior. The high fatality and transmission rates of the Ebola-like disease, when applied to Hamilton's rule (Hamilton 1964), generated costs that were greater than the benefits of care-giving, even to close relatives, thus, all agents avoided ill kin, rather than providing care. Such diseases are not likely to have facilitated the evolution of care-giving or increased social cognition. The CCHF-like disease had intermediate probabilities of fatality and transmission, leading to care-giving only to close kin (parents and offspring: r=0.5), and not

660 to more distant relatives like grandparents, grandchildren, or siblings (r=0.25) who were avoided
661 when ill. This produced substantial care-giving behavior and selection for increasing
662 intelligence, but the selection was weaker than for the measles-like disease, where care was
663 given to both close and more distant relatives. The scabies-like disease, while it produced care-
664 giving for both close and more distant relatives, produced only low rates of care-giving and
665 correspondingly weak selection for increasing intelligence. These effects result from the very
666 low fatality rate of the scabies-like disease; the population size appears to have been regulated
667 largely by the carrying capacity set in the model (i.e., habitat supports 200 individuals) rather
668 than by the disease. Therefore, as disease prevalence increased, there was a lack of healthy
669 individuals who could provide care to their diseased kin, leading to a low rate of care-giving,
670 lower population turnover, and lower increases in average population intelligence. Overall, these
671 simulations suggest that diseases that are most likely to have led to the evolution of care-giving
672 in the human lineage were those with low costs to caregivers which persisted at a prevalence low
673 enough not to disrupt the kin networks along which care was provided. Although only healthy
674 agents could give care and reproduce in our model, high rates of costly care-giving may not be
675 expected if kin have sublethal diseases that do not reduce their reproductive success.
676 It is noteworthy that for all three diseases that produced care-giving, the final rate of care-
677 giving was low, with a maximum of 6.7% of the diseased receiving care under measles-like
678 conditions. Furthermore, a recovery rate of only 50% after care suggests that over the course of
679 hominin evolution even low rates of relatively ineffective care may have been sufficient to select
680 for increasing intelligence and disease recognition. We expect that the first forms of care-giving
681 among hominins would have included assistance with hygiene, such as cleaning of wounds and
682 topical infections (Dittus and Ratnayeke 1989) and provisioning with food and water (Sugiyama

683 2004). These mechanisms would not have required an understanding of disease processes and
684 could have piggybacked on basal social grooming behaviors observed in nonhuman primates
685 (Dittus and Ratnayeke 1989) and communal provisioning behaviors that may have evolved
686 during the evolution of cooperative breeding (Burkart et al. 2009; Gurven et al. 2000; Hawkes
687 2003; Hill et al. 2009; Hrdy 2009; Sugiyama 2004; Sugiyama and Chacon 2000).
688
689 *Model 2*
690 The Model 2 results demonstrate that avoidance alone does not select for greater cognitive
691 abilities. Avoidance produced no net change in average population intelligence in the scabies-
692 like and measles-like conditions and a *decrease* in average population intelligence in for the
693 CCHF-like and Ebola-like diseases. The scabies-like and measles-like diseases produced higher
694 population sizes and disease prevalences *above* 50%, thus an agent who moves away from
695 infected kin is likely to encounter other infected individuals. This results in a lack of selection for
696 disease recognition and avoidance. In contrast, the CCHF-like and Ebola-like diseases produced
697 lower population sizes and prevalences *below* 50%, thus an agent who avoids infected kin is less
698 likely to encounter other infected agents. This results in selection to isolate oneself. The most
699 efficient way for agents to isolate themselves in a population with a prevalence under 50%, is to
700 miscategorize healthy individuals as ill, thus triggering avoidance. Because lower intelligence
701 agents have less accurate disease recognition, this produces selection to *decrease* intelligence.
702 These findings are relevant for species that do not give care. It suggests that avoidance of
703 high prevalence, low fatality diseases is likely to be an ineffective strategy. As a result these
704 diseases do not exert selection for or against cognitive abilities under an avoidance only

paradigm. In contrast, avoidance is an effective strategy against low prevalence, high fatality diseases producing selection for avoidance behavior and selection against sociality.

Implications of care-giving

A comparison of the results from Model 1 (care-giving model) with Model 2 (avoidance model) indicates that care-giving alters the progression of the disease through the population. For the scabies-like and measles-like diseases, care-giving resulted in significantly higher population sizes and lower prevalences than an avoidance only strategy. Thus for these diseases, which are the two diseases for which care was given to both close and distant kin ($r=0.5$ and $r=0.25$, respectively), care-giving served to control the disease in the population.

Two of the diseases, the measles-like and the CCHF-like diseases, show distinct cycles of disease outbreaks and population crashes ("boom and bust" dynamic, Fig. 2-3). The lack of congruence between the relatively constant slope of the intelligence curves (Fig. 4) and the boom-bust oscillations of population size and prevalence, is a reflection of the fact that selection on intelligence is occurring throughout the boom-bust cycle and not intermittently only when specific conditions are met (e.g., a particular population size or prevalence). This dynamic is quantified through the interaction term of the mixed model analysis in which intelligence increases are the result of complex interactions between prevalence and population size. Because the two diseases progress differently through the population, they also exert selection on intelligence in slightly different ways. The measles-like disease produces one oscillation of the boom-bust outbreak cycle of population and prevalence peaks and crashes; the CCHF-like disease produces multiple, more rapid oscillations.

