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

68 **Introduction**

69 Exposure to disease is a major cost of sociality (McCabe et al. 2015; Nunn and Altizer
70 2006; Rifkin et al. 2012). Despite this, hominins have evolved extraordinary social
71 complexity (Tomasello 2014), including a strikingly social way of mitigating the effects of
72 socially transmitted diseases—we provide care to diseased individuals. Such care hinges on
73 the ability to recognize disease in others. Currently, the cognitive basis of this ability is not
74 well understood. In this paper, we present the novel hypothesis that the ability to recognize
75 disease may have evolved together with social cognition in hominins.

76 A synthesis of paleoanthropological, ethnographic, and host-parasite research suggests
77 that increasing social complexity during the origin of *Homo* dramatically increased disease
78 risk, i.e., (Harper and Armelagos 2013; McCabe et al. 2015; Rifkin et al. 2012; Sugiyama
79 2004). Thus, part of the selection for increasing cognitive abilities in *Homo* may have been
80 selection to accurately assess the disease risk presented by interaction partners. We integrate
81 findings from the literature on hominin social structure, hominin disease ecology, disease
82 recognition in nonhuman animals, and human social cognition. Based on these data, we
83 create an agent-based model to examine under what conditions increased cognition and care-
84 giving could have evolved in the hominin lineage. Using our results, we create predictions
85 deriving from our novel disease recognition hypothesis of hominin cognitive evolution that
86 can be tested by paleoanthropologists, paleogenticists, bioarchaeologists, and primatologists.
87

88 **Broadening social networks between hominin subgroups**

89 Across birds and mammals, larger communities show greater levels of contagious
90 parasites, environmentally transmitted parasites, and vector-borne parasites (Rifkin et al.
91 2012). Though network modularity (sub-grouping) may reduce the transmission risks in large
92 communities where many dyads do not interact (Griffin and Nunn 2012), hominin networks

93 appear to have connected spatially distant subgroups, facilitating transmission within a
94 fission-fusion, multi-level society (Grove et al. 2012; Hill et al. 2011).
95 Hominin community sizes have been reconstructed as having expanded over time, from
96 ~50 in apes and small-brained australopiths to 100-120 in late *H. erectus* and *H.*
97 *heidelbergensis* to 120-150 in *H. neandertalensis* and *H. sapiens* (Aiello and Dunbar 1993;
98 Dunbar 1998; Gamble et al. 2011; Grove et al. 2012; Layton et al. 2012). This is believed to
99 have produced an increase, not only in social network size, but also in complexity (Grove et
100 al. 2012). As hominins dispersed towards northern latitudes and community sizes increased,
101 the home-range requirements for sustaining them would have also increased (Grove et al.
102 2012). This produced communities whose daily nutritional needs were too large to be
103 fulfilled in the amount of space a cohesive group could cover each day (Grove et al. 2012).
104 The result is thought to have been the evolution of a multi-level fission-fusion system in
105 which larger communities subdivide, rather than foraging cohesively (Grove et al. 2012).
106 This would have enabled large communities of hominins to forage across greater areas and
107 expand into new habitats, yet still obtain the benefits of a large social network, such as
108 information transfer, social learning, and cooperation (Grove et al. 2012; Layton et al. 2012).
109 Thus, even though mean population density decreased over time as hominins dispersed
110 northward, overall community size and social network size likely increased (Grove et al.
111 2012; Layton et al. 2012).
112 Community size estimates for modern hunter-gatherers range from 125 to a few thousand
113 (Layton et al. 2012). The extensiveness of human social networks was documented in a study
114 showing that while chimpanzee males typically only interact with about 20 other males, a
115 modern male hunter-gather may watch over 300 other men make tools (Hill et al. 2014). The
116 evolution of such long-distance social networks linking different subgroups (Hill et al. 2014)
117 may have prevented the reduction in disease risk that might otherwise be expected to have

118 occurred as hominin density decreased, i.e., (Armelagos et al. 2005). Hominins' extensive,
119 community-wide social networks would have facilitated widespread pathogen transmission,
120 including any novel pathogens acquired as hominins spread into new habitats (McCabe et al.
121 2015).
122
123 **Increasing connectedness within groups**
124 Simultaneously with the expansion of networks connecting subgroups, the complexity of
125 networks within the subgroups also likely increased with the evolution of cooperative
126 breeding during the origin of *Homo*. Early *Homo* fossil assemblages show an increased
127 number of immature relative to mature individuals compared to australopith assemblages
128 (Tobias 2006), suggesting shortened interbirth intervals, increasing energetic demands on
129 reproducing females, and a shift towards cooperative breeding (Aiello and Key 2002).
130 Ethnographic work supports this view of humans as cooperative breeders, revealing greatly
131 expanded social networks that include multiple providers (hunting males, post-reproductive
132 females) for females and young (Hawkes 2003; Hill et al. 2009; Hrdy 2009). This contrasts
133 with chimpanzees in which the young are solely dependent upon their mothers (Burkart et al.
134 2009). Collectively, these studies suggest that as community size increased during the origin
135 of *Homo,* so did the complexity of the social networks linking both greater numbers of
136 individuals and different demographics (e.g., young dependents, post-reproductive females,
137 hunting males). The close cooperation, interdependence, and density of social networks
138 within cooperatively breeding hominin groups would have facilitated the spread of diseases
139 within these groups (McCabe et al. 2015).
140
141 **Hominin Disease Ecology**

142 The shift to larger networks linking subgroups within a larger community and greater
143 connectedness within cooperatively breeding groups is believed to have selected for
144 enhanced social cognition (e.g., prosociality, shared-intentionality, theory of mind) which
145 facilitated prolonged, close interactions among individuals and promoted social learning,
146 cooperation, technological advances and cumulative culture (Burkart et al. 2014; Byrne and
147 Bates 2007; Herrmann et al. 2007; Tomasello et al. 2005; van Schaik et al. 2012; Whiten
148 2000). However, such intense, close proximity interactions would have also facilitated
149 disease transmission (McCabe et al. 2015). Recent work in genetics and evolutionary
150 medicine indicates that hominins harbored numerous pathogens before the advent of
151 agriculture and animal domestication (Harper and Armelagos 2013). This includes
152 endoparasitic worms (Hoberg et al. 2001; Hurtado et al. 2008), lice (Harper and Armelagos
153 2013), tuberculosis (Stone et al. 2009), typhoid fever (Harper and Armelagos 2013),
154 whooping cough (Harper and Armelagos 2013), and viruses, e.g., herpes viruses, Epstein
155 Barr virus (Harper and Armelagos 2013). Thus, hominins were likely under strong selection
156 to assess the disease status of others.
157
158 **Disease recognition in animals and humans**
159 Comparative evidence suggests that disease recognition may have been present in early
160 hominins (citations below). Several species with relatively low social complexity have been
161 documented to recognize disease, often either avoiding diseased conspecifics or taking
162 advantage of sick and weakened competitors, e.g., social lobsters (Behringer et al. 2006),
163 pipefish (Rosenqvist and Johansson 1995), bullfrog tadpoles (Kiesecker et al. 1999), rodents
164 (Kavaliers et al. 1997), house finches (Bouwman and Hawley 2010; Zylberberg et al. 2012),
165 but see (Nunn 2003). While the underlying cognitive processes are not well understood, these
166 studies suggest that recognition is based on diverse symptoms including olfactory/chemical

cues (Kavaliers et al. 1997; Kiesecker et al. 1999), visual detection of spots (Rosenqvist and Johansson 1995), and behavioral changes including lethargy and feather fluffing (Bouwman and Hawley 2010; Zylberberg et al. 2012). Though the amount of cognitive processing required to detect disease may differ by symptom type, the wide array of cues and recognition in multiple species suggests that some simple form of disease recognition could have been basal in hominins.

Infectious pathogens can cause noticeable symptoms that could potentially be detected via the perceptual-cognitive pathways that are integral to social cognition in primates. Subtle differences perceived in conspecific faces (Leopold and Rhodes 2010; Sartori et al. 2011), voices (Belin 2006; Belin et al. 2004), and movement/gait (Loula et al. 2005; Peterman et al. 2014; Sartori et al. 2011) may enable, not only the decoding of conspecifics' identities, emotions, and intentions, but also facilitate the detection of disease. This could include changes in facial coloration and texture due to fever, rashes, or nasal discharge, changes in vocalizations due to coughing, nasal discharge or reduced lung capacity, and changes in movement/gait due to weakness, lethargy, or signs of pain (Chapman et al. 2005; Fink and Matts 2008; Hart 1988). Thus, if the detection of social information and disease involve the same perceptual-cognitive pathways, then disease circulating within hominin populations may have selected for increased 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

192 causation (Sherman 1988; Tinbergen 1963), favoring individuals who were able to
193 discriminate who was healthy and who was not. Those who avoided infectious individuals or
194 provided care to ill kin would increase their reproductive fitness. Similarly to how kin
195 recognition can operate without individuals having an abstract concept of kin (Rendall 2004),
196 disease recognition could operate without a concept of disease.
197
198 **Care-giving among animals and humans**
199 The literature contains numerous reports of striking cases of social care given by animals,
200 including dolphins that cooperatively supported a dying conspecific who could no longer
201 swim (Park et al. 2013), an elephant that attempted to lift a collapsed and dying conspecific to
202 her feet (Douglas-Hamilton et al. 2006), primates that groom, stand watch over, and/or chase
203 others away from dying group members (Anderson et al. 2010; Bezerra et al. 2014;
204 Nakamichi et al. 1996), and an otter group that provisioned an elderly female (Davenport
205 2010). Though very interesting, these reports do not provide evidence of widespread long-
206 term care which would be expected to have a more significant selective influence on a
207 species' evolution.
208 Some of the best opportunities for systematically investigating care-giving in animals
209 have come from studies of populations with high prevalences of severe injuries (Beamish and
210 O'Riain 2014; Byrne and Stokes 2002; Stokes and Byrne 2006) or congenital disabilities
211 (Turner et al. 2014). These studies generally suggest that, instead of relying on social care,
212 severely injured or disabled individuals survive by adapting and making adjustments
213 themselves, rather than receiving accommodation or assistance (Beamish and O'Riain 2014;
214 Byrne and Stokes 2002; Stokes and Byrne 2006; Turner et al. 2014). The exception to this is
215 social grooming (Dittus and Ratnayeke 1989). Wound cleaning has been shown to be an
216 important mechanism for avoiding infections and it is widespread in animals (Dittus and

217 Ratnayeke 1989; Hart 2011). Thus wound cleaning may have been a basal form of social care
218 in hominins.

219 In addition, evidence from modern foraging, hunting, and horticultural peoples, suggests
220 that provisioning people who are ill or injured is important in reducing the mortality rate
221 (Sugiyama 2004). For example, Sugiyama (2004) found that over 50% of individuals
222 reported at least one time in their lives when they were incapacitated and could not forage for
223 at least a month. During such times, provisioning was critical to their survival (Sugiyama
224 2004). Based on this evidence, we expect that hominins could have significantly reduced the
225 mortality arising from disease and infection-related injuries through provisioning (Sugiyama
226 2004) and wound cleaning (Dittus and Ratnayeke 1989). Additionally, food sharing networks
227 of hunting males also served as provisioning networks during times of illness (Gurven et al.
228 2000; Sugiyama 2004; Sugiyama and Chacon 2000), suggesting that the evolution of social
229 care may have co-evolved with cooperative breeding.

