

# Infectious Disease Stigmas: Maladaptive in Modern Society

Rachel A. Smith & David Hughes

*At multiple times in human history people have asked if there are good stigmas. Is there some useful function stigmas serve in the context of our evolutionary history; is stigma adaptive? This article discusses stigmas as a group-selection strategy and the human context in which stigmas likely appeared. The next section explores how human patterns have changed in modern society and the consequences for infectious disease (ID) stigmas in the modern age. The concluding section suggests that while social-living species may be particularly apt to create and communicate ID stigmas and enact ID-related stigmatization, such stigma-related processes no longer function to protect human communities. Stigmas do not increase the ability of modern societies to survive infectious diseases but, in fact, may be important drivers of problematic disease dynamics and act as catalysts for failures in protecting public health.*

*Keywords: Evolution; Group Membership; Infectious Disease; Ostracism; Stigma*

At multiple times in human history people have asked if there are *good* stigmas. Is there a useful function that they serve? This question is asked in the context of evolutionary history: It is assumed that humans evolved not just out of Africa but also from a hunter-gather society to an agricultural one and then, more recently, to a mostly urban society. Stigmas and stigmatization are ubiquitous in human societies, globally and historically (Smith, 2007), and appear in other social species,

---

Rachel A. Smith is an Associate Professor in Communication Arts & Sciences and David Hughes is an Assistant Professor in Entomology and Biology at The Pennsylvania State University. Smith and Hughes are both investigators in the Center for Infectious Disease Dynamics and the Methodology Center at The Pennsylvania State University. Correspondence to: Rachel A. Smith, PhD, Department of Communication Arts & Sciences, Center for Infectious Disease Dynamics, and The Methodology Center, The Pennsylvania State University, W-252 Millennium Science Complex University Park, PA 16803, USA. E-mail: ras57@psu.edu

from ants to chimpanzees (Goodall, 1986; Hughes & Cremer, 2007; Wilson, 2000). Stigmas' appearance across human cultures, through human development, and across species suggests that they are wholly, or partially, the product of natural selection and therefore useful. In the lexicon of behavioral ecologists examining behavioral evolution, the question emerges, "Is stigma adaptive?" As Neuberger, Smith, and Asher (2000) argue, stigmas follow one principle: Humans generate stigmas for and stigmatize people presenting a threat to effective group functioning. One major challenge to group functioning is infectious disease (ID); many historical and modern examples highlight stigmas associated with infection. Indeed, evolutionary psychologists argue that all stigmas and stigmatization processes evolved from disease-avoidance mechanisms (Park, Faulker, & Schaller, 2003), highlighting the fundamental connection between disease and stigmas.

We explain how ID stigmas may have evolved and what adaptive function they might have played in a social-living species such as early humans or our close relatives. The next section discusses how human interaction patterns have changed in modern society and the consequences for ID stigmas in the modern age. This article focuses on relational aspects to stigmas and not on identity or cultural perspectives; in other words, it addresses the mesolevel of stigma processes in the framework integrating normative influences on stigma (FINIS; Pescosolido, Martin, Lang, & Olafsdottir, 2008). The thesis of this brief essay is that, although ID stigmas may have served functions for early humans, in modern times, stigmas are no longer adaptive for the human species, much less good.