727 The measles-like disease shows a very pronounced "bust" phase early in the run. Population
728 size is high when the disease is first introduced (Fig. 2B, Model 1 curve). This produces a high
729 rate of care-giving and strong selection for intelligence (left panel, Fig. 6B). As the prevalence
730 increases (Fig. 3B, Model 1 curve), low intelligence matrilines recognize diseased kin less
731 accurately, and provide less successful care, causing them to succumb to the disease. This
732 produces a decrease in population size and an increase in average population intelligence (Fig.
733 4B, Model 1 curve). At high prevalences, selection for intelligence is maintained regardless of
734 the population size (right panel, Fig. 6B). Intelligence plateaus about half way through the run
735 when the population size rebounds slightly but remains low and prevalence decreases slightly
736 from its earlier peak and remains moderate. With a low population size, intermediate prevalence,
737 and a decreased rate of care-giving (Fig. 1B, measles-like curve), the population maintains the
738 higher intelligence, but does not continue to increase it (change in intelligence approaches 0 in
739 left side of middle panel, Fig. 6B). Intelligence plateaus as the boom-bust outbreak oscillations
740 cease.
741 The CCHF-like disease produces a very pronounced boom-bust cycle with several peaks and
742 crashes in population size and prevalence. Selection for increasing intelligence occurs both
743 during low population sizes and low prevalences (left panel, Fig. 7B) and during high population
744 size and high prevalences (right panel, Fig. 7B). When the boom-bust dynamic stops about
745 halfway through the run and the population stabilizes at intermediate population sizes and
746 prevalences, intelligence plateaus (Figs. 2C, 3C, 4C Model 1 curves and middle panel, Fig. 7B).
747 Interestingly, when the population infected with the measles-like disease engages in care-
748 giving, it experiences less pronounced oscillations of the "boom and bust" outbreak cycle (Fig.
749 3) indicating that care-giving serves to control the spread of the disease through the population.

Because of the higher risks of providing care under the CCHF-like conditions, only close kin (r=0.5) receive care. This lower level of care is less effective at controlling the spread of the disease, perhaps suggesting that a certain threshold must be achieved in order to disrupt the boom-bust outbreak cycle (boom-bust dynamics: (Keeling and Grenfell 1997)). Alternatively, the higher fatality rate and more rapid transmission of the CCHF-like diseases produces faster outbreak cycles, which may make it more difficult for care-giving to disrupt the boom-bust outbreak cycle even though it still selects for increasing cognitive abilities.

For both the measles-like and CCHF-like diseases, the most pronounced outbreaks occur early in the model run, which is also when the greatest increases in intelligence are occurring (Fig. 6A and 7A). In the second half of the run, when the boom-bust dynamic is less pronounced, intelligence plateaus. This suggests that over the course of human evolution, sustained increases in intelligence may have occurred through repeated introductions of novel diseases into naïve populations. The greatest selection would have occurred shortly after the introduction when the disease was spreading and care-giving behavior had not yet managed to reduce the size of the outbreaks and subsequent population crashes.

Significance for human evolution

Our model was parameterized based upon group sizes, spatial scales, and population densities derived from the fossil record and modern foraging peoples (Grove et al. 2012; Layton et al. 2012). Our goal was not to recreate a particular hominin population, but to explore the effects of different disease characteristics on the evolution of care-giving and increased cognition in a population with hominin characteristics.

We created an SIS model (susceptible-infected-susceptible) where recovered individuals are just as susceptible as those who were never infected. However, for many diseases, recovered individuals are temporarily or permanently immune to re-infection, potentially increasing their ability to provide care. We expect that immunity would increase the rate of care-giving. Diseases likely to select for care-giving among kin may be diseases which frequently infect children and then convey lifetime immunity. Under this scenario, adults who survived to reproduce would have extensive knowledge of the disease's symptoms, making recognition likely, and the immunity to enable them to provide effective care. Several well-known childhood diseases that follow this pattern (e.g., measles, smallpox) have been dated to the origins of agriculture, animal domestication, and the subsequent population increases (Harper and Armelagos 2013). However, as more genetic studies are conducted, increasing numbers of pathogens are showing pre-agricultural origins, including some that were previously believed to be post-agricultural (e.g., tapeworms, TB (Harper and Armelagos 2013; Hoberg et al. 2001; Hurtado et al. 2008; Stone et al. 2009). Tapeworms, TB, typhoid fever, whooping cough, and Epstein Barr virus, among others, have been shown to predate agriculture (Harper and Armelagos 2013; Hoberg et al. 2001; Hurtado et al. 2008; Stone et al. 2009), suggesting that ancestral hominins harbored significant numbers of infectious diseases. Based on our models, diseases with low risks to care-givers, high inclusive fitness pay-offs for care-givers, and prevalences low enough not to disrupt the kin networks along which care could be given would have exerted the strongest selection for increased cognition. Through repeated introductions of novel diseases over millions of years, such diseases could have selected for accurate disease recognition, increased care-giving among kin, and produced the social and cognitive origins of human medical care.