230

231 **Care-giving in the fossil record**

232 Fossil evidence of hominins surviving illness, injuries, and disabilities goes back nearly 2
233 million years to include fossils from *H. erectus, H. heidelbergensis, H. neandertalensis,* and
234 *H. sapiens*. While the following discussion is not exhaustive, it does illustrate the variety of
235 conditions hominins survived, the time depth of the fossil record, and the taxa included.
236 Below we follow, when possible, the taxonomic classifications provided in Grove et al.
237 (2012). In *H. erectus* this includes: premortem loss of all but one tooth in the 1.77 mya
238 cranium and mandible from Dmanisi (D3444 and D3900 (Lordkipanidze et al. 2005;
239 Lordkipanidze et al. 2006)), possible hypervitaminosis A in the 1.6 mya KNM-ER 1808
240 (Walker et al. 1982), evidence of a herniated disc in the 1.5-1.6 mya Nariokotome boy KNM-
241 WT 15000 (Grove et al. 2012; Haeusler et al. 2013; Schiess et al. 2014), and a healed cranial

lesion caused by trauma or burning in the 0.6 mya Hulu 1 cranuim, also called Nanjing 1 and Tangshan 1 (Shang and Trinkaus 2008; Wu et al. 2011). Among *H. heidelbergensis* this includes craniosynostosis and neurocranial deformities in a 0.53 mya immature, cranium 14, who survived for at least approximately 5 years (Gracia et al. 2009), a 0.53 mya adult male pelvis and lumbar spine, SH Pelvis 1, showing lesions and degeneration possibly resulting from lumbar kyphotic deformity, spondylolisthesis, and Baastrup disease (Bonmati et al. 2010), and a squamous temporal lesion that shows healing on the 0.35 mya Broken Hill cranium Kabwe 1 (Grove et al. 2012; McBrearty and Brooks 2000; Montgomery et al. 1994). For Neandertals this includes Aubesier 11, dated to at least 0.17 mya, which shows significant tooth loss and alveolar lesions (Lebel and Trinkaus 2002; Lebel et al. 2001) and Shanidar 1 dated at 73-40 kya who lost much of his right arm, may have been blind on one side, and suffered from hyperostotic disease (Crubezy and Trinkaus 1992; Hublin 2009). *H. sapiens* individuals that survived severe conditions include: a child, 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

292 powerful, quantitative insights into disease transmission, including predicting the impact of
293 current/future outbreaks and planning intervention/prevention strategies, e.g., influenza (Guo
294 et al. 2015), Ebola (Merler et al. 2015). We take the innovative approach of applying these
295 techniques to reconstruct the potential impact of disease on hominin evolution.
296 A modeling approach is valuable because, while our knowledge is increasing, i.e.,(Harper
297 and Armelagos 2013), we do not have sufficiently detailed data concerning how/when
298 disease load changed during hominin evolution to be able to test whether the evolution of
299 care-giving co-occurred with increasing cognitive abilities, social complexity and disease
300 risk. Therefore, we use agent-based modeling to examine under which conditions disease
301 could select for increased cognition and care-giving. We hypothesize that 1) disease will
302 produce care-giving among kin and an increase in average population intelligence, that 2)
303 varying disease characteristics will produce variation in the strength of selection, and that 3)
304 care-giving will produce greater selection for cognition than an avoidance strategy.
305

306 **Material and methods**

307 *Study design*

308 We created two models for comparison. The first (Model 1: Care-giving model) simulates
309 disease transmission in a population of hominins who give care (The ODD description is in
310 Appendix A at the end of the paper. The code is available in supplementary file 1). In order to
311 more fully explore the model and how care-giving may alter the progression of disease
312 through the population, we then created a control model (Model 2: Avoidance only) similar
313 to the first except that agents avoid diseased kin and provide no care. (The ODD description
314 is in Appendix B at the end of the paper. The code is available in supplementary file 2).
315

316 ***Model 1: Care-giving model***

317 *Disease characteristics*

318 We programmed an SIS model (susceptible – infected – susceptible) in Netlogo 5.0.5
319 (Railsback and Grimm 2011; Wilensky 1999). We created four hypothetical diseases with
320 case fatality rates modeled after Ebola [2014 outbreak: 70% (Aylward et al. 2014; WHO
321 2014a), Crimean-Congo hemorrhagic fever (40% (WHO 2013), CCHF, hereafter), measles
322 (~10% (WHO 2014b)), and a low risk comparison, such as scabies (fatality rate set at 1%,
323 though scabies is generally not fatal (WHO 2015)]. We did not attempt to precisely simulate
324 the natural history of these diseases. Rather, these diseases were chosen to represent a range
325 of fatality rates occurring in socially transmitted diseases.

326

327 *Optimizing the disease transmission rates*

328 Because transmission rates have complex relationships with virulence and host density
329 (e.g., trade-off hypothesis (Alizon et al. 2009)), we screened possible transmission rates to
330 determine what would be optimal for persistence of these diseases in this population. For the
331 Ebola-like, CCHF-like, and measles-like diseases, we ran the model 1000 times in Netlogo's
332 BehaviorSpace, varying the probability of transmission from 10-100% by increments of 10.
333 For the scabies-like disease, we ran the model 1000 times varying the probability of
334 transmission from 1% to 98.5% by increments of 2.5. The inclusion of lower transmission
335 values for the scabies-like disease is based on literature showing that less virulent diseases
336 tend propagate slower, e.g., (Alizon et al. 2009; Ewald 1993). Then, for each disease, we
337 selected the runs which had both healthy and diseased individuals after 100 time steps. We
338 averaged the probability of transmission across those successful runs to obtain a transmission
339 rate that is optimal for each respective disease: Ebola-like 78%, CCHF-like 33%, measles-
340 like 10%, scabies-like 2%. The higher transmission rates in the diseases with higher fatality

rates is consistent with the relationship between virulence and transmission documented in the literature (Alizon et al. 2009).

Determining the probability of recovery after care

We expect that the earliest forms of social care given by hominins would have been 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

366 summarizes the group sizes, densities, and space requirements presented in Grove et al.
367 (2012).

368 **[Table 1]**

369 The carrying capacity of the landscape is set at 200. Two hundred was chosen because it
370 is large enough to encompass the group sizes predicted for hominins based on cranial
371 capacities, brain volumes, and neocortex ratios of fossil hominins [Table 1, (Aiello and
372 Dunbar 1993; Gamble et al. 2011; Grove et al. 2012)], but is generally smaller than
373 community sizes reported for modern humans, e.g., (Hill et al. 2014; Layton et al. 2012). We
374 set the carrying capacity above the calculated community sizes for hominins, e.g., ~150 or
375 smaller (Aiello and Dunbar 1993; Dunbar 1998; Gamble et al. 2011; Grove et al. 2012), to
376 allow for the event that the actual community sizes of the model populations would likely be
377 lower than the carrying capacity.

378

379 *Initialization*

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

389

390 *Population growth and genetic structure*

391 The population grows at each time step of the model when healthy agents reproduce
392 according to the formula: [(1 - (number of agents / carrying-capacity)) * number of healthy
393 agents]. Reproduction occurs asexually. Offspring are placed within a radius of 3 of the
394 parent, producing spatial clustering of kin as is consistent with human and nonhuman primate
395 groups (Chapais and Berman 2004; Hatchwell 2010; Hill et al. 2011; Silk 2009).
396 Relatedness is tracked by links between agents with the links containing the relatedness
397 value. Parent-offspring relationships receive relatedness values of 0.5 and offspring inherit
398 the links of the parent but with ½ the relatedness value. Because offspring inherit the links of
399 the parent, sibling relationships are included in the model with a relatedness value 0.25. To
400 prevent the model from becoming too computationally intensive, patrilineal relationships and
401 matrilineal relationships beyond a relatedness of 0.25 were not modeled. This decision is
402 supported by findings showing that kin recognition occurs most reliably for close matrilineal
403 kin identified via familiarity, e.g., (Chapais and Berman 2004; Chapais et al. 1997). The
404 population represents a single, kin structured community with multiple matrilines. Space
405 displays the contact structure between agents and random movement simulates mixing within
406 the population.
407
408 *Space*
409 With a carrying capacity of 200 individuals and a landscape of 200 km^2, our model has a
410 maximum population density of 1 individual / km^2, which is within the confidence intervals
411 calculated for *H. habilis* and *H. erectus* [Table 1, (Grove et al. 2012)]. However, the purpose
412 of our model is not to attempt to reconstruct a particular hominin species or population. We
413 made this decision because the population densities and number of levels of fissioning have
414 been reconstructed to vary dramatically even within species, depending upon the habitat
415 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

441 the disease status of the kin agent. Thus, the likelihoods of false positive and false negative
442 errors are functions of disease prevalence. As the proportion of diseased agents increases,
443 false positives decrease and false negatives increase.
444 Agents randomly select one of their perceived diseased kin and decide whether to provide
445 care based on a modification of Hamilton's rule, which predicts altruism when: (relatedness
446 between the recipient and altruist)*(benefit to the recipient)>(cost to the altruist) (Hamilton
447 1964). We adapted this formula so that agents provide care when: (relatedness between the
448 care-giver and the recipient)*(probability of recovery after care) > (probability of
449 transmission to care-giver)*(probability of infection being fatal). If the inequality is fulfilled
450 (thus care is given) and the recipient was in fact diseased (not just *perceived* to be diseased), a
451 random number between 0 and 1 is generated and if it is below the probability of recovery,
452 the diseased individual recovers. If the random number was above the probability of
453 recovery, the recipient remains diseased. A new random number is drawn for the care-giver
454 and if it is below the probability of transmission to the care-giver, then the care-giver is
455 infected. If the recipient was erroneously categorized as diseased, but is actually healthy (a
456 false positive error), there is no change in the disease statuses of the recipient or the care-
457 giver. It is worth noting that when a false negative error occurs (diseased kin are classified as
458 healthy), the agent that made the error does not incur a cost that is explicitly coded into the
459 model. However, the agent does potentially incur emergent costs through the interactions
460 between agents. This may occur in two ways: a) if that diseased kin agent dies (later in the
461 model run), this reduces the kin network available to give care, simulating a loss of inclusive
462 fitness to the agent that failed to recognize the disease in its kin, and b) the presence of
463 diseased kin in the population increases the risk that others will become infected, including
464 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

515 change in average population intelligence between the first and 100th time step, tested
516 whether the differences were different from zero using one-sample T-tests, and whether these
517 differences varied across disease types using a one-way ANOVA. We calculated maximum
518 slopes for the curves of the average percent care and the average population intelligence
519 using grofit (Kahm et al. 2010) in R 2.13.1 (RCoreTeam 2011) and RStudio 0.98.1062
520 (RStudio 2014). We tested whether the slopes differed across disease types using a one-way
521 ANOVA. Some violations of normality and equal variances existed (Supplementary files 3
522 and 4). One-way t-tests were bootstrapped with 1000 samples for robusticity to non-
523 normality and 95% bias corrected accelerated confidence intervals were calculated (Field
524 2013). Though one-way ANOVAs are generally robust to such violations when groups have
525 equal sample sizes, when variances were unequal, we used the Brown-Forsythe F-ratio.
526 Alpha was set at 0.05 and multiple comparisons across disease types were Bonferroni
527 corrected when variances were equal and Tamhane T2 corrected when they were unequal.
528 Statistical tests were run in SPSS Statistics 22 or 23 unless otherwise stated.
529
530 ***Model 2: Control model – Avoidance only***
531 Following the initial analysis of the care-giving model (Model 1), we programmed a
532 control (Model 2: Avoidance only) to further explore how care-giving may have altered the
533 progression of disease through hominin populations. This model used the same population
534 and diseases, but differed in two ways. First, agents who have perceived diseased kin avoid
535 them instead of providing care. All agents with perceived diseased kin move randomly to an
536 empty grid cell within a radius of 8. Second, if the agent has no perceived diseased kin or
537 there are no empty grid cells within a radius of 8, the agent does not move. This differs from
538 the care-giving model in which agents with no diseased kin also move to an empty grid cell
539 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:

565 prevalence * population size. As the data are longitudinal (i.e., time series) we allow for
566 autocorrelated errors using an ARMA process, incorporate time as a fixed effect, and use the
567 averaged simulation run as the random effect. We check for issues of multicollinearity using
568 variation inflation factor, and check the residuals of the models for non-normality,
569 heteroscedasticity, and autocorrelation. (model: change in mean intelligence ~ time +
570 prevalence*population size + random intercept). In order to keep the paper focused on the
571 evolution of increasing average population intelligence, we did not conduct this analysis on
572 the Model 2 curves, which showed either no increase or a decrease in average intelligence.
573

574 **Results**

575 *Model 1: Care-giving model*

576 After 100 time steps the four diseases produced significantly different population sizes,
577 disease prevalence, percentages of the diseased who received care, and average population
578 intelligences (Tables 2-3, Fig. 1).
579

580 **[Table 2]**

581 **[Table 3]**

582 **[Figure 1]**
583

584 The Ebola-like disease, unlike the other three, produced no care-giving and no change in
585 average population intelligence (Table 4, Fig. 1).
586

587 **[Table 4]**
588

589 Both the Crimean-Congo hemorrhagic fever-like (CCHF-like) and measles-like diseases
590 show initial increases in both care-giving and intelligence followed by a plateau (Fig. 1). The
591 CCHF-like disease produced a care-giving rate of 4.7%, a final intelligence level of 0.62, and
592 a 12% net change in intelligence. Of the four diseases, the measles-like disease produced the
593 highest rate of care-giving (6.7%) and the highest average population intelligence (0.71) at
594 the final time step. This was generated by the greatest maximum slopes for care-giving and
595 intelligence changes and the greatest net change in intelligence over time (21%). The scabies-
596 like disease showed a strikingly different pattern. As prevalence steadily increased, because
597 the fatality rate was low, care-giving decreased. Infected individuals did not provide care and
598 rarely died, meaning that the number of healthy individuals able to provide care decreased.
599 This produced a negative slope for care-giving, though low increases in average population
600 intelligence were still observed (care-giving rate: 1.4%, final average population intelligence:
601 0.53, net intelligence change: 3%, Tables 2-3).
602
603 *Model 2: Control model – Avoidance only*
604 The model two results revealed two important findings. First, an avoidance strategy did
605 not result in an increase in average population intelligence (Tables 5 and 6). The net change
606 in intelligence overtime was not significantly different from zero under the scabies-like and
607 measles-like conditions (Table 5). Under the CCHF-like and Ebola-like conditions the
608 average population intelligence decreased significantly (Table 5).
609 **[Table 5]**
610 **[Table 6]**
611 Second, a visual inspection of Figures 2-4 shows that the progression of the diseases
612 through the population differed under Model 1 (care-giving) and Model 2 (avoidance only).
613 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

639 the variation explained by the analysis (marginal $R^2 = 0.15$) was reduced compared to the
640 measles-like (marginal $R^2 = 0.57$) and scabies-like (marginal $R^2 = 0.47$) diseases. However,
641 similar to the measles-like disease, an interaction effect between prevalence and population
642 size was present, indicating that at low prevalences, changes in intelligence were negatively
643 related to population size, but at higher prevalences, they were positively related with
644 population size (Fig. 7). Thus the greatest changes in intelligence occurred at low prevalences
645 and low population sizes or high prevalences and high population sizes.