### **A Brief History of the Intelligent Ape**

This essay is based on the premise that humans, *Homo sapiens*, are the product of natural selection evolving from ape-like ancestors in the same genus (group) called *Homo* to be a singularly inventive species earning the name, intelligent ape (*Homo sapiens*) (Schwartz & Tattersall, 2010). Modern humans appeared between 250,000 and 100,000 years ago in Africa and subsequently moved out of Africa to colonize the world (Henn, Cavalli-Sforza, & Feldman, 2012). Both humans and their close relatives live in groups. (Notably, group communication theories and research [Frey, Gouran, & Poole, 1999] also highlight modern human's focus on groups. This scholarship represents a diverse array of perspectives on human behavior, such as evolutionary, culturally determined, etc.) When animals live in groups, natural selection can act on traits for individuals and for the group (Wilson, 2000). For a solitary organism, self-defense has an evolutionary advantage, so natural selection favors adaptations that lead to improved self-defense. (Notably, for a group living species, personal survival is important but so is the survival of other group members, including relatives [kin selection] and nonrelatives [group selection].) An adaptation associated with living in larger groups with nonrelatives, such as flocks of starlings or shoals of fish, is linked to predator-prey dynamics: As prey live in higher densities, the probability of an individual prey getting eaten decreases (Molles, 2002). Even though each member of the flock or shoal, then, benefits from the safety in numbers

rule and move together, they may only share food with and raise the offspring of relatives. What this emphasizes is that selection acts at different levels, such as the individual, family unit (kin), or group (Bourke, 2011). Importantly, as social defenses for groups evolved, group member conformity became more pronounced (Wilson, 2000). One social defense for groups is stigma.

### **Evolutionary Function of Infectious Disease Stigmas**

A stigma may be defined as “a simplified, standardized image of the disgrace of certain people that is held in common by a community at large” (Smith, 2007, p. 464). As stated earlier, stigmas follow one principle: Humans will generate stigmas for and stigmatize people who present a threat to effective group functioning (Neuberg et al., 2000). For ID stigmas, the disgrace is being infected with a contagious disease. IDs threaten a community’s ability to perform effectively by limiting infected members’ ability to perform their roles within a group or by killing them. Further, IDs inherently tap into the social nature of groups, by spreading from member to member through interaction. IDs can destroy communities and societies as they diffuse through social systems. For example, measles, which emerged in humans about 7,000 years ago (Weiss & McMichael, 2004), creates severe symptoms and death, is transmitted from person to person via respiratory droplets (Atkinson, Wolfe, & Hamborsky, 2012), is credited for destroying the Aztec empire historically and is a significant contributor to current, global death rates (Weiss & McMichael, 2004). Species, then, who created, communicated, and enacted ID stigmas were able to protect uninfected members by identifying those infected with a contagious disease and eliminating infected members’ ability to interact with uninfected members.

In species depending on each other for survival, ostracism from the community meant certain and speedy death from a combination of the infection, from a lack of access to resources (e.g., food), or from a lack of protection from predators (Wilson, 2000). Indeed, predators adapted to social defenses by watching for deviant members failing to participate in social defense strategies, because the deviants become more vulnerable to predation (Wilson, 2000). When humans lived in small, family-based groups, intergroup interactions were less common. Groups mixed for war or trade. Group members transitioned to new kin groups on a more permanent basis for procreation, and these were typically healthy adolescents shifting groups for reproductive opportunities since kin mating is always selected against because of the harmful genetic cost imposed (Wilson, 2000). Thus, ostracized members rarely gain entry into another group. Ostracism, then, eliminated not only further spread through the community but also the disease’s reservoir for future infections.

Even with these functions, ID stigmas did not stem disease-diffusion perfectly for a few reasons. First, sometimes people can be infected with an ID and be infectious (i.e., able to spread it to others) before or without showing symptoms (e.g., human herpes virus; Koelle & Wald, 2000). Human stigmas rely on some sort of mark to identify stigmatized persons; for disease-related stigmas, these are typically visible symptoms (Park et al., 2003). Some IDs, then, may spread before community

members recognize symptom-stigmata and enact stigmatization-based ostracism. Notably, other species, such as lobsters, use other senses that better identify infected, infectious lobsters before they become symptomatic (Behringer, Butler, & Shields, 2006). Second, IDs evolve in reaction to changes in the environment. Pathogens evolve to take advantage of species' contact patterns (Messinger & Ostling, 2009) and can even influence behavioral changes in hosts to promote the transmission (Lefèvre et al., 2009). For example, rabies induces interpersonal aggressiveness and increased salivation (in animals and humans), which both increase viral transmission (Lefèvre et al., 2009).