795 **A novel hypothesis of human cognitive evolution and future directions**

796 Our novel hypothesis of primate, including human cognitive evolution, is ***not*** mutually
797 exclusive with the social brain hypothesis (Dunbar 1998). As social species evolved the
798 cognitive capacities for social cognition, such as processing information gleaned from faces
799 (Leopold and Rhodes 2010; Sartori et al. 2011), voices (Belin 2006; Belin et al. 2004), and
800 movement patterns (Loula et al. 2005; Peterman et al. 2014; Sartori et al. 2011), they may have
801 also obtained the ability to use this information to recognize disease symptoms. They could
802 detect changes in facial coloration and texture due to fever or rashes, changes in vocalizations
803 due to coughing, nasal discharge or reduced lung capacity, and changes in movement/gait due to
804 weakness, lethargy, or signs of pain (Chapman et al. 2005; Fink and Matts 2008; Hart 1988). The
805 proximate mechanisms are relatively simple in that they do ***not*** require individuals to have an
806 abstract concept of "disease." Instead, individuals that are able to accurately recognize disease
807 would have increased fitness due to being able to avoid infectious individuals or provide care to
808 kin. Though studies of disease recognition in nonhuman animals are relatively rare, several
809 species do appear to recognize the health status of conspecifics, i.e., social lobsters (Behringer et
810 al. 2006), pipefish (Rosenqvist and Johansson 1995), bullfrog tadpoles (Kiesecker et al. 1999),
811 rodents (Kavaliers et al. 1997), house finches (Bouwman and Hawley 2010; Zylberberg et al.
812 2012), but see (Nunn 2003).

813 We predict that as hominin social complexity increased, i.e., group sizes, social network
814 sizes, frequencies of cooperation and social learning, etc. (Aiello and Dunbar 1993; Burkart et al.
815 2014; Burkart et al. 2009; Dunbar 1998; Gamble et al. 2011; Grove et al. 2012; Layton et al.
816 2012; Tomasello 2014), hominins would have substantially increased their risk of disease
817 transmission, producing heightened selection for disease recognition and care-giving. We make

818 several predictions that enable paleoanthropologists, archaeologists, primatologists, human
819 ecologists, geneticists and immunologists to test our novel hypothesis of human cognitive
820 evolution:

821 1) Humans and nonhuman primates have very similar disease profiles in that we share many
822 of the same diseases with viral, bacterial, and gastrointestinal parasitic zoonoses
823 occurring from nonhuman primates to humans and vice versa (Chapman et al. 2005;
824 Jones et al. 2008; Lloyd-Smith et al. 2009; Wolfe et al. 2007). However, what has
825 received very little attention is how humans and nonhuman primates may differ in the
826 expression of disease symptoms. Humans, relative to nonhuman primates have much less
827 body hair. Though our nakedness may reduce ectoparasite load (Pagel and Bodmer 2003;
828 Weiss 2007), it also provides a visually unobstructed surface for displaying rashes,
829 lesions, swelling, and inflammation, and bruising. Humans, relative to nonhuman
830 primates, also have white scaleras around their eyes, a signal that has been argued to
831 draw attention to gaze direction (Kobayashi and Kohshima 2001; Tomasello et al. 2007),
832 but also turns a dramatic "bloodshot" red when we are under emotional stress or ill
833 (Provine et al. 2011). ***Prediction 1:*** *If humans have been selected to solicit care from*
834 *others, they should display exaggerated signals of ill health, relative to nonhuman*
835 *primates experiencing the same disease and degree of morbidity/mortality.*
836 2) It is becoming increasingly possible to date the origins of many diseases afflicting
837 humans i.e., (Harper and Armelagos 2013; Stone et al. 2009). As more accurate dates are
838 obtained for more diseases, it will be possible to examine whether hominin populations
839 carried an increased disease load as they increased in social complexity. Social
840 complexity could be operationalized in the fossil record through the brain size – group

size relationship (Aiello and Dunbar 1993; Dunbar 1998; Gamble et al. 2011; Grove et al. 2012; Layton et al. 2012), through evidence of increased behavioral and technological complexity in the archaeological record (Gowlett et al. 2012; Shultz et al. 2012), or through fossil evidence for the shift to cooperative breeding (Aiello and Key 2002; Shultz et al. 2012). ***Prediction 1*****:** *If larger hominin communities sustained greater disease loads, then periods of rapidly increasing community sizes (operationalized with expanding brain sizes (Aiello and Dunbar 1993; Dunbar 1998; Gamble et al. 2011; Grove et al. 2012)) should coincide with the evolution of diseases new to hominins.* ***Prediction 2:*** *If social learning/cooperation lead to increased disease transmission (McCabe et al. 2015), then increasing behavioral/technological complexity in the archaeological record (Gamble et al. 2011; Gowlett et al. 2012; Shultz et al. 2012) should coincide with the evolution of diseases new to hominins.* ***Prediction 3:*** *If cooperatively breeding increased disease transmission, then evidence for cooperative breeding in the fossil record (Aiello and Key 2002; Shultz et al. 2012) should coincide with the evolution of diseases new to hominins, particularly those that afflict children.* These predictions are not mutually exclusive. According to the results of our model, we would expect a high proportion of these diseases to present low costs and high fitness payoffs to care-givers and persist at prevalences that are low enough not to disrupt the kin networks along which care is provided. Possibilities include infections that leave survivors immune.