646 No relationships between time, prevalence or population size were found for the Ebola-
647 like disease because the Ebola-like disease produced no changes in intelligence (Tables 5 and
648 7, Fig. 8).

649 **[Table 7]**

650 **[Fig. 5]**

651 **[Fig. 6]**

652 **[Fig. 7]**

653 **[Fig. 8]**

654

655 **Discussion**

656 *General discussion*

657 Our findings suggest that the evolution of care-giving may have created a profound
658 shift in how hominins evolved in the presence of their pathogens. The avoidance approach
659 (Model 2) likely represents the basal condition, under which disease either does not select for
660 or against increasing cognitive abilities (high prevalence, low fatality diseases) or selects
661 against it (low prevalence, high fatality diseases). In contrast, under the care-giving condition
662 (Model 1), care-giving not only selected for increasing cognitive abilities, but also altered and

663 controlled the progression of some of the diseases throughout the population. We discuss
664 both models and their implications in detail below.
665
666 *Model 1*
667 Our results from Model 1 suggest that disease circulating among kin can select for care-
668 giving among kin and greater cognitive abilities. Furthermore, the diseases produced
669 selection of varying strengths, with higher care-giving rates producing greater increases in
670 average population intelligence.
671 The findings are relevant to the evolution of care-giving in hominins as they suggest that
672 not all diseases produce care-giving behavior. The high fatality and transmission rates of the
673 Ebola-like disease, when applied to Hamilton's rule (Hamilton 1964), generated costs that
674 were greater than the benefits of care-giving, even to close relatives, thus, all agents avoided
675 ill kin, rather than providing care. Such diseases are not likely to have facilitated the
676 evolution of care-giving or increased social cognition. The CCHF-like disease had
677 intermediate probabilities of fatality and transmission, leading to care-giving only to close kin
678 (parents and offspring: r=0.5), and not to more distant relatives like grandparents,
679 grandchildren, or siblings (r=0.25) who were avoided when ill. This produced substantial
680 care-giving behavior and selection for increasing intelligence, but the selection was weaker
681 than for the measles-like disease, where care was given to both close and more distant
682 relatives. The scabies-like disease, while it produced care-giving for both close and more
683 distant relatives, produced only low rates of care-giving and correspondingly weak selection
684 for increasing intelligence. These effects result from the very low fatality rate of the scabies-
685 like disease; the population size appears to have been regulated largely by the carrying
686 capacity set in the model (i.e., habitat supports 200 individuals) rather than by the disease.
687 Therefore, as disease prevalence increased, there was a lack of healthy individuals who could

provide care to their diseased kin, leading to a low rate of care-giving, lower population turnover, and lower increases in average population intelligence. Overall, these simulations suggest that diseases that are most likely to have led to the evolution of care-giving in the human lineage were those with low costs to caregivers which persisted at a prevalence low enough not to disrupt the kin networks along which care was provided. Although only healthy agents could give care and reproduce in our model, high rates of costly care-giving may not be expected if kin have sublethal diseases that do not reduce their reproductive success.

It is noteworthy that for all three diseases that produced care-giving, the final rate of care-giving was low, with a maximum of 6.7% of the diseased receiving care under measles-like conditions. Furthermore, a recovery rate of only 50% after care suggests that over the course of hominin evolution even low rates of relatively ineffective care may have been sufficient to select for increasing intelligence and disease recognition. We expect that the first forms of care-giving among hominins would have included assistance with hygiene, such as cleaning of wounds and topical infections (Dittus and Ratnayeke 1989) and provisioning with food and water (Sugiyama 2004). These mechanisms would not have required an understanding of disease processes and could have piggybacked on basal social grooming behaviors observed in nonhuman primates (Dittus and Ratnayeke 1989) and communal provisioning behaviors that may have evolved during the evolution of cooperative breeding (Burkart et al. 2009; Gurven et al. 2000; Hawkes 2003; Hill et al. 2009; Hrdy 2009; Sugiyama 2004; Sugiyama and Chacon 2000).

Model 2

The Model 2 results demonstrate that avoidance alone does not select for greater cognitive abilities. Avoidance produced no net change in average population intelligence in

713 the scabies-like and measles-like conditions and a *decrease* in average population intelligence
714 in for the CCHF-like and Ebola-like diseases. The scabies-like and measles-like diseases
715 produced higher population sizes and disease prevalences *above* 50%, thus an agent who
716 moves away from infected kin is likely to encounter other infected individuals. This results in
717 a lack of selection for disease recognition and avoidance. In contrast, the CCHF-like and
718 Ebola-like diseases produced lower population sizes and prevalences *below* 50%, thus an
719 agent who avoids infected kin is less likely to encounter other infected agents. This results in
720 selection to isolate oneself. The most efficient way for agents to isolate themselves in a
721 population with a prevalence under 50%, is to miscategorize healthy individuals as ill, thus
722 triggering avoidance. Because lower intelligence agents have less accurate disease
723 recognition, this produces selection to *decrease* intelligence.
724 These findings are relevant for species that do not give care. It suggests that avoidance of
725 high prevalence, low fatality diseases is likely to be an ineffective strategy. As a result these
726 diseases do not exert selection for or against cognitive abilities under an avoidance only
727 paradigm. In contrast, avoidance is an effective strategy against low prevalence, high fatality
728 diseases producing selection for avoidance behavior and selection against sociality.
729
730 *Implications of care-giving*
731 A comparison of the results from Model 1 (care-giving model) with Model 2 (avoidance
732 model) indicates that care-giving alters the progression of the disease through the population.
733 For the scabies-like and measles-like diseases, care-giving resulted in significantly higher
734 population sizes and lower prevalences than an avoidance only strategy. Thus for these
735 diseases, which are the two diseases for which care was given to both close and distant kin
736 (r=0.5 and r=0.25, respectively), care-giving served to control the disease in the population.

737 Two of the diseases, the measles-like and the CCHF-like diseases, show distinct cycles of
738 disease outbreaks and population crashes ("boom and bust" dynamic, Fig. 2-3). The lack of
739 congruence between the relatively constant slope of the intelligence curves (Fig. 4) and the
740 boom-bust oscillations of population size and prevalence, is a reflection of the fact that
741 selection on intelligence is occurring throughout the boom-bust cycle and not intermittently
742 only when specific conditions are met (e.g., a particular population size or prevalence). This
743 dynamic is quantified through the interaction term of the mixed model analysis in which
744 intelligence increases are the result of complex interactions between prevalence and
745 population size. Because the two diseases progress differently through the population, they
746 also exert selection on intelligence in slightly different ways. The measles-like disease
747 produces one oscillation of the boom-bust outbreak cycle of population and prevalence peaks
748 and crashes; the CCHF-like disease produces multiple, more rapid oscillations.
749 The measles-like disease shows a very pronounced "bust" phase early in the run.
750 Population size is high when the disease is first introduced (Fig. 2B, Model 1 curve). This
751 produces a high rate of care-giving and strong selection for intelligence (left panel, Fig. 6B).
752 As the prevalence increases (Fig. 3B, Model 1 curve), low intelligence matrilines recognize
753 diseased kin less accurately, and provide less successful care, causing them to succumb to the
754 disease. This produces a decrease in population size and an increase in average population
755 intelligence (Fig. 4B, Model 1 curve). At high prevalences, selection for intelligence is
756 maintained regardless of the population size (right panel, Fig. 6B). Intelligence plateaus about
757 half way through the run when the population size rebounds slightly but remains low and
758 prevalence decreases slightly from its earlier peak and remains moderate. With a low
759 population size, intermediate prevalence, and a decreased rate of care-giving (Fig. 1B,
760 measles-like curve), the population maintains the higher intelligence, but does not continue to

761 increase it (change in intelligence approaches 0 in left side of middle panel, Fig. 6B).
762 Intelligence plateaus as the boom-bust outbreak oscillations cease.

763 The CCHF-like disease produces a very pronounced boom-bust cycle with several peaks
764 and crashes in population size and prevalence. Selection for increasing intelligence occurs
765 both during low population sizes and low prevalences (left panel, Fig. 7B) and during high
766 population size and high prevalences (right panel, Fig. 7B). When the boom-bust dynamic
767 stops about halfway through the run and the population stabilizes at intermediate population
768 sizes and prevalences, intelligence plateaus (Figs. 2C, 3C, 4C Model 1 curves and middle
769 panel, Fig. 7B).

770 Interestingly, when the population infected with the measles-like disease engages in
771 care-giving, it experiences less pronounced oscillations of the "boom and bust" outbreak
772 cycle (Fig. 3) indicating that care-giving serves to control the spread of the disease through
773 the population. Because of the higher risks of providing care under the CCHF-like conditions,
774 only close kin (r=0.5) receive care. This lower level of care is less effective at controlling the
775 spread of the disease, perhaps suggesting that a certain threshold must be achieved in order to
776 disrupt the boom-bust outbreak cycle (boom-bust dynamics: (Keeling and Grenfell 1997)).
777 Alternatively, the higher fatality rate and more rapid transmission of the CCHF-like diseases
778 produces faster outbreak cycles, which may make it more difficult for care-giving to disrupt
779 the boom-bust outbreak cycle even though it still selects for increasing cognitive abilities.

780 For both the measles-like and CCHF-like diseases, the most pronounced outbreaks occur
781 early in the model run, which is also when the greatest increases in intelligence are occurring
782 (Fig. 6A and 7A). In the second half of the run, when the boom-bust dynamic is less
783 pronounced, intelligence plateaus. This suggests that over the course of human evolution,
784 sustained increases in intelligence may have occurred through repeated introductions of novel
785 diseases into naïve populations. The greatest selection would have occurred shortly after the

786 introduction when the disease was spreading and care-giving behavior had not yet managed
787 to reduce the size of the outbreaks and subsequent population crashes.
788
789 *Significance for human evolution*
790 Our model was parameterized based upon group sizes, spatial scales, and population
791 densities derived from the fossil record and modern foraging peoples (Grove et al. 2012;
792 Layton et al. 2012). Our goal was not to recreate a particular hominin population, but to
793 explore the effects of different disease characteristics on the evolution of care-giving and
794 increased cognition in a population with hominin characteristics.
795 We created an SIS model (susceptible-infected-susceptible) where recovered individuals
796 are just as susceptible as those who were never infected. However, for many diseases,
797 recovered individuals are temporarily or permanently immune to re-infection, potentially
798 increasing their ability to provide care. We expect that immunity would increase the rate of
799 care-giving. Diseases likely to select for care-giving among kin may be diseases which
800 frequently infect children and then convey lifetime immunity. Under this scenario, adults who
801 survived to reproduce would have extensive knowledge of the disease's symptoms, making
802 recognition likely, and the immunity to enable them to provide effective care. Several well-
803 known childhood diseases that follow this pattern (e.g., measles, smallpox) have been dated
804 to the origins of agriculture, animal domestication, and the subsequent population increases
805 (Harper and Armelagos 2013). However, as more genetic studies are conducted, increasing
806 numbers of pathogens are showing pre-agricultural origins, including some that were
807 previously believed to be post-agricultural (e.g., tapeworms, TB (Harper and Armelagos
808 2013; Hoberg et al. 2001; Hurtado et al. 2008; Stone et al. 2009). Tapeworms, TB, typhoid
809 fever, whooping cough, and Epstein Barr virus, among others, have been shown to predate
810 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.

