Journal of Human Evolution 108 (2017) 92–109

Contents lists available at ScienceDirect

Journal of Human Evolution

journal homepage: www.elsevier.com/locate/jhevol

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

Sharon E. Kessler [a, b, *], Tyler R. Bonnell [c], Richard W. Byrne [d], Colin A. Chapman [a, e]

[a] Department of Anthropology, McGill University, Canada
[b] Durham University, Department of Anthropology, UK
[c] Department of Psychology, University of Lethbridge, Canada
[d] School of Psychology and Neuroscience, University of St. Andrews, UK
[e] Wildlife Conservation Society, New York, USA

ARTICLE INFO

Article history:
Received 24 November 2015
Accepted 26 February 2017
Available online 17 May 2017

Keywords:
Agent-based model
Disease transmission
Cooperation
Hominin evolution
Social complexity
Kin selection

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, care may have minimized the costs of socially transmitted diseases at a time of increasing social complexity, although individual care-givers probably incurred increased transmission risks. We propose that care-giving likely originated within kin networks, where the costs may have been balanced by fitness increases obtained through caring for ill kin. We test a novel hypothesis of hominin cognitive evolution in which disease may have selected for the cognitive ability to recognize when a conspecific is infected. 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. Using agent-based modeling, we test 1) under what conditions disease can select for increasing disease recognition and care-giving among kin, 2) whether providing care produces greater selection for cognition than an avoidance strategy, and 3) whether care-giving alters the progression of the disease through the population. The greatest selection was produced by diseases with lower risks to the care-giver and prevalences low enough not to disrupt the kin networks. When care-giving and avoidance strategies were compared, only 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, 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 that we encourage paleoanthropologists, bioarchaeologists, primatologists, and paleogeneticists to test.

1. Introduction

Exposure to disease is a major cost of sociality (Nunn and Altizer, 2006; Rifkin et al., 2012; McCabe et al., 2015). 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 (Sugiyama, 2004; Rifkin et al., 2012; Harper and Armelagos, 2013; McCabe et al., 2015). 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. In this paper, we integrate findings from the literature on hominin social structure, hominin disease ecology, disease recognition in

* Corresponding author.
E-mail address: Sharon.E.Kessler@durham.ac.uk (S.E. Kessler).

http://dx.doi.org/10.1016/j.jhevol.2017.02.009
0047-2484/

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, paleogeneticists, bioarchaeologists, and primatologists.

1.1. 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 (Hill et al., 2011; Grove et al., 2012).

Hominin community sizes have been reconstructed as having expanded over time, from ~50 individuals in apes and small-brained australopiths to 100–120 in late *Homo erectus* and *Homo heidelbergensis*, and 120–150 in *Homo neanderthalensis* and *Homo 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 in social network size and 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 people (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 modern male hunter-gather may watch over 300 other men make tools (Hill et al., 2014). The evolution of such long-distance social networks linking different subgroups (Hill et al., 2014) may have prevented the reduction in disease risk that might otherwise be expected to have occurred as hominin density decreased (Armelagos et al., 2005). The extensive, community-wide social networks of hominins would have facilitated widespread pathogen transmission, including any novel pathogens acquired as hominins spread into new habitats (McCabe et al., 2015).

1.2. Increasing connectedness within groups

Simultaneously with the expansion of networks connecting subgroups, the complexity of networks within the subgroups also likely increased with the evolution of cooperative breeding during the origin of *Homo*. *Homo habilis* and *H. erectus* fossil assemblages show an increased number of immature relative to mature individuals compared to *Australopithecus africanus* assemblages, suggesting high mortality among immatures (Tobias, 2006), shortened interbirth intervals, increasing energetic demands on reproducing females, and a shift towards cooperative breeding (Aiello and Key, 2002). Ethnographic work supports this view of humans as cooperative breeders, revealing greatly expanded social networks that include multiple providers (hunting males, post-reproductive females) for females and young (Hawkes, 2003; Hill et al., 2009; Hrdy, 2009). This contrasts with chimpanzees, in which the young are solely dependent upon their mothers (Burkart et al., 2009). Collectively, these studies suggest that as community size increased during the origin of *Homo*, so did the complexity of the social networks linking both greater numbers of individuals and different demographics (e.g., young 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).

1.3. 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) that facilitated prolonged, close interactions among individuals and promoted social learning, cooperation, technological advances, and cumulative culture (Whiten, 2000; Tomasello et al., 2005; Byrne and Bates, 2007; Herrmann et al., 2007; van Schaik et al., 2012; Burkart et al., 2014). 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), herpes viruses (Harper and Armelagos, 2013), and Epstein Barr virus (Harper and Armelagos, 2013). Thus, hominins were likely under strong selection to assess the disease status of others.

1.4. 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 of sick and weakened competitors, e.g., social lobsters (Behringer et al., 2006), pipefish (Rosenqvist and Johansson, 1995), bullfrog tadpoles (Kiesecker et al., 1999), rodents (Kavaliers et al., 1997), house finches (Bouwman and Hawley, 2010; Zylberberg et al., 2012), but see (Nunn, 2003) for a study which found that primates did not use genital inspections to avoid mating with partners infected with sexually transmitted diseases. While the underlying cognitive processes are not well understood, these 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