3) An additional avenue for examining the role of disease during the evolution of human social complexity would be through cross-species comparisons of immune investment. If hominins have experienced an unusually high rate of disease exposure, either through their extensive social networks or through providing care to diseased kin, they may have

invested heavily in immune defenses. Recent work on introgression between anatomically modern humans (AMH) and neandertals has proposed that one of the major advantages may have been the acquisition of novel immune genes from neandertals as AMH expanded northward into novel environments and encountered novel pathogens (Houldcroft and Underdown 2016). Prior studies indicate that there are cross-species differences in immune investment according to mating system (but not group size or density in primates) (Nunn et al. 2000), the risk of environmentally transmitted parasites and injuries due to predator attacks in anthropoids (Semple et al. 2002), coloniality in birds (Moller et al. 2001), and cooperative breeding in birds (Spottiswoode 2008). ***Prediction 1*****:** *If hominins' increased social complexity required them to invest heavily in immune defenses, the human immune system should show similar adaptations to other species that have extremely large social networks and high interaction rates.* ***Prediction 2:*** *If the evolution of cooperative breeding required hominins to invest heavily in immune defenses, then the human immune system should show similar adaptations to other cooperatively breeding species.* ***Prediction 3:*** *If the evolution of providing care to diseased conspecifics required hominins to invest heavily in immune defenses, the human immune system should show adaptations that are either extreme or unusual.* (These predictions are not mutually exclusive). While many of the earlier studies were done with white blood cell counts, i.e., (Nunn et al. 2000), the field of ecological immunology is growing rapidly with new techniques being continually developed (Downs et al. 2014; Larsen et al. 2014). This should make it increasingly possible to parse out how different selective forces may have acted on different elements of a species' immune system.

887 **Conclusions**

888 Our model indicates that disease circulating amongst kin groups can select for care-giving
889 among kin and greater cognitive abilities. Moreover, the characteristics of the diseases can
890 generate different strengths of selection. Diseases with lower costs and higher pay offs produced
891 stronger selection, yielding higher care-giving rates and greater increases in average population
892 intelligence.

893 When a care-giving strategy was compared with an avoidance only strategy, the care-giving
894 strategy controlled the transmission of the disease through the population by reducing the
895 severity of disease outbreaks and population crashes. Because this cycle of outbreaks and
896 population crashes was associated with the most rapid increases in intelligence, we propose that
897 the repeated introduction of novel diseases into naïve populations may have led to sustained
898 selection for increasing disease recognition and cognitive abilities throughout human evolution.
899 Moreover, the unique ability of hominins to control the spread of disease through care-giving
900 behaviors may have facilitated increased social complexity, and ultimately lead to the evolution
901 of medical care in humans. Finally, we set out predictions derived from our disease recognition
902 hypothesis of hominin cognitive evolution that can be tested by paleoanthropologists,
903 archaeologists, geneticists, and primatologists.

904

905 **Data accessibility**

906 The ODD descriptions of Model 1 (caregiving) and Model 2 (avoidance only) are found in
907 Appendices A and B, respectively. The code is available in supplementary files 1 and 2,
908 respectively. The files containing the code can be opened with standard text editing programs
909 such as WordPad.

Conflict of interests

None.

Authors' contributions

SEK designed the study, programmed the model, analyzed the data, and wrote the manuscript. TRB and CAC contributed to all stages. RWB contributed to the development of the ideas and manuscript preparation.

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Tables

Table 1. Summary data calculated from the hominin dataset presented in Appendix Table A1 of Grove et al. (2012). Values and confidence intervals are medians calculated from the published dataset. To keep our terminology consistent, we refer to community size where Grove et al. (2012) refers to group size.

		Community Size			Population Density (I/km2)			Area Required (km2)		
Genus	**Species**	**Lower CI**	**Median**	**Upper CI**	**Lower CI**	**Median**	**Upper CI**	**Lower CI**	**Median**	**Upper CI**
Homo	Early *Homo*	43.249	56.276	71.402	0.366	0.584	0.802	51.529	92.525	188.043
Homo	*habilis*	46.8415	60.476	76.2795	0.577	0.822	1.068	43.8705	73.56	132.306
Homo	*erectus*	66.43	83.158	102.406	0.545	0.785	1.025	70.289	113.994	200.766
Homo	*heidelbergensis*	70.9845	88.389	108.389	0.3	0.514	0.728	94.736	164.6655	339.368
Homo	*neanderthalensis*	72.622	90.266	110.5325	0.196	0.407	0.618	116.066	217.395	536.199
Homo	*sapiens*	78.763	97.292	118.541	0.196	0.407	0.618	127.537	240.876	613.916

Table 2. Means and standard deviations for each disease for the final population size, final disease prevalence, final percent care, final average population intelligence, the net intelligence change between time steps 1 and 100 (Intel Change), the maximum slope for percent care, and the maximum slope for average population intelligence from Model 1 (Care-giving).

Disease	**Pop. Size**		**Prevalence (%)**		**Percent Care**		**Intelligence**		**Intel Change**		**Slope Care**		**Slope Intel**	
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
Scabies	184.07	0.77	84.78	0.42	1.37	0.11	0.53	0.01	0.03	<0.01	-0.00006	0.00003	0.0006	0.00007
Measles	133.64	2.02	70.15	0.76	6.74	0.43	0.71	0.01	0.21	0.01	0.00053	0.00006	0.0043	0.00032
CCHF	120.96	3.47	33.63	1.75	4.73	0.50	0.62	0.01	0.12	0.02	0.00022	0.00005	0.0025	0.00042
Ebola	157.24	3.25	10.32	0.51	---	---	0.50	0.01	0.00	0.01	---	---	0.0003	0.00020

Table 3. One-way ANOVAs showing significant differences across disease types for the final population size, final disease prevalence, final percent care, final average population intelligence, the net intelligence change between time steps 1 and 100, the maximum slope for percent care, and the maximum slope for average population intelligence for Model 1 (Care-giving).