A novel hypothesis of human cognitive evolution and future directions

Our novel hypothesis of primate, including human cognitive evolution, is ***not*** mutually exclusive with the social brain hypothesis (Dunbar 1998). As social species evolved the cognitive capacities for social cognition, such as processing information gleaned from faces (Leopold and Rhodes 2010; Sartori et al. 2011), voices (Belin 2006; Belin et al. 2004), and movement patterns (Loula et al. 2005; Peterman et al. 2014; Sartori et al. 2011), they may have also obtained the ability to use this information to recognize disease symptoms. They could detect changes in facial coloration and texture due to fever or rashes, changes in vocalizations due to coughing, nasal discharge or reduced lung capacity, and changes in movement/gait due to weakness, lethargy, or signs of pain (Chapman et al. 2005; Fink and Matts 2008; Hart 1988). The proximate mechanisms are relatively simple in that they do ***not*** require individuals to have an abstract concept of "disease." Instead, individuals that are able to accurately recognize disease would have increased fitness due to being able to avoid infectious individuals or provide care to kin. Though studies of disease recognition in nonhuman animals are relatively rare, several species do appear to recognize the health status of conspecifics, i.e., social lobsters (Behringer et al. 2006), pipefish (Rosenqvist and

835 Johansson 1995), bullfrog tadpoles (Kiesecker et al. 1999), rodents (Kavaliers et al. 1997),
836 house finches (Bouwman and Hawley 2010; Zylberberg et al. 2012), but see (Nunn 2003).
837 We predict that as hominin social complexity increased, i.e., group sizes, social network
838 sizes, frequencies of cooperation and social learning, etc. (Aiello and Dunbar 1993; Burkart
839 et al. 2014; Burkart et al. 2009; Dunbar 1998; Gamble et al. 2011; Grove et al. 2012; Layton
840 et al. 2012; Tomasello 2014), hominins would have substantially increased their risk of
841 disease transmission, producing heightened selection for disease recognition and care-giving.
842 We make several predictions that enable paleoanthropologists, archaeologists, primatologists,
843 human ecologists, geneticists and immunologists to test our novel hypothesis of human
844 cognitive evolution:
845 1) Humans and nonhuman primates have very similar disease profiles in that we share
846 many of the same diseases with viral, bacterial, and gastrointestinal parasitic zoonoses
847 occurring from nonhuman primates to humans and vice versa (Chapman et al. 2005;
848 Jones et al. 2008; Lloyd-Smith et al. 2009; Wolfe et al. 2007). However, what has
849 received very little attention is how humans and nonhuman primates may differ in the
850 expression of disease symptoms. Humans, relative to nonhuman primates have much
851 less body hair. Though our nakedness may reduce ectoparasite load (Pagel and
852 Bodmer 2003; Weiss 2007), it also provides a visually unobstructed surface for
853 displaying rashes, lesions, swelling, and inflammation, and bruising. Humans, relative
854 to nonhuman primates, also have white scaleras around their eyes, a signal that has
855 been argued to draw attention to gaze direction (Kobayashi and Kohshima 2001;
856 Tomasello et al. 2007), but also turns a dramatic "bloodshot" red when we are under
857 emotional stress or ill (Provine et al. 2011). ***Prediction 1:*** *If humans have been*
858 *selected to solicit care from others, they should display exaggerated signals of ill*

health, relative to nonhuman primates experiencing the same disease and degree of morbidity/mortality.

2) It is becoming increasingly possible to date the origins of many diseases afflicting humans i.e., (Harper and Armelagos 2013; Stone et al. 2009). As more accurate dates are obtained for more diseases, it will be possible to examine whether hominin populations carried an increased disease load as they increased in social complexity. Social 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

884 enough not to disrupt the kin networks along which care is provided. Possibilities
885 include infections that leave survivors immune.
886 3) An additional avenue for examining the role of disease during the evolution of human
887 social complexity would be through cross-species comparisons of immune
888 investment. If hominins have experienced an unusually high rate of disease exposure,
889 either through their extensive social networks or through providing care to diseased
890 kin, they may have invested heavily in immune defenses. Recent work on
891 introgression between anatomically modern humans (AMH) and neandertals has
892 proposed that one of the major advantages may have been the acquisition of novel
893 immune genes from neandertals as AMH expanded northward into novel
894 environments and encountered novel pathogens (Houldcroft and Underdown 2016).
895 Prior studies indicate that there are cross-species differences in immune investment
896 according to mating system (but not group size or density in primates) (Nunn et al.
897 2000), the risk of environmentally transmitted parasites and injuries due to predator
898 attacks in anthropoids (Semple et al. 2002), coloniality in birds (Moller et al. 2001),
899 and cooperative breeding in birds (Spottiswoode 2008). ***Prediction 1*****:** *If hominins'*
900 *increased social complexity required them to invest heavily in immune defenses, the*
901 *human immune system should show similar adaptations to other species that have*
902 *extremely large social networks and high interaction rates.* ***Prediction 2:*** *If the*
903 *evolution of cooperative breeding required hominins to invest heavily in immune*
904 *defenses, then the human immune system should show similar adaptations to other*
905 *cooperatively breeding species.* ***Prediction 3:*** *If the evolution of providing care to*
906 *diseased conspecifics required hominins to invest heavily in immune defenses, the*
907 *human immune system should show adaptations that are either extreme or unusual.*
908 (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.

Conclusions

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

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

Data accessibility

The ODD descriptions of Model 1 (caregiving) and Model 2 (avoidance only) are found in Appendices A and B, respectively. The code is available in supplementary files 1 and 2, respectively. The files containing the code can be opened with standard text editing programs 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)			
Genus	**Species**	**Lower CI**	**Median**	**Upper CI**	**Lower CI**	**Median**	**Upper CI**	**Low**
Homo	Early *Homo*	43.249	56.276	71.402	0.366	0.584	0.802	51.5
Homo	*habilis*	46.8415	60.476	76.2795	0.577	0.822	1.068	43.8
Homo	*erectus*	66.43	83.158	102.406	0.545	0.785	1.025	70.2

Homo	*heidelbergensis*	70.9845	88.389	108.389	0.3	0.514	0.728	94.7
Homo	*neanderthalensis*	72.622	90.266	110.5325	0.196	0.407	0.618	116.
Homo	*sapiens*	78.763	97.292	118.541	0.196	0.407	0.618	127.

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**	
	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	0.0
Measles	133.64	2.02	70.15	0.76	6.74	0.43	0.71	0.01	0.21	0.01	0.00053	0.0
CCHF	120.96	3.47	33.63	1.75	4.73	0.50	0.62	0.01	0.12	0.02	0.00022	0.0
Ebola	157.24	3.25	10.32	0.51	---	---	0.50	0.01	0.00	0.01	---	---

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	≥ 12.68[T]	<0.001
Final Prevalence	11,275.24[BF]	3, 15.24	<0.001	≥ 0.15[T]	< 0.001
Final Percent Care	492.03[BF]	2, 18.61	<0.001	≥ 0.02[T]	<0.001
Final Intelligence	579.51[UC]	3, 36	<0.001	≥ 0.03[B]	<0.001
Intelligence Change	464.463[BF]	3, 23.13	<0.001	≥ 0.03[T]	<0.001
Max. Slope Percent Care	377.10[UC]	2, 27	<0.001	≥0.0003[B]	<0.001
Max. Slope Intelligence	421.732[BF]	3, 21.61	<0.001	≥ 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

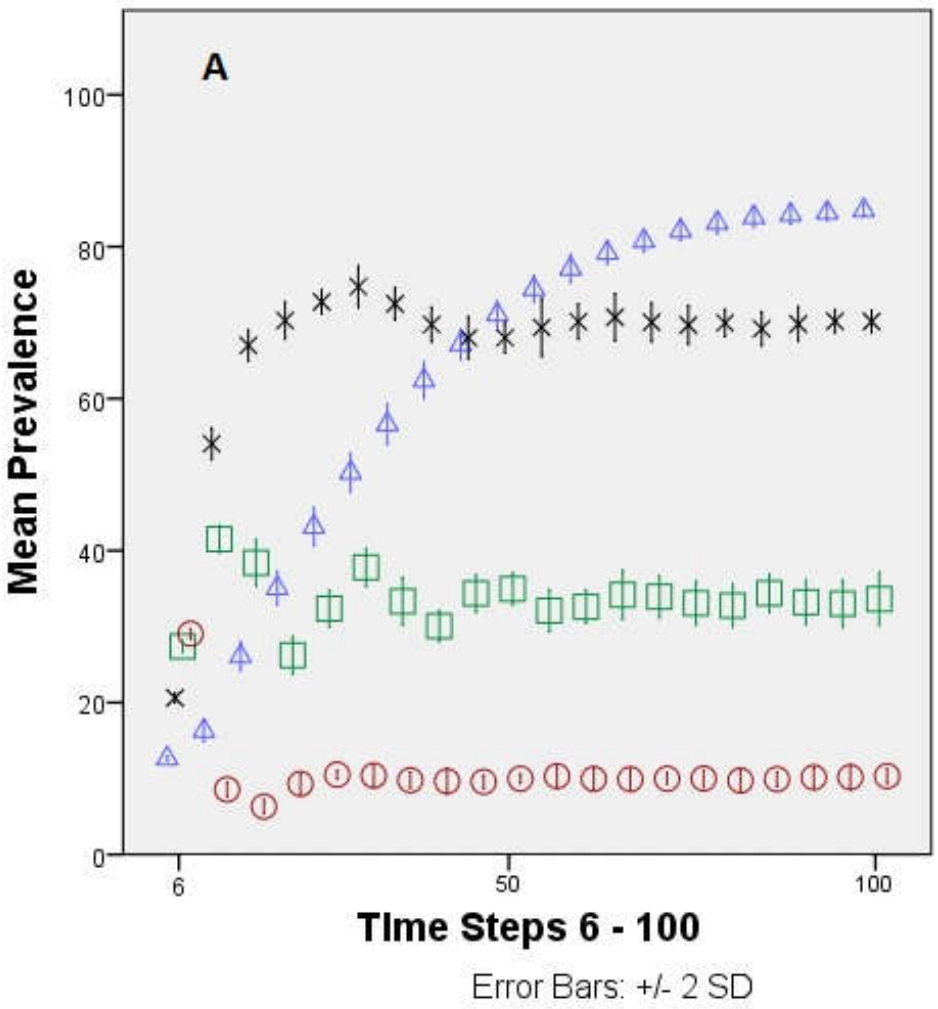
Table 7. Mixed-model analyses run on the Model 1 (care-giving) results examining the effects of prevalence, population size and the interaction between the two on intelligence changes for each disease. r2m measures how much variation in mean intelligence can be explained by the fixed effects (time+prevalence*population size).

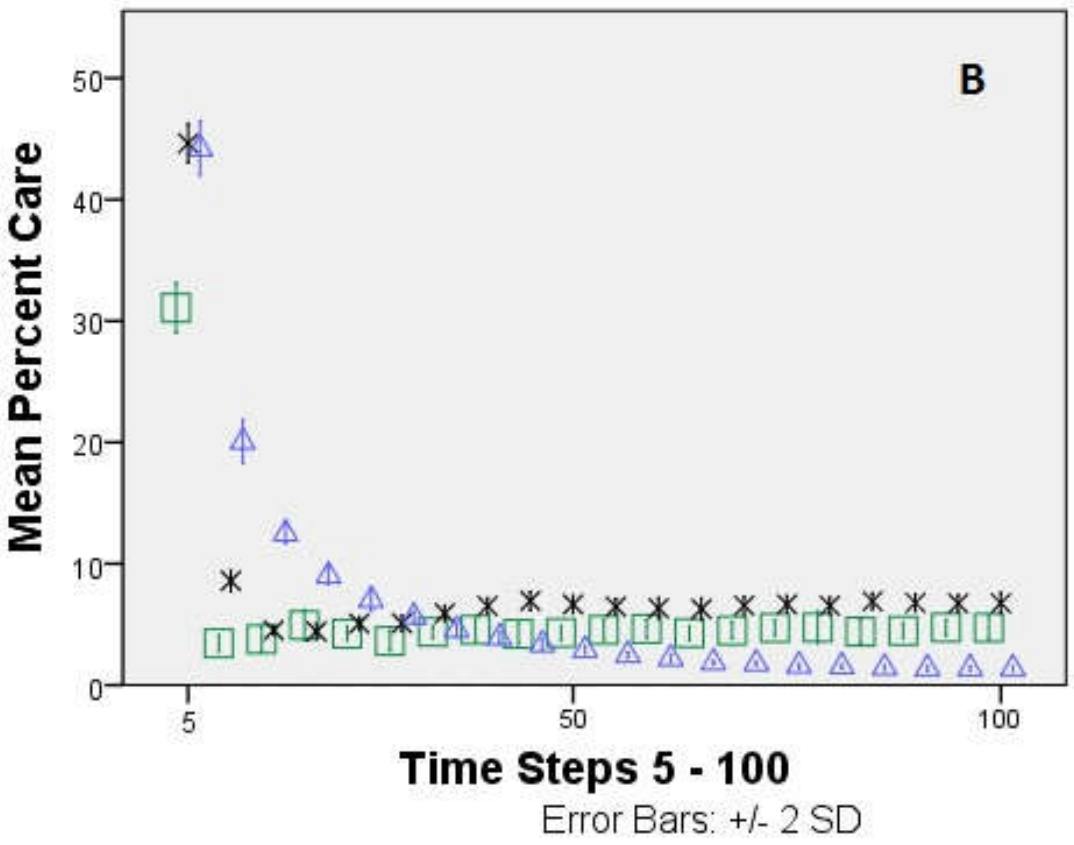
β values are standardized regression coefficients. SE is the standard error and df is the degrees of freedom.