### **ID Stigmas in Modern Times**

For most of human history we lived in small, family-based groups, but since the advent of agriculture 9,000–11,000 years ago, population sizes have increased as humans transitioned from roaming packs of hunters to settlements (Diamond, 1999). More recently (3,000–5,000 years ago), humans began to build and live in cities, and now, for the first time in history, more than half of humans live in urban landscapes. With cities came booming populations and many mouths to feed, which required increased agricultural productivity. Both high-density living and close encounters with the animals that humans domesticated for food has meant exposure to more infectious diseases (Diamond, 2002; Wolfe, Dunavan, & Diamond, 2007).

In recent history (3000 years), the human population has grown dramatically, transportation has allowed for frequent and widespread contact between humans, and medicines have been developed for treating IDs. For this modern phase, stigmatizing people with IDs does not have the same fitness outcomes as in premodern times because human patterns have changed. Infected persons are unlikely to perish before interacting with others. Even infectious diseases that have been created into biological weapons, like *franscisella tularensis*, take a few days before they create symptoms and kill their host (Centers for Disease Control and Prevention, 2013). People, in general, interact with multiple people a day (Salathé et al., 2010). Further, ostracism from one group may not lead to complete isolation: Infected, ostracized persons' contact may shift to other infected persons and/ or new communities who are unaware of their infection (Goffman, 1963). Indeed, through increased connectivity, ostracized people can find each other and may have quicker bonding based on shared beliefs and ostracism experiences (Herek, 2002). In modern society, ID stigmas are not an effective means to stem the spread of infectious diseases. Further, ID stigmas may make things much worse.

If infected persons shift their interactions to spend more time with each other, they may infect each other with different strains of a disease. Co-infections can lead to new strains that are more lethal and/or more infectious (e.g., Balmer & Tanner, 2011). Through diagnostic procedures, people may learn about an infection and remain infectious before showing stigmatizing symptoms; in order to avoid the pain of stigmatization, infected persons may keep the infection secret (see Smith, 2011 for a review). Even if an infected person can enact enough prevention behaviors to avoid

infecting others, the stress of secrecy and anticipated stigmatization creates biological stress responses (e.g., elevated cortisol; Dickerson & Zoccola, 2013), which comes at the cost of a depressed immune system (Cohen, Janicki-Deverts, & Miller, 2007). Others may avoid testing altogether in order to avoid potential stigmatization, eliminating abilities to access medical treatments for the infection (see Smith, 2011 for a review). Further, hosts (humans) with depressed immune systems may become more infected with an ID, allowing for new strains to develop within a host that are more lethal and/or more infectious.

In addition to complicating disease dynamics, stigmas are persistent: There is no consistent, solid, reliable means by which to remove a stigma (Smith, 2011), even when there is compelling evidence that a person is not (e.g., cured) or never was infected or infectious (e.g., false positive results). The mistakenly stigmatized person still lives with the stress and stigmatization, leaving those persons with the lack of resources and/or social stress that can compromise health and well-being for the rest of their lives. These effects can transfer to the secret-keepers (e.g., children keeping a family secret) as well (Goffman, 1963).

Last, stigma communication is the messages spread through communities to teach members to recognize the stigmatized groups and to enact stigmatization (Smith, 2007, 2011). The content used to facilitate this process includes information about marks by which to identify stigmatized persons, about responsibility for their condition, and about the moral and physical peril linked to them as well as labels for the stigmatized group. Communication about stigmas, then, makes *person*-based attributions salient to the likely exclusion of other explanations. Many infectious diseases are transmitted via a vector (e.g., mosquito, mouse) not from person to person directly. Stigmas around vector-based infectious diseases can generate attention to person-based explanations and person-based responses, which can hinder the recognition of a nonhuman mechanism. For example, when an outbreak of a lethal respiratory infection was noted in the Four Corners area of the United States in 1993, the stigma communication around the Navajo flu quickly spread, and those in the area were subject to stigmatization. The actual illness, hanta virus, was spread via mice; until the mice became the center of attention, the hanta virus continued to spread. The stigma experienced by the Navajo continued long after the outbreak ended (Harper & Meyer, 1999).