All multiple comparisons between disease types were significant, thus only the smallest mean difference and corresponding p-value are shown per test.

Test	F-statistic	Df	P	Smallest Mean Difference	P
Final Pop. Size	1131.78^{BF}	3, 24.47	<0.001	$\geq 12.68^{T}$	<0.001
Final Prevalence	$11{,}275.24^{BF}$	3, 15.24	<0.001	$\geq 0.15^{T}$	< 0.001
Final Percent Care	492.03^{BF}	2, 18.61	<0.001	$\geq 0.02^{T}$	<0.001
Final Intelligence	579.51^{UC}	3, 36	<0.001	$\geq 0.03^{B}$	<0.001
Intelligence Change	464.463^{BF}	3, 23.13	<0.001	$\geq 0.03^{T}$	<0.001
Max. Slope Percent Care	377.10^{UC}	2, 27	<0.001	$\geq 0.0003^{B}$	<0.001
Max. Slope Intelligence	421.732^{BF}	3, 21.61	<0.001	$\geq 0.0002^{T}$	≤ 0.03

UC F-statistic, uncorrected

BF Brown-Forsythe F-statistic

B Bonferroni correction for multiple comparisons

T Tamhane's T2 test for multiple comparisons

Table 4. One-sample T-tests on the ***Model 1 results*** showing that the difference in average population intelligence between the first and 100th time steps were significantly different from zero for the scabies-like, measles-like, CCHF-like diseases, but not for the Ebola-like disease. Significant p-values are bolded

Test	**T**	**Df**	**P**	**CI: Lower**	**CI: Upper**
Scabies-like	22.18	9	**<0.001**	0.028	0.033
Measles-like	44.78	9	**<0.001**	0.196	0.216
CCHF-like	19.36	9	**<0.001**	0.111	0.137
Ebola-like	-0.824	9	0.431	-0.010	0.005

Table 5. One-sample T-tests on the ***Model 2 results*** showing that the difference in average population intelligence between the first and 100th time steps were significantly different from zero for the CCHF-like and Ebola-like diseases, but not for the scabies-like and measles-like diseases. Significant p-values are bolded.

Test	**T**	**Df**	**P**	**CI: Lower**	**CI: Upper**

Scabies-like	-.997	9	0.352	-0.005	0.001
Measles-like	-1.292	9	0.236	-0.025	0.005
CCHF-like	-24.000	9	**0.001**	-0.160	-0.138
Ebola-like	-58.939	9	**0.001**	-0.216	-0.200

Table 6. Two-sample T-tests comparing population size, prevalence, and mean intelligence values at the 100th time step for each disease under Model 1 (care-giving) versus Model 2 (avoidance) conditions. When Levene's test indicated that the variances are unequal, we report the T values, degrees of freedom (df), p-values, and confidence intervals calculated without assuming equal variances (Field 2013). Significant p-values are bolded.

Disease	**Variable**	**T**	**Df**	**P**	**CI: Lower**	**CI: Upper**
Scabies-like	Pop. Size	43.178	11.011	**0.001**	28.833	31.344
	Prevalence	-49.675	18	**0.001**	-0.105	-0.096
	Intelligence	7.786	18	**0.001**	0.031	0.052
Measles-like	Pop. Size	9.669	18	**0.001**	9.621	14.569
	Prevalence	-3.000	18	**0.016**	-0.029	-0.007

	Intelligence	30.699	11.148	**0.001**	0.205	0.233
CCHF-like	Pop. Size	-3.165	18	**0.003**	-5.906	-1.296
	Prevalence	0.740	18	0.464	-0.007	0.015
	Intelligence	37.944	18	**0.001**	0.254	0.282
Ebola-like	Pop. Size	-0.024	14.171	0.982	-3.696	3.923
	Prevalence	0.305	18	0.748	-0.004	0.005
	Intelligence	46.049	18	**0.001**	0.200	0.218

1234

1235 Table 7. Mixed-model analyses run on the Model 1 (care-giving) results examining the effects of prevalence, population size and the
1236 interaction between the two on intelligence changes for each disease. r2m measures how much variation in mean intelligence
1237 can be explained by the fixed effects (time+prevalence*population size). β values are standardized regression coefficients. SE
1238 is the standard error and df is the degrees of freedom.

Disease	**Analysis**	**r^2m***	**Variable**	**B**	**SE**	**df**	**t**	**p**
Scabies-like*	Prevalence	0.468	Intercept	-0.002	0.034	888	-0.055	0.956
			Time	-1.084	0.086	888	-12.641	<0.001
			Prevalence	0.460	0.085	888	5.411	<0.001
Measles-	Prevalence	0.565	Intercept	-0.065	0.075	946	-0.871	0.384

like			Time	-0.585	0.076	946	-7.650	<0.001
			Population Size	0.291	0.063	946	4.590	<0.001
			Prevalence	0.431	0.046	946	9.276	<0.001
			Population Size*Prevalence	-0.143	0.021	946	-6.713	<0.001
CCHF-like	Prevalence	0.146	Intercept	0.039	0.050	946	0.785	0.433
			Time	-0.400	0.051	946	-7.848	<0.001
			Population Size	0.052	0.051	946	1.014	0.311
			Prevalence	-0.104	0.052	946	-2.023	0.043
			Population Size*Prevalence	0.060	0.020	946	3.023	0.003
Ebola-like	Prevalence	0.001	Intercept	0.008	0.039	946	0.218	0.827
			Time	-0.010	0.039	946	-0.247	0.805
			Population Size	-0.043	0.049	946	-0.873	0.383
			Prevalence	0.002	0.073	946	0.031	0.976
			Population Size*Prevalence	0.013	0.022	946	0.571	0.568

***** r^2c values were the same as r^2m. r^2c measures how much variation is explained by the whole model (including the random effect of simulation run). That the two measures were the same indicates that there were no systematic differences between runs of a given disease.