Disease	Analysis	r^2m*	Variable	B	SE	df	t	
Scabies-like*	Prevalence	0.468	Intercept	-0.002	0.034	888	-0.055	0
			Time	-1.084	0.086	888	-12.641	<0
			Prevalence	0.460	0.085	888	5.411	<0
Measles-like	Prevalence	0.565	Intercept	-0.065	0.075	946	-0.871	0
			Time	-0.585	0.076	946	-7.650	<0
			Population Size	0.291	0.063	946	4.590	<0
			Prevalence	0.431	0.046	946	9.276	<0
			Population Size*Prevalence	-0.143	0.021	946	-6.713	<0
CCHF-like	Prevalence	0.146	Intercept	0.039	0.050	946	0.785	0
			Time	-0.400	0.051	946	-7.848	<0
			Population Size	0.052	0.051	946	1.014	0
			Prevalence	-0.104	0.052	946	-2.023	0
			Population Size*Prevalence	0.060	0.020	946	3.023	0
Ebola-like	Prevalence	0.001	Intercept	0.008	0.039	946	0.218	0
			Time	-0.010	0.039	946	-0.247	0
			Population Size	-0.043	0.049	946	-0.873	0
			Prevalence	0.002	0.073	946	0.031	0
			Population Size*Prevalence	0.013	0.022	946	0.571	0

*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 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.




B
Mean Percent Care
50
40
30
20
10
0
5
50
100
Time Steps 5 - 100
Error Bars: +/- 2 SD

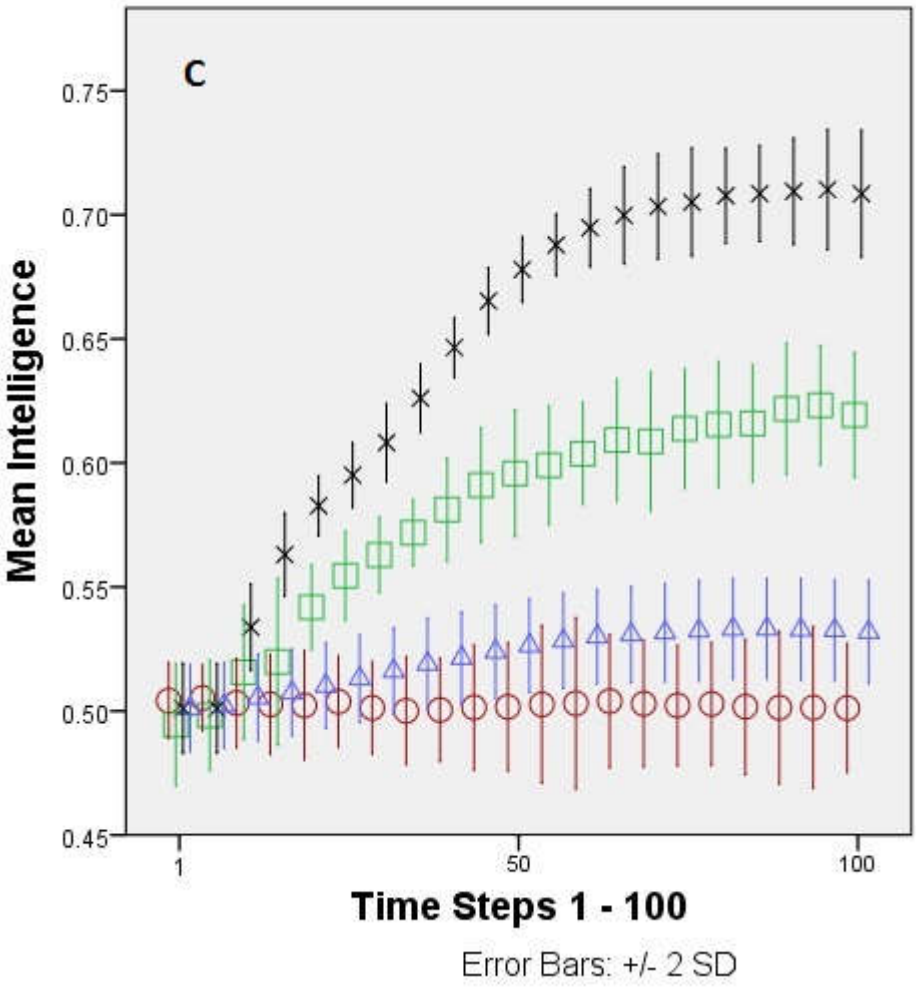
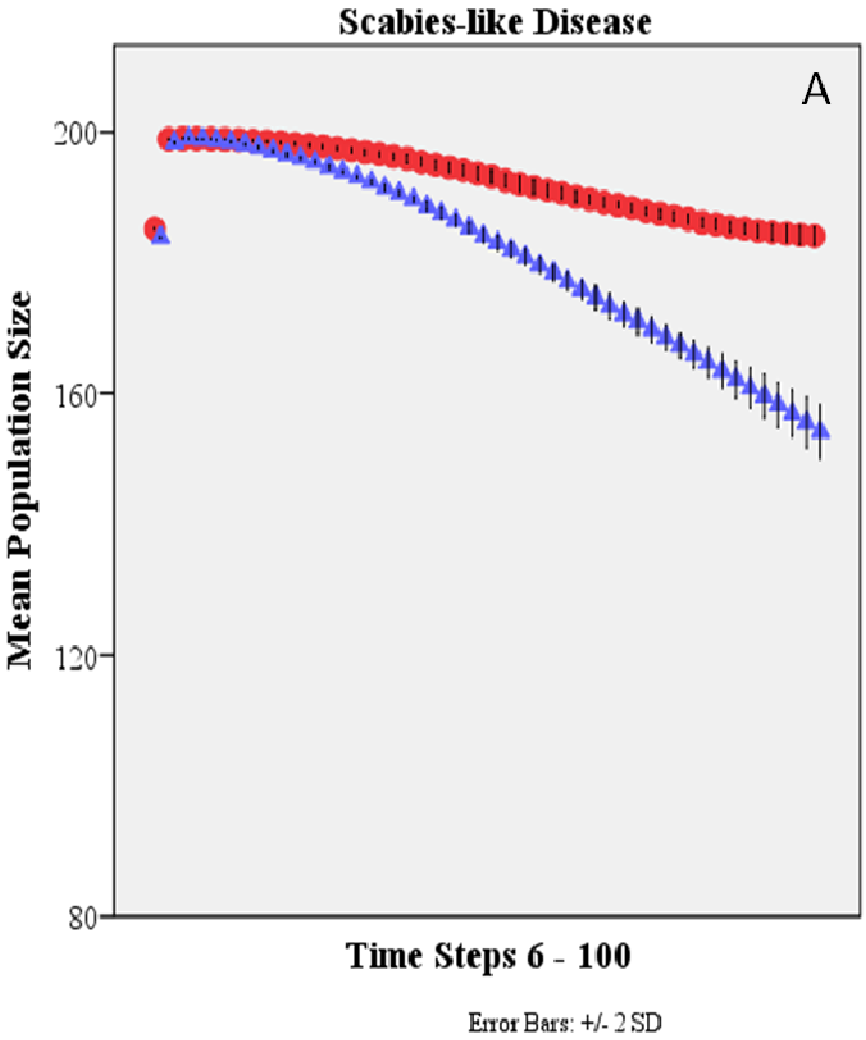
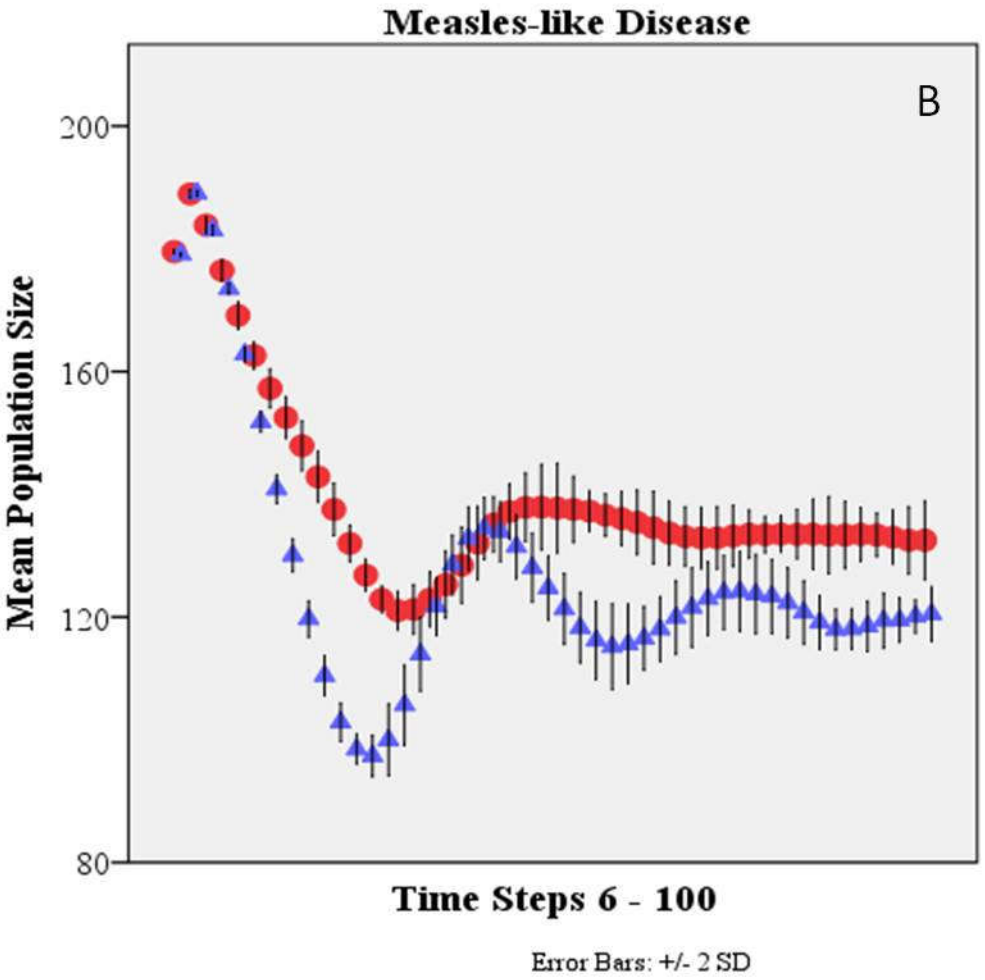
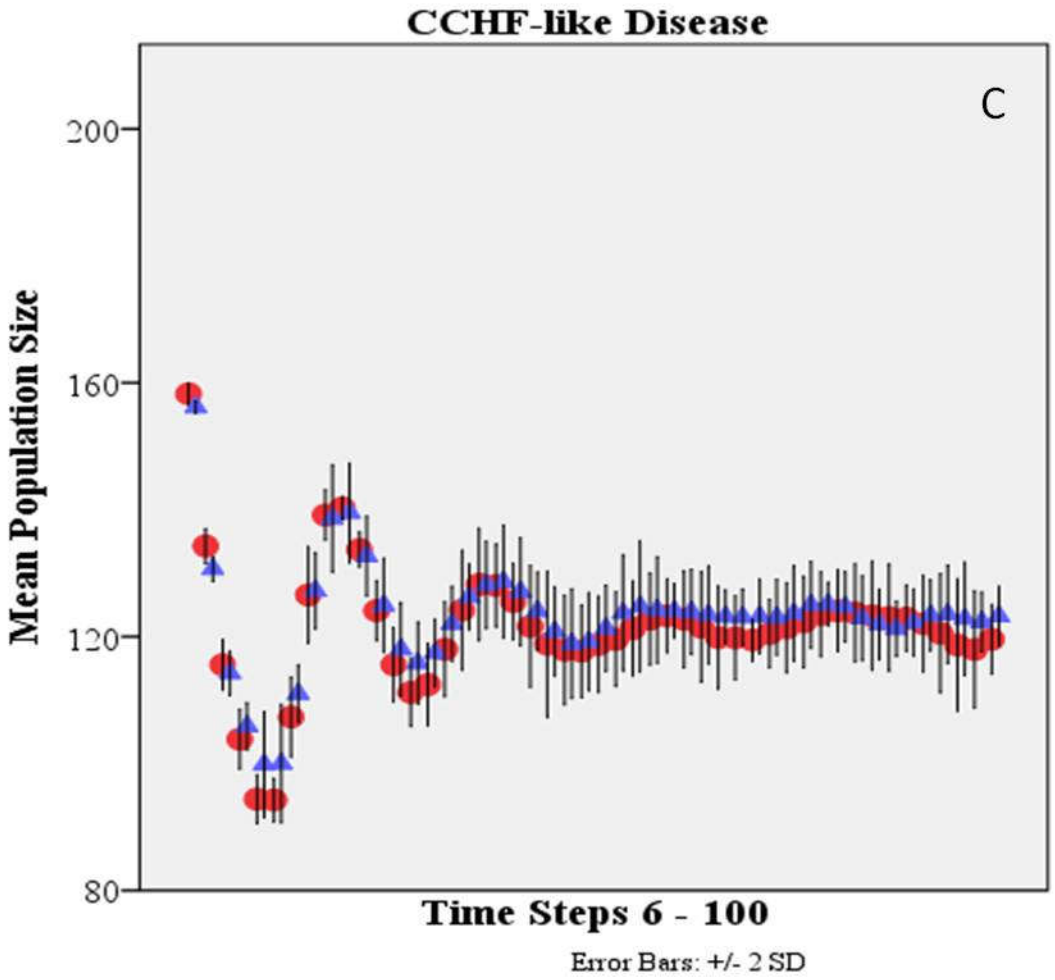

C
Mean Intelligence
0.75
0.70
0.65
0.60
0.55
0.50
0.45
1
50
100
Time Steps 1 - 100
Error Bars: +/- 2 SD

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.