## Conclusion

In the modern age, there is no fitness advantage to stigmatizing those infected with IDs. The harms of ID stigmas are significant, and they provide no benefit to public health. Indeed, stigmas may change the environment in which ID pathogens exist, allowing the pathogens to become more problematic than without stigmas in place. Whether viewed through an ethic to do no harm, to protect the public's health, or for social justice, there is no support for stigmatizing infectious diseases and every reason to actively avoid stigma-promoting communication. Social-living species may be particularly apt to create and communicate ID stigmas, and to enact ID-related

stigmatization, due to holdover adaptations that evolved in the precolonizing age. In modern times, ID stigma-related processes no longer function to protect effective group functioning in human communities. Stigmas do not increase the ability of modern societies to survive infectious diseases but, in fact, may be important drivers of problematic disease dynamics and act as catalysts for failures in protecting public health.

### Acknowledgements

Our thanks to Lydia Glick, Amanda Applegate, and members of the Center for Infectious Disease Dynamics for their feedback on this article. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institute on Drug Abuse or the National Institutes of Health.

### Funding

This article was supported by Award Number P50-DA010075 from the National Institute on Drug Abuse.

### References

- Atkinson, W., Wolfe, C., & Hamborsky, J. (Eds.). (2012). *The pink book: Epidemiology and prevention of vaccine-preventable diseases* (12th ed.). Atlanta, GA: Center for Disease Control and Prevention.
- Balmer, O., & Tanner, M. (2011). Prevalence and implications of multiple-strain infections. *The Lancet Infectious Diseases*, *11*, 868–878. doi: 10.1016/S1473-3099(11)70241-9
- Behringer, D. C., Butler, M. J., IV, & Shields, J. D. (2006). Ecology: Avoidance of disease in social lobsters. *Nature*, *441*, 421. doi: 10.1038/441421a
- Bourke, A. F. G. (2011). *Principles of social evolution*. Oxford, UK: Oxford University Press.
- Centers for Disease Control and Prevention. (2013). *Key facts about Tularemia*. Retrieved from <http://emergency.cdc.gov/agent/tularemia/facts.asp>
- Cohen, S., Janicki-Deverts, D., & Miller, G. E. (2007). Psychological stress and disease. *Journal of the American Medical Association*, *298*, 1685–1687. doi: 10.1001/jama.298.14.1685
- Diamond, J. (1999). *Guns, germs, and steel: The fates of human societies*. New York, NY: Norton.
- Diamond, J. (2002). Evolution, consequences and future of plant and animal domestication. *Nature*, *418*, 700–707. doi: 10.1038/nature01019
- Dickerson, S. S., & Zoccola, P. M. (2013). Cortisol responses to social exclusion. In C. N. de Wall (Ed.), *Oxford handbook of social exclusion* (pp. 143–151). Oxford, UK: Oxford University Press.
- Frey, L. R., Gouran, D. S., & Poole, M. S. (1999). *The handbook of group communication theory and research*. Thousand Oaks, CA: Sage.
- Goffman, E. (1963). *Stigma: Notes on the management of spoiled identity*. Englewood Cliffs, NJ: Prentice-Hall.
- Goodall, J. (1986). Social rejection, exclusion, and shunning among the Gombe chimpanzees. *Ethology and Sociobiology*, *7*, 227–236. doi: 10.1016/0162-3095(86)90050-6
- Harper, D. R., & Meyer, A. S. (1999). *Of mice, men, and microbes: Hantavirus*. San Diego, CA: Academic Press.
- Henn, B. M., Cavalli-Sforza, L. L., & Feldman, M. W. (2012). The great human expansion. *Proceedings of the National Academy of Sciences*, *109*, 17758–17764. doi: 10.1073/pnas.1212380109
- Herek, G. M. (2002). Thinking about AIDS and stigma: A psychologist's perspective. *The Journal of Law, Medicine, & Ethics*, *30*, 594–607. doi: 10.1111/j.1748720X.2002.tb00428.x