Figure legends

Figure 1. Changes over time in disease prevalence (A), percentage of diseased individuals who received care (B), and average population intelligence (C). For each disease the 10 average runs have been averaged within each time step. The Ebola-like, CCHF-like, measles-like, and scabies-like diseases are shown in red circles, green squares, black Xs, and blue triangles, respectively. Approximately every fourth time step is shown. Error bars are +/- two standard deviations. Fig. 1B does not show the Ebola-like disease because no care was given.

Figure 2. Changes in population size over time produced by Model 1 (Care-giving) and Model 2 (Avoidance only) in the scabies-like (A), measles-like (B), CCHF-like (C), and Ebola-like (D) diseases. Models 1 and 2 are shown in red circles and blue triangles, respectively. Only even numbered time steps are shown. Error bars are +/- two standard deviations.

Figure 3. Changes in prevalence over time produced by Model 1 (Care-giving) and Model 2 (Avoidance only) in the scabies-like (A), measles-like (B), CCHF-like (C), and Ebola-like (D) diseases. Models 1 and 2 are shown in red circles and blue triangles, respectively. Only even numbered time steps are shown. Error bars are +/- two standard deviations.

Figure 4. Changes in average population intelligence over time produced by Model 1 (Care-giving) and Model 2 (Avoidance only) in the scabies-like (A), measles-like (B), CCHF-like (C), and Ebola-like (D) diseases. Models 1 and 2 are shown in red circles and blue triangles, respectively. Only even numbered time steps are shown. Error bars are +/- two standard deviations.

Figure 5. Graphs showing the results of the analyses exploring the effects of prevalence on the change in intelligence for the scabies-like disease. Change in intelligence was calculated as the mean intelligence in a given time step minus the mean intelligence in the previous time step. (A) Change in intelligence is negatively correlated with time and (B) positively correlated with prevalence (Table 7).

Figure 6. Graphs showing the results of the analyses exploring the effects of prevalence, population size, and their interactions on the change in intelligence for the measles-like disease. Change in intelligence was calculated as the mean intelligence in a given time step minus the mean intelligence in the previous time step. (A) Change in intelligence is negatively correlated with time (Table 7). (B) Interaction effects between population size and prevalence ("Prev"). Population size is on the X axis with data points represented by the small black lines. The difference in intelligence is shown on the Y axis. The prevalences shown represent the range of prevalences experienced by the population (see Figure 1A). The greatest positive selection on intelligence occurred when prevalence and population size are high. Population size has a large effect when prevalence is low (left panel of B) and a small effect when prevalence is high (right panel of B).

Figure 7. Graphs showing the results of the analyses exploring the effects of prevalence, population size, and their interactions on the change in intelligence for the CCHF-like disease. Change in intelligence was calculated as the mean intelligence in a given time step minus the mean intelligence in the previous time step. (A) Change in intelligence is negatively correlated with time (Table 7). (B) Interaction effects between population size and prevalence. Population size is on the X axis with data points represented by the small black lines. The difference in intelligence is shown on the Y axis. The prevalences shown represent the range of prevalences experienced by the population (see Figure 1A). The greatest increases in average population intelligence occurred at low population sizes and low prevalences (B, left panel) and at high population sizes and high prevalences (B, right panel).

Figure 8. Graphs showing the results of the analyses exploring the effects of prevalence, population size, and their interactions on change in intelligence for the Ebola-like disease. Change in intelligence was calculated as the mean intelligence in a given time step minus the mean intelligence in the previous time step. (A) No significant change in intelligence over time. (B) Potential interaction effects between population size and prevalence. Population size is on the X axis with data points represented by the small black lines. The difference in intelligence is shown on the Y axis. The prevalences shown represent the range of prevalences experienced by the population (see Figure 1A). Because intelligence does not change over time, there are no significant correlations with prevalence, population size or the interaction of the two (Table 7).

Appendix A. **ODD Protocol for *Selection to Outsmart the Germs* in Netlogo (Model 1: Care-giving)**

Purpose

The purpose of this model is to test 1) under what conditions disease can select for increasing disease recognition and care-giving among kin and 2) whether the strength of selection varies according to the disease's characteristics. We compare the selection produced by diseases with fatality rates similar to Ebola, Crimean-Congo hemorrhagic fever, measles, and scabies.

Entities, state variables and scales

This model consists of three entities: the landscape, agents moving on the landscape, and links between agents. The landscape is a 40 x 40 cell grid that wraps horizontally and vertically. The model space simulates individuals moving and interacting on a landscape. The grid cells do not have any variables of their own.

The following global variables can be user-adjusted via the interface:

1) **Carrying-capacity**: maximum number of agents on the landscape
2) **Prob-fatality**: the probability that a diseased agent will die (0-1).
3) **Prob-transmission**: the probability that an agent within the transmission radius will become infected (0-1)
4) **Prob-recovery:** the probability that an agent will recover from the disease after receiving care (help), coded as 0-1

5) **Num-matrilines:** the number of unrelated agents created at set-up.
6) **Initial-prevalence:** the number of agents who are randomly infected in the fifth time step

Agents have the following state variables:

1) **Disease?**: a true/false variable determining the agent's disease status
2) **Intelligence**: the probability that an agent will correct identify the disease status of another agent (0-1)

Links represent relatedness between two agents. Links have one variable, r, which represents the matrilineal relatedness between the linked agents. Links representing parent-offspring relationships (r = 0.5) are colored white. Links representing matrilineal siblings/grandparents (r = 0.25) are colored red.