Measles-like Disease
B
200
160
120
80
Mean Population Size
Time Steps 6 - 100
Error Bars: +/- 2 SD


CCHF-like Disease
C
200
160
120
80
Mean Population Size
Time Steps 6 - 100
Error Bars: +/- 2 SD

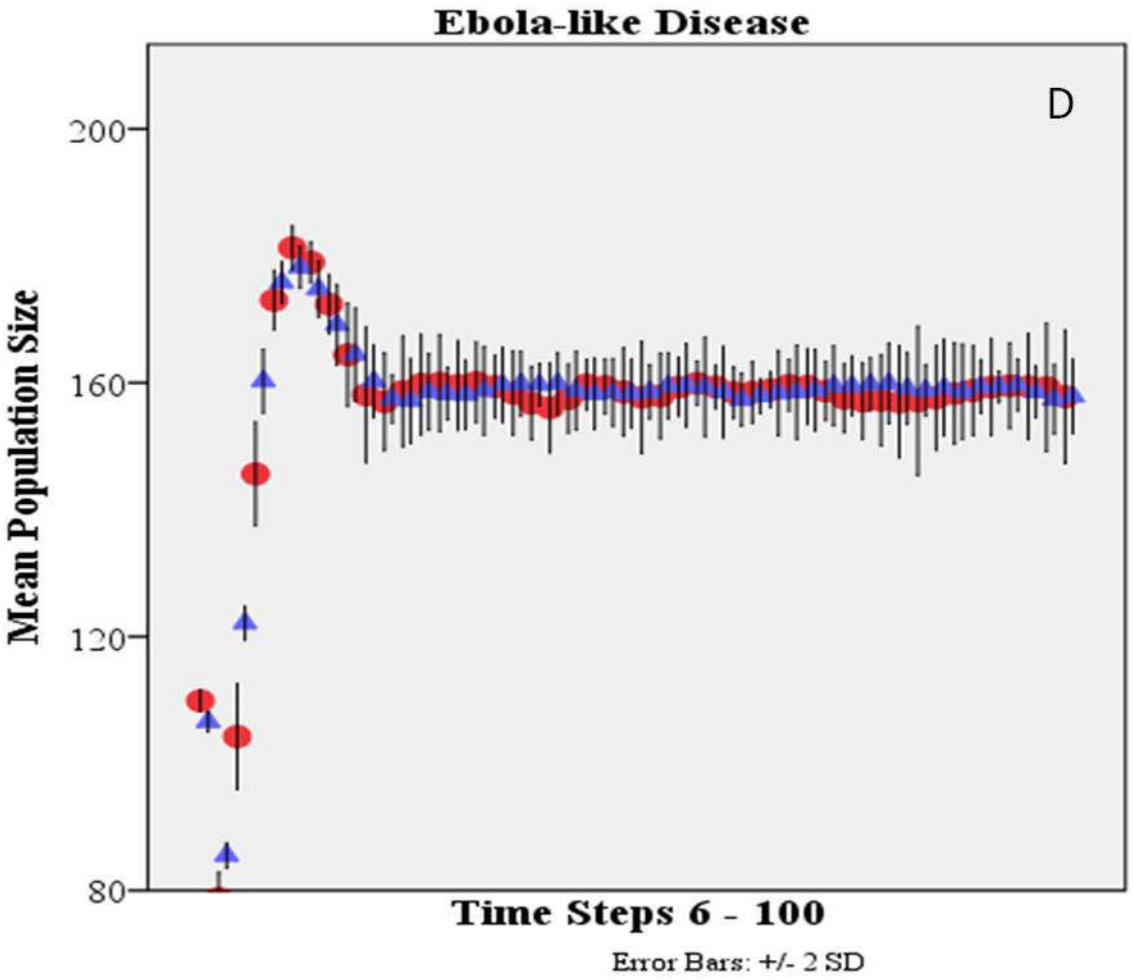
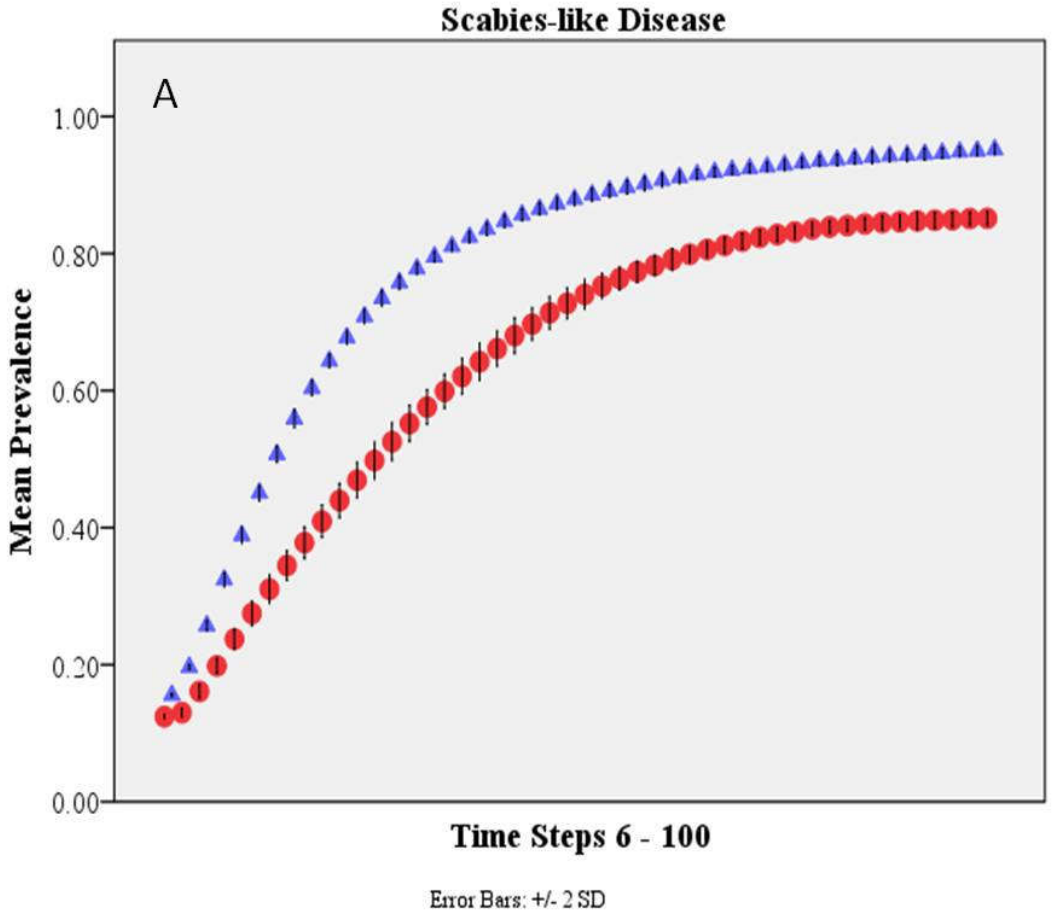
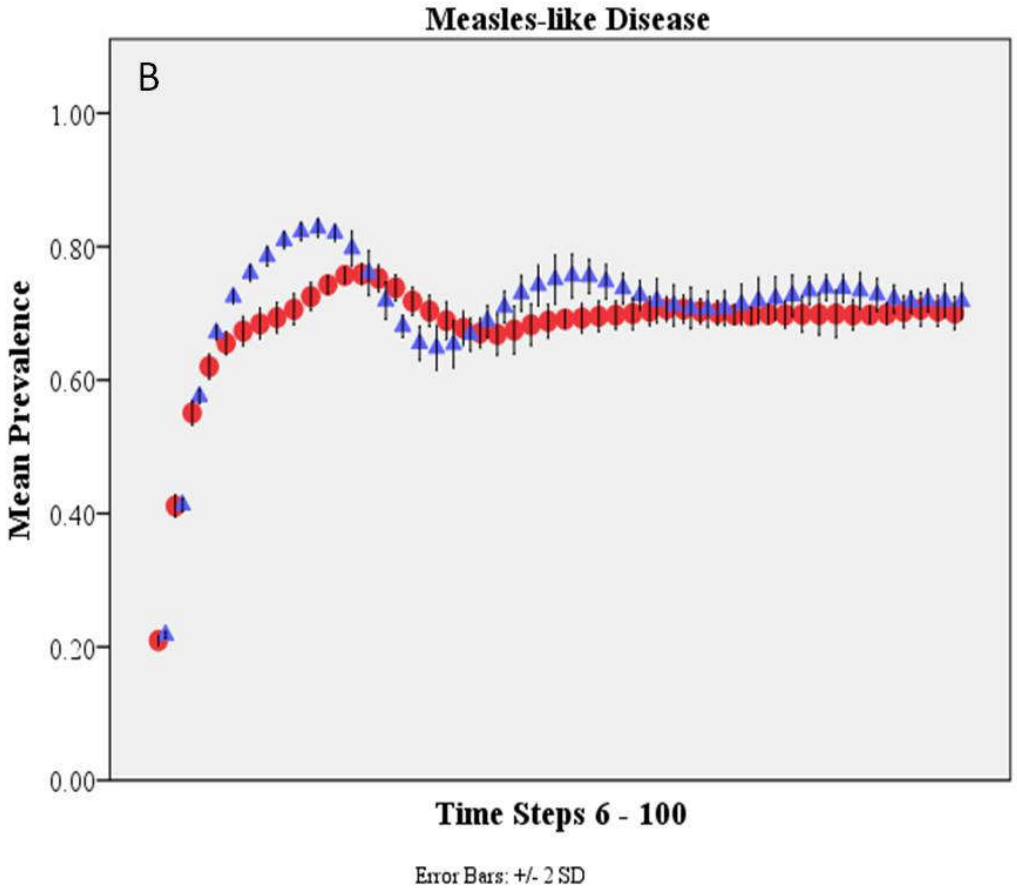
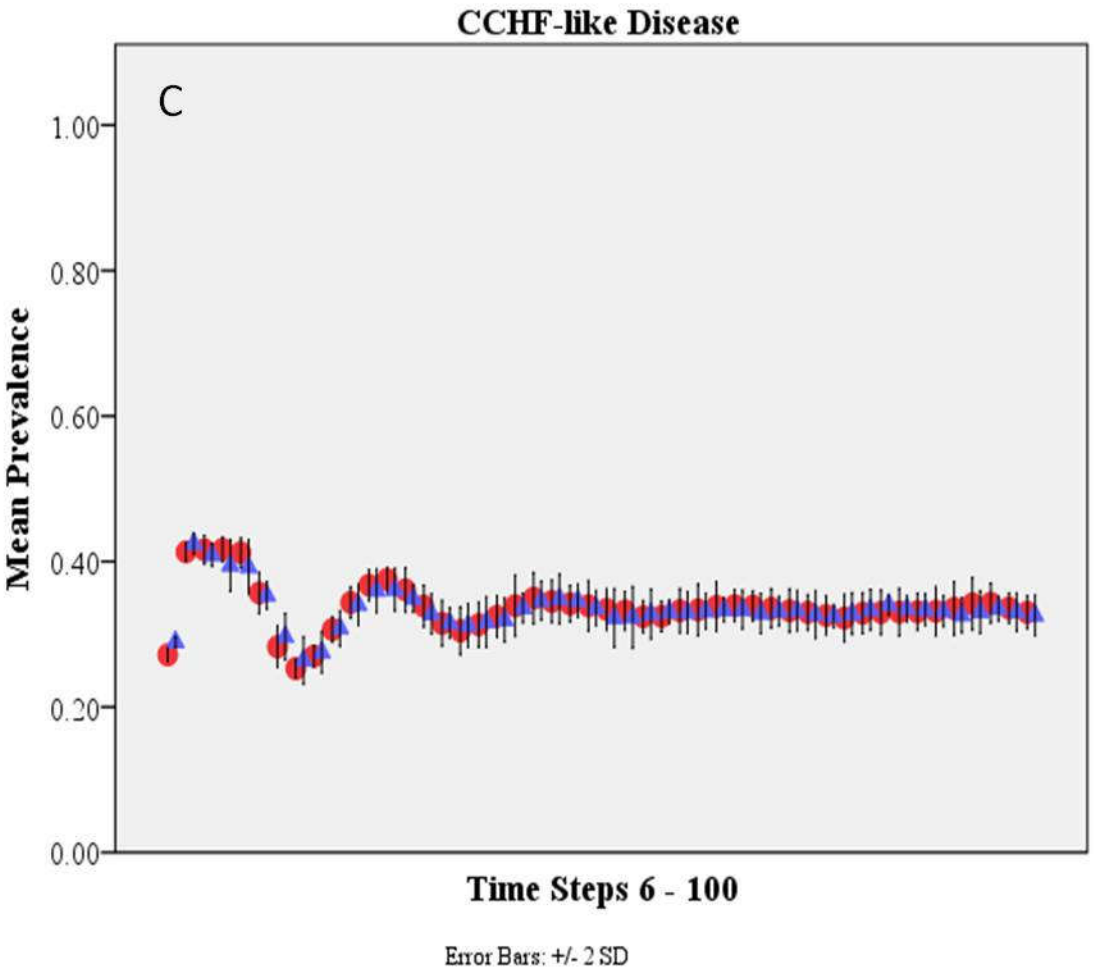

Ebola-like Disease
D
200
160
120
80
Mean Population Size
Time Steps 6 - 100
Error Bars: +/- 2 SD

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.