- Hughes, D., & Cremer, S. (2007). Plasticity in antiparasite behaviours and its suggested role in invasion biology. *Animal Behaviour*, *74*, 1593–1599. doi: 10.1016/j.anbehav.2006.12.025
- Koelle, D. M., & Wald, A. (2000). Herpes simplex virus: The importance of asymptomatic shedding. *Journal of Antimicrobial Chemotherapy*, *45*, 1–8. doi: 10.1093/jac/45.suppl\_4.1
- Lefèvre, T., Adamo, S. A., Biron, D. G., Missé, D., Hughes, D., & Thomas, F. (2009). Invasion of the body snatchers: The diversity and evolution of manipulative strategies in host-parasite interactions. *Advances in Parasitology*, *68*, 45–83. doi: 10.1016/S0065308X(08)00603-9
- Messinger, S. M., & Ostling, A. (2009). The consequences of spatial structure for the evolution of pathogen transmission rate and virulence. *The American Naturalist*, *174*, 441–454. doi: 10.1086/605375
- Molles, M. C., Jr. (2002). *Ecology: Concepts and applications*. New York, NY: McGraw-Hill.
- Neuberg, S. L., Smith, D. M., & Asher, T. (2000). Why people stigmatize: Toward a biocultural framework. In T. F. Heatherton, R. E. Kleck, M. R. Hebl, & J. G. Hull (Eds.), *The social psychology of stigma* (pp. 31–61). New York, NY: Guilford Press.
- Park, J. H., Faulkner, J., & Schaller, M. (2003). Evolved disease-avoidance processes and contemporary anti-social behavior: Prejudicial attitudes and avoidance of people with physical disabilities. *Journal of Nonverbal Behavior*, *27*, 65–87. doi: 10.1023/A:1023910408854
- Pescosolido, B. A., Martin, J. K., Lang, A., & Olafsdottir, S. (2008). Rethinking theoretical approaches to stigma: A framework integrating normative influences on stigma (FINIS). *Social Science & Medicine*, *67*, 431–440. doi: 10.1016/j.socscimed.2008.03.018
- Salathé, M., Kazandjieva, M., Lee, J. W., Levis, P., Feldman, M. W., & Jones, J. H. (2010). A high-resolution human contact network for infectious disease transmission. *Proceedings of the National Academy of Science*, *107*, 22020–22025. doi: 10.1073/pnas.1009094108
- Schwartz, J. H., & Tattersall, I. (2010). Fossil evidence for the origin of *Homo sapiens*. *American Journal of Physical Anthropology*, *51*, 94–121. doi: 10.1002/ajpa.21443
- Smith, R. (2007). Language of the lost: An explication of stigma communication. *Communication Theory*, *17*, 462–485. doi: 10.1111/j.1468-2885.2007.00307.x
- Smith, R. (2011). Stigma communication and health. In T. Thompson, R. Parrott, & J. Nussbaum (Eds.), *Handbook of health communication* (pp. 455–468). New York, NY: Taylor & Francis.
- Weiss, R. A., & McMichael, A. J. (2004). Social and environmental risk factors in the emergence of infectious diseases. *Nature Medicine*, *10*, S70–S76. doi: 10.1038/nm1150
- Wilson, E. O. (2000). *Sociobiology: The new synthesis*. Cambridge, MA: Harvard University Press.
- Wolfe, N. D., Dunavan, C. P., & Diamond, J. (2007). Origins of major human infectious diseases. *Nature*, *447*, 279–283. doi: 10.1038/nature05775

Copyright of Communication Studies is the property of Central States Communication Association and its content may not be copied or emailed to multiple sites or posted to a listserv without the copyright holder's express written permission. However, users may print, download, or email articles for individual use.