Simulations last for 100 time steps. Agents reproduce at the beginning of each time step, but because no maximum life span is set, the time steps do not translate directly into generations or years.

Process overview and scheduling

Each time step, the following sequences occur:

1) The model initializes by setting a list of global tracking variables to 0 or false [see submodel *initialize* for details].

2) The population repopulates at each time step when healthy agents reproduce [see submodel *repopulate* for details].
3) Each agent's links are reduced to only links with an r greater than or equal to 0.25. Links with r=0.5 and r=0.25 are white and red, respectively.
4) The model checks whether it is running time step 5. If so, a number of agents equal to the value of *initial-prevalence* are randomly infected with the disease. Those agents change their color to be 3 shades darker. If the current time step is not the fifth, this procedure is skipped.
5) Agents evaluate the disease status of nearby agents with an accuracy that is based on their intelligence score. Each agent maintains a list of the other agents it believes to be its' diseased kin [variable: diseased-kin, see submodel *assess-neighbors2* for details].
6) The model updates the values for the global tracking variables: total-turtles and total-disease. (Note: The program language refers to agents as "turtles," thus the variables "total-turtles" is the total number of agents.)
7) Healthy agents randomly select an agent they believe to be diseased kin (from variable: diseased-kin) and decide whether or not to provide care based on a modification of Hamilton's rule of inclusive fitness (Hamilton 1964). See submodel *help* for details.
8) The model updates following global tracking variables: total-helped, total-correct-helped, total-incorrect-helped.
9) The model generates a random number for each diseased agent. If that number is below the probability of the disease being fatal, that agent dies.

10) Healthy agents who are near diseased agents become infected according to the probability of transmission [see submodel *infect* for details].
11) The model outputs the following values for the current time step: *total-turtles, total-diseased,* and population average for *intelligence.* If the number of time steps is greater than four, the model also outputs, *total-correct-helped.*

Design concepts

Emergence: Over time, because higher intelligence individuals will direct their care-giving more accurately to kin who are actually diseased, higher intelligence matrilines reproduce faster than lower intelligence matrilines. Higher average population intelligence emerges.

Adaptive behavior: Agents receive an intelligence value based on that of their parent. They do not adapt over their lifetimes.

Objectives: Agents' objective is to maximize their own fitness by either providing care to or avoiding diseased kin. They decide what alternative to perform based on a modification of Hamilton's rule of inclusive fitness [see submodel *help*].

Learning: Agents do not learn from their mistakes.

Prediction: Agents explicitly calculate the potential costs and benefits when deciding whether to give care or avoid ill kin based on Hamilton's rule [see submodel *help*].

Sensing: Agents know their own disease status, the disease characteristics (probability of fatality, probability of transmission, and probability of recovery after care), and their relatedness to all other agents (link variable: r). The accuracy with which they sense the disease status of their kin is based on their intelligence score (which they do not sense). Agents do not sense when they make mistakes.

Interaction: Individuals interact directly by infecting and providing care to others. They also interact indirectly because when they provide care to a sick individual who recovers, they reduce the danger of infection for all other agents within the infection radius of that individual.

Stochasticity: Disease parameters are represented as likelihoods in order to incorporate the uncertainty of disease transmission and mortality.

Collectives: Matrilines are collectives of agents deriving from the same matriline (with a relatedness depth of $r \geq 0.25$). Agents less related than r=0.25 do not recognize each other as kin and will not provide care to each other.

Observation: For model testing, the following variables are output: *total-turtles* (total agents), *total-diseased*, *total-correct-helped* and population average for *intelligence*. The hypotheses stated in the purpose are tested by comparing these variables under diseases with different characteristics (probability of fatality and probability of transmission).

Initialization

The program is initialized in set-up with a number of agents equal to *num-matrilines.* Agents are randomly placed on the grid. Each of these agents is randomly assigned an intelligence value ranging from 0 to 1. The carrying capacity of the landscape is set at the value of the variable *carrying-capacity*.

Input

The user does not need to input additional files.

Submodels

Initialize: The model initializes by having a set of global tracking variables set to 0/false [see submodel *initialize* for details]. In addition each agent sets several of its' own tracking variables to zero. These variables are used later to calculate and store values that will be output at the end of each time step.

Global tracking variables:

a. **Total-turtles**: total number of agents on the landscape (referred to as turtles in Netlogo's programming language)
b. **Total-disease**: total number of agents who are diseased
c. **Total-helped**: total number of agents that have received care *in the current time step*
d. **Total-correct-helped**: total number of agents who received care *in the current time step* who were in fact diseased

Agent tracking variables:

a. **Helped?:** a true/false variable indicating whether the agent has received care *in the current time step*
b. **Correct-helped?:** a true/false variable indicating whether the agent has received care when it was diseased *in the current time step*
c. **Diseased-kin**: a set of agents that the current agent believes to be its diseased kin *in the current time step*

Repopulate: The population grows at each time step of the model when healthy agents reproduce according to the formula: [(1 - (number of agents / carrying-capacity)) * number of healthy agents]. Reproduction occurs asexually. Offspring are placed within a radius of 3 of the parent. Each offspring's intelligence is drawn from a normal distribution with the parent's intelligence as the mean and a standard deviation of 0.15. Matrilineal relatedness is tracked by links between agents with the links containing the relatedness value. Parent-offspring relationships receive relatedness values of 0.5 and offspring inherit the links of the parent but with ½ the relatedness value. Patrilineal relatedness is not included in the model.