Measles-like Disease
B
1.00
0.80
0.60
0.40
0.20
0.00
Mean Prevalence
Time Steps 6 - 100
Error Bars: +/- 2 SD


CCHF-like Disease
C
1.00
0.80
0.60
0.40
0.20
0.00
Mean Prevalence
Time Steps 6 - 100
Error Bars: +/- 2 SD

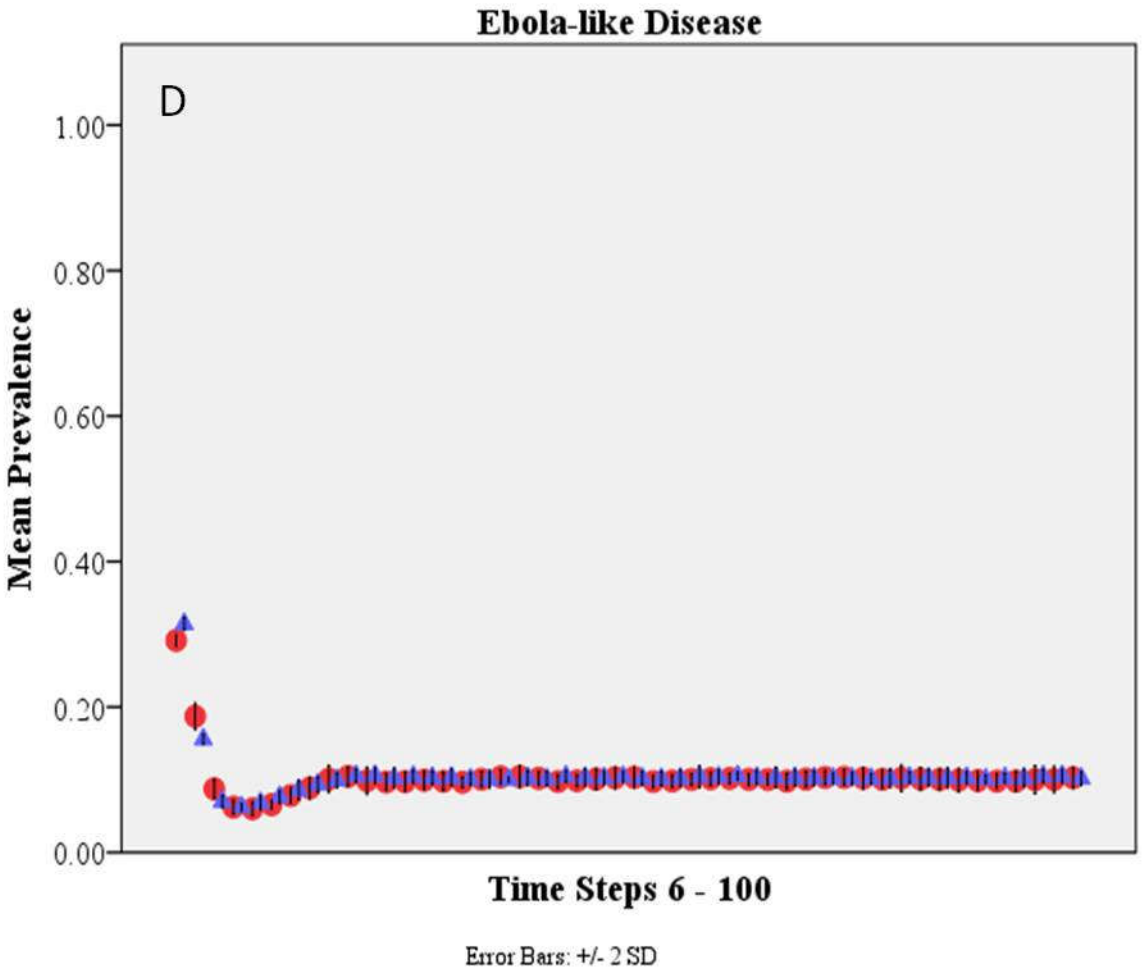
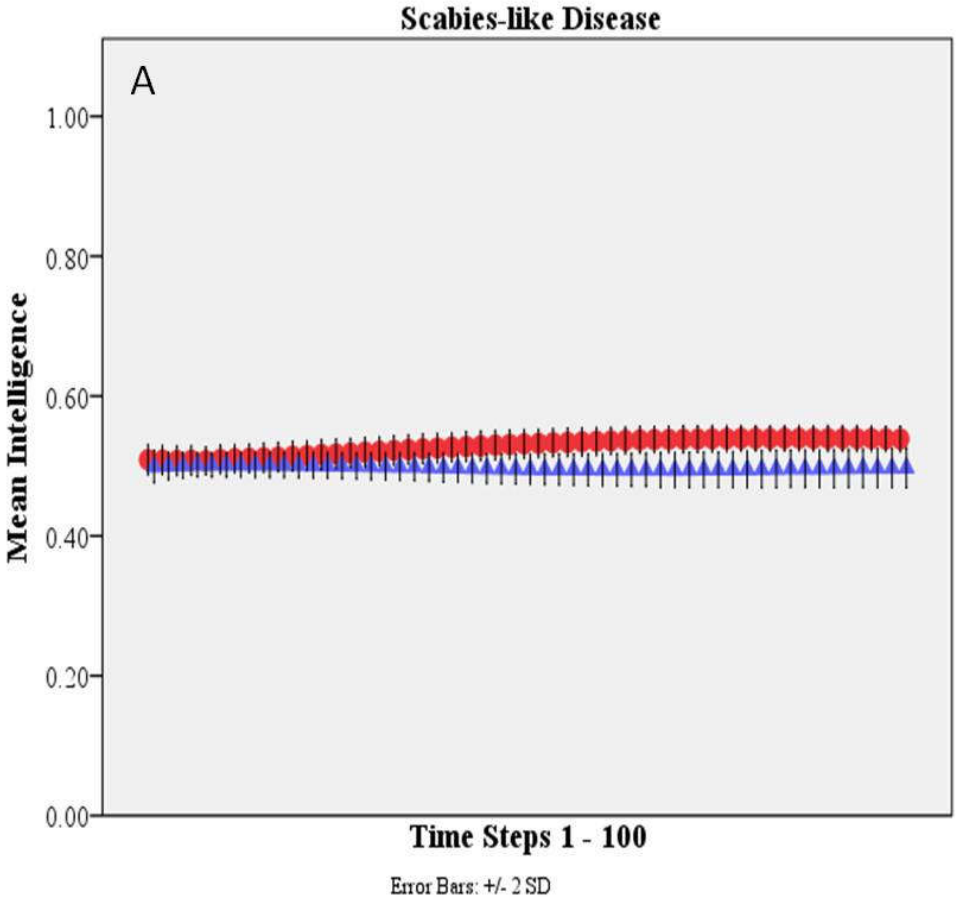
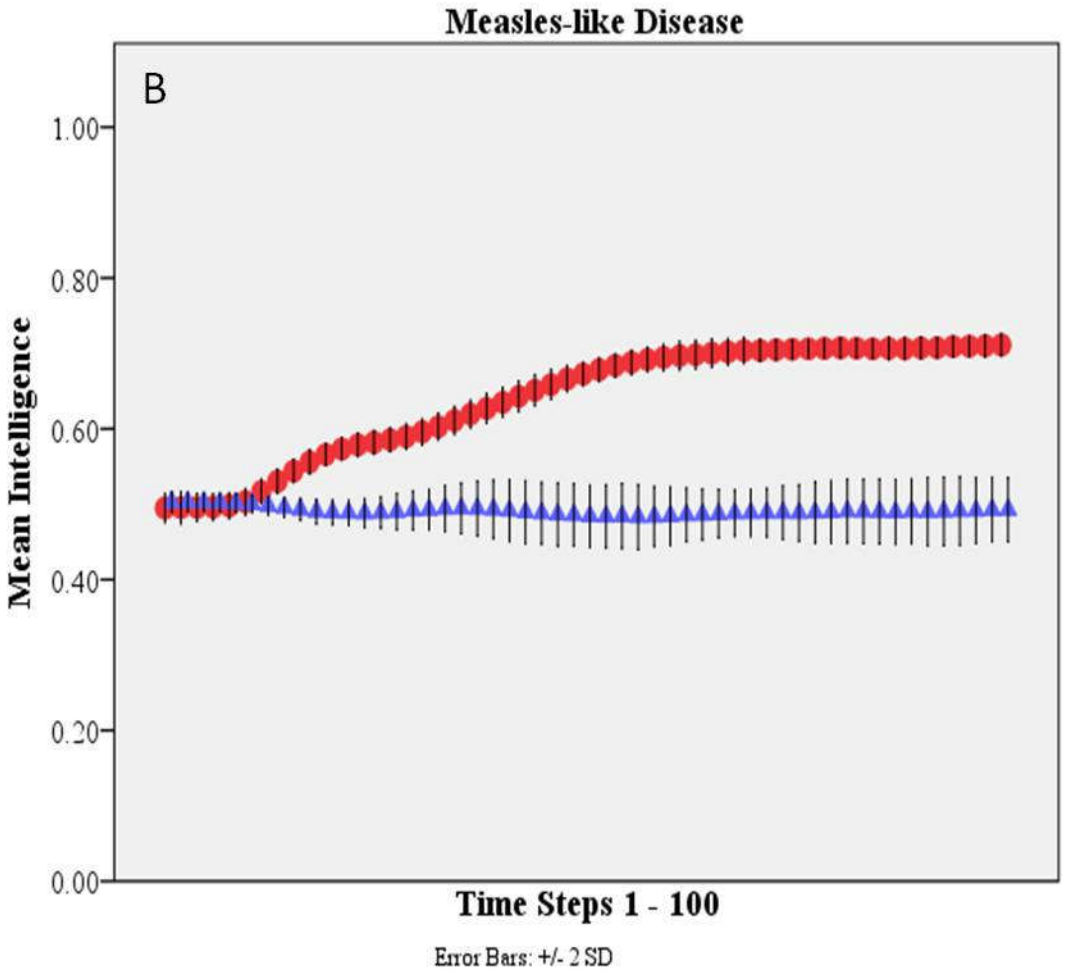
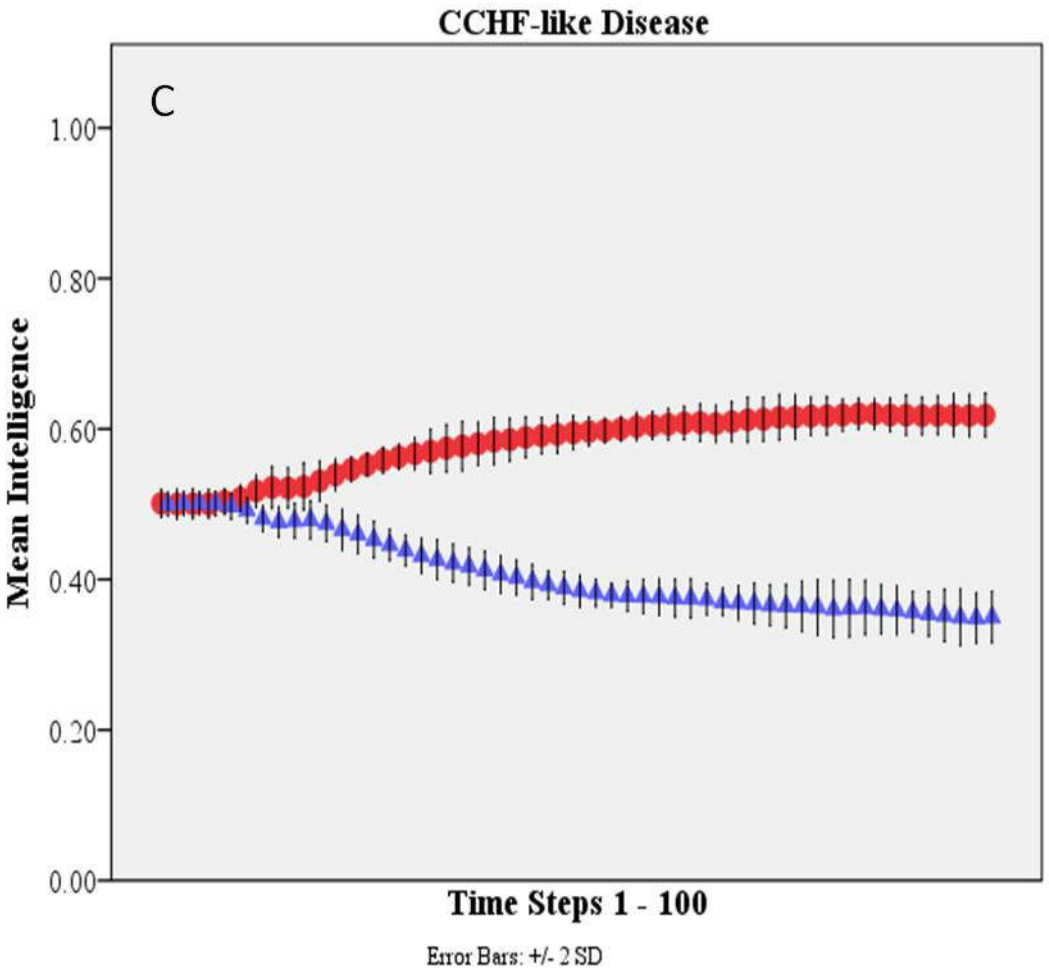

Ebola-like Disease
D
1.00
0.80
0.60
0.40
0.20
0.00
Mean Prevalence
Time Steps 6 - 100
Error Bars: +/- 2 SD

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.