Assess-neighbors2: All healthy agents evaluate the relatedness and disease status of other agents within a radius equivalent to 5 grid cells. Kin are accurately recognized and the accuracy of disease recognition is a function of the agent's intelligence. A random number between 0-1 is drawn. If the number is below the agent's intelligence value, the disease status is correctly recognized. Otherwise, the

agent's disease status is incorrectly recognized (healthy kin are classified as diseased or diseased kin are classified as healthy). These individuals make up the group the agent *believes* are its diseased kin (variable: *diseased-kin*).

Help: Healthy agents randomly select an agent they believe to be diseased kin (variable: *diseased-kin*) and decide whether or not to provide care based on a modification of Hamilton's rule of inclusive fitness which predicts altruism when the relatedness between the recipient and altruist * benefit to the recipient > the cost to the altruist (Hamilton 1964). We adapted this formula so that agents provide care when the relatedness between the care-giver and the recipient * probability of recovery after care > the probability of transmission to the care-giver * probability of an infection being fatal. If the inequality is fulfilled (thus care is given) and the recipient was in fact diseased (not just *perceived* to be diseased), a random number between 0 and 1 is generated and if it is below the probability of recovery, the diseased individual recovers. If the random number was above the probability of recovery, the recipient remains diseased. A new random number is drawn for the care-giver and if it is below the probability of transmission to the care-giver, then the care-giver is infected. If the recipient was erroneously categorized as diseased, but is actually healthy, there is no change in the disease statuses of the recipient or the care-giver. If healthy agents have no perceived diseased kin or the randomly selected recipient does not fulfill the inequality for receiving care, the agent can attempt to avoid the diseased agent by moving to a grid cell with no other agents on it within a radius of 8. If no empty cells are available, the agent does nothing.

Infect: All healthy agents have a probability of becoming infected from any infected agent within a radius of 5 grid cells, based on the probability of transmission. A random number between 0 and 1 is drawn for each of the healthy agents in danger of infection. If the number is below the probability of transmission, the agent is infected. If an agent is in danger of infection from more than one diseased agent, the process is repeated for each infectious agent in the 5 grid cell radius.

Model implementation

Note that the model contains the submodel *Avoid*, but that *Avoid* is commented out. In the Avoidance Model (Model 2) the submodel *Help* is replaced by *Avoid.*

The model is implemented in Netlogo 5.0 and can be run using the buttons on the interface or through the BehaviorSpace tool.

If run through the interface buttons, the model continues beyond 100 time steps.

If run in BehaviorSpace, enter 1 for the number of runs to be conducted in parallel (BehaviorSpace/Run Options window). This will prevent data from data from multiple runs being intermixed in the output files.

References

1442 Hamilton WD (1964) The genetical evolution of social behavior. I and II. J Theor Biol 7:1-52

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Appendix B. **ODD Protocol for the Model 2: Control model – Avoidance only**

Purpose

The purpose of this model is to test 1) whether an avoidance response to diseased conspecifics can select for increasing intelligence and 2) whether the strength of selection varies according to the disease's characteristics. We compare the selection produced by diseases with fatality rates similar to Ebola, Crimean-Congo hemorrhagic fever, measles, and scabies. The data produced by this model will be used for comparison with the data produced by Model 1.

Entities, state variables and scales

Same as Model 1

Process overview and scheduling

Same as Model 1, except for number 7.

7) Healthy agents run the submodel *avoid.* If they have agents they believe to be diseased kin and there are empty patches within a radius of 8, the agent randomly selects and moves to one of those patches. See submodel *avoid* for details.

Design concepts

Same as Model 1 unless discussed below:

Emergence: Agents do not provide care, so unlike model 1, higher intelligence is not expected to emerge.

Objectives: Agents' objectives are to maximize their own fitness by avoiding diseased kin. [see submodel *avoid*].

Prediction: Agents do not calculate the potential costs and benefits when deciding whether to avoid ill kin. All kin perceived as ill are avoided. [see submodel *avoid*].

Interaction: Individuals interact directly by infecting others. They also interact indirectly because when avoiding ill kin, they occupy an open patch which reduces the number of open patches available for other agents to move to.

Observation: For model testing, the following variables are output: *total-turtles* (total agents), *total-diseased*, *total-correct-helped* and population average for *intelligence*. The hypotheses stated in the purpose are tested by comparing these variables across models (model 1 vs. model 2) within each disease.

Initialization

Same as Model 1

Input

Same as Model 1

Submodels

Same as Model 1 unless described below

Help: The submodel *help* does not run in Model 2. It is replaced by the submodel *avoid.*

Avoid: Healthy agents assess whether they have diseased kin (kin they **believe** to be diseased). Agents who have none exit the submodel. Agent who have diseased kin assess whether there are any patches without agents within a radius of 8. If there are, the agent randomly selects one of those patches and moves to it.

Model implementation

Same as Model 1

References

Hamilton WD (1964) The genetical evolution of social behavior. I and II. J Theor Biol 7:1-52