Measles-like Disease
B
1.00
0.80
0.60
0.40
0.20
0.00
Mean Intelligence
Time Steps 1 - 100
Error Bars: +/- 2 SD


CCHF-like Disease
C
1.00
0.80
0.60
0.40
0.20
0.00
Mean Intelligence
Time Steps 1 - 100
Error Bars: +/- 2 SD

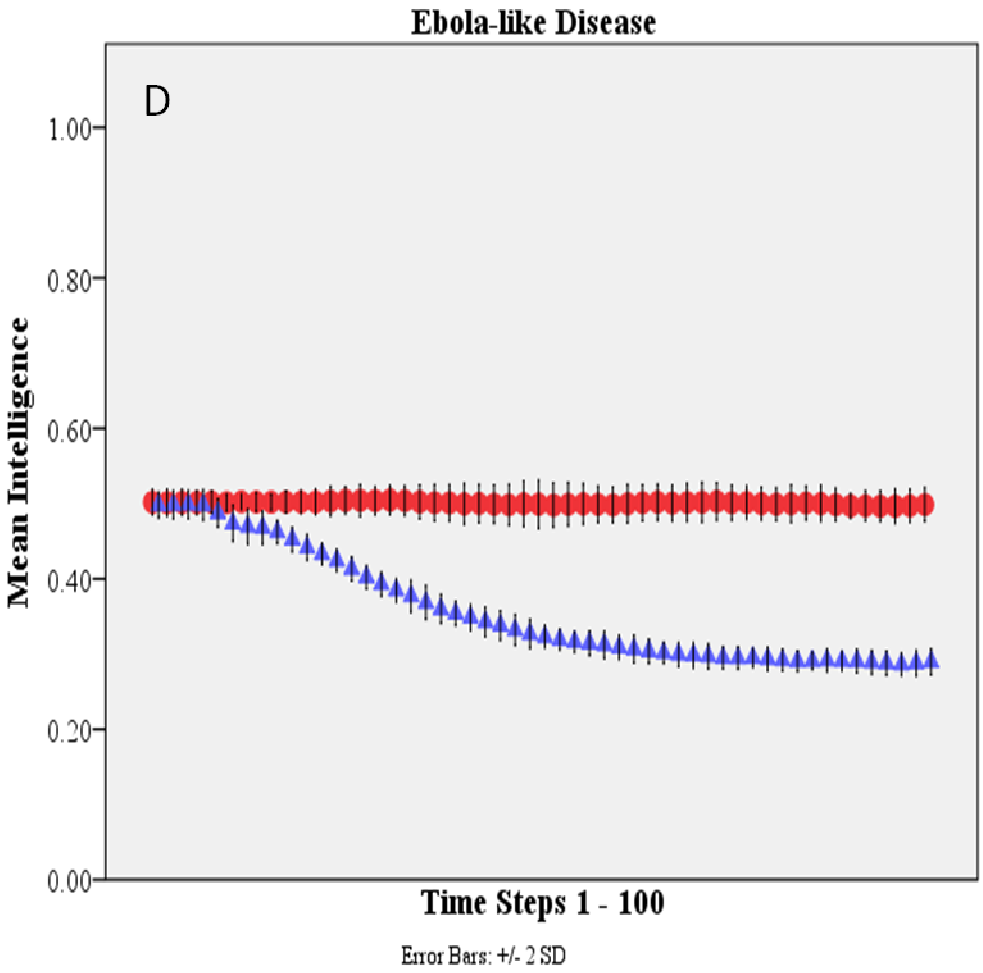

Ebola-like Disease
D
1.00
0.80
0.60
0.40
0.20
0.00
Mean Intelligence
Time Steps 1 - 100
Error Bars: +/- 2 SD

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).

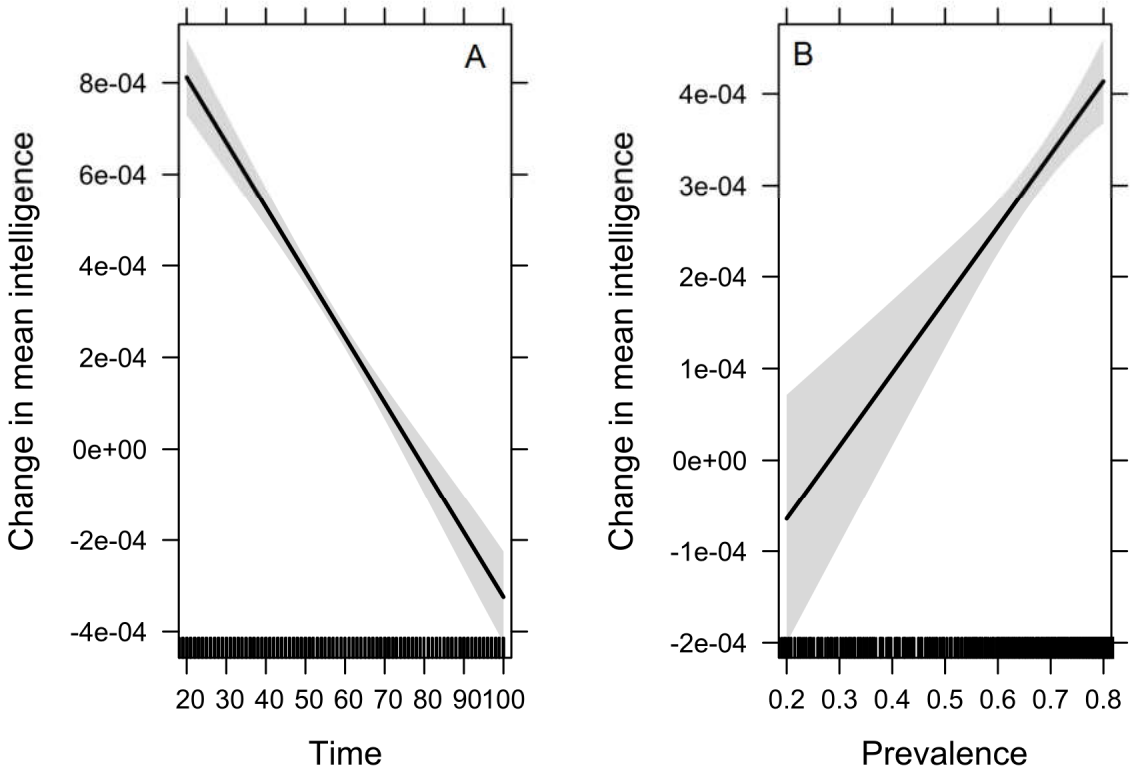


Figure 6. Graphs showing the results of the analyses exploring the effects of prevalence, population size, and their interactions on the change in intelligence for the measles-like disease. Change in intelligence was calculated as the mean intelligence in a given time step minus the mean intelligence 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).

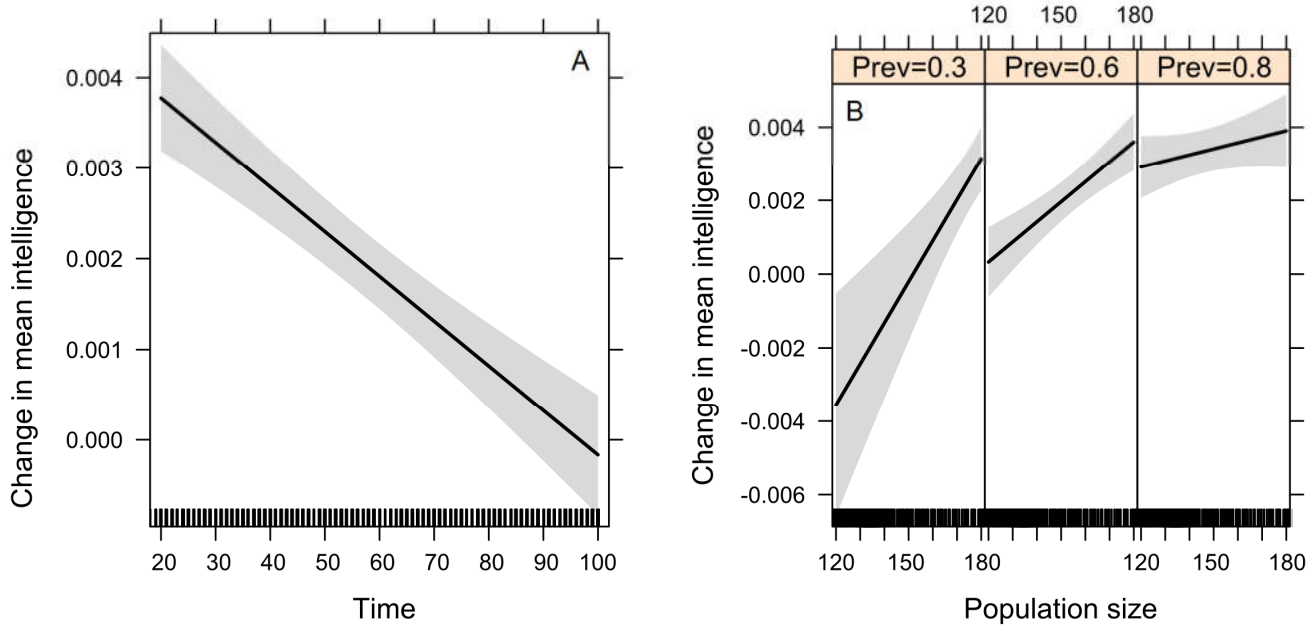


Figure 7. Graphs showing the results of the analyses exploring the effects of prevalence, population size, and their interactions on the change in intelligence for the CCHF-like disease. Change in intelligence was calculated as the mean intelligence in a given time step minus the mean intelligence in 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).

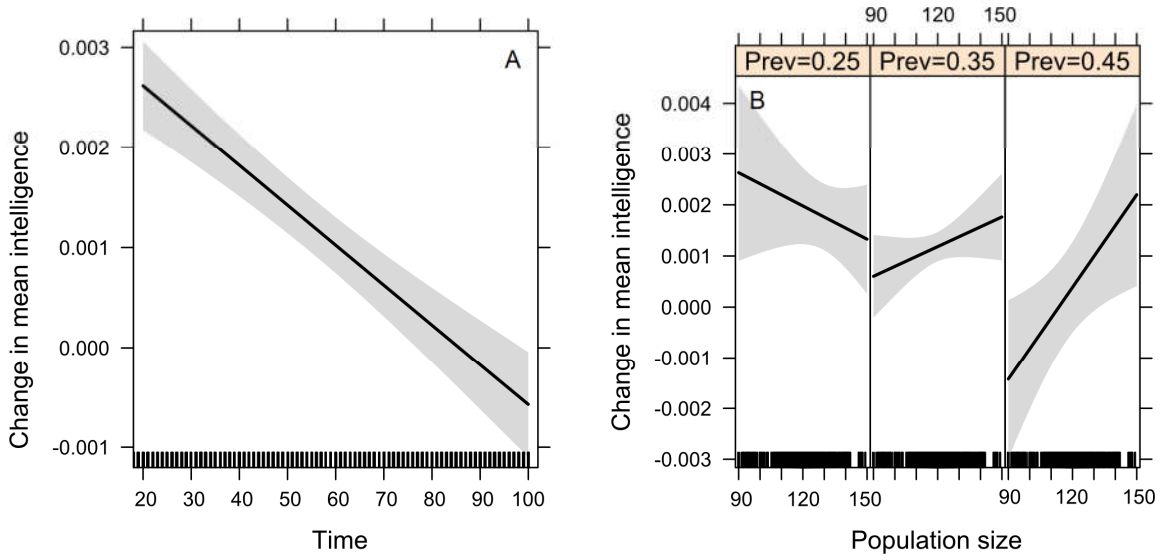


Figure 8. Graphs showing the results of the analyses exploring the effects of prevalence, population size, and their interactions on change in intelligence for the Ebola-like disease. Change in intelligence was calculated as the mean intelligence in a given time step minus the mean intelligence in the 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).