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Mosquitoes in the Fields:
Malaria, Farmers and Culturally-Induced Evolution

INTRODUCTION TO NICHE CONSTRUCTION

Ever since Charles Darwin's revolutionary book *On the Origin of Species by Means of Natural Selection* was published in 1859, evolution via adaptation and natural selection has dominated the life sciences. Since the 1980s, a complement to genetic selection has been developed: *Niche construction theory*. Niche construction is the coevolutionary feedback loop in which organisms make modifications (ecosystem engineering) to their local environments (niches)—usually as a non-genetic adaptation—and in which these self-modified environments then exert an evolutionary pressure back onto the organism (Odling-Smee et al. 2003:1-2). Organisms influence their environment and vice versa, but this interaction only recently came to be thought of as playing a role on par with genetics, in spite of it being "far easier to observe individual organisms *doing* niche construction than to observe them *being* affected by natural selection" (Odling-Smee et al. 2003:1).

Because advocates of niche construction argue that it is an evolutionary process in its own right, and not a symptom of natural selection, critics deem it to be too radical (Laland and Sterelny 2006:1751). As Laland and Sterelny posit, however, "not [...] all evolutionary consequential niche construction is under genetic control," (2006:1756) and that we inherit not only our genes, but also a modified environment from our ancestors (2006:1757-1758). Traditional evolutionary theory is solely based on the former while ignoring the (sometimes more important) evolutionary consequences of the latter.

Niche construction has been applied to numerous cases in the life sciences: the rise of biodiversity in the Phanerozoic era (Erwin 2008), the evolution of hominin social intelligence (Sterelny 2007), the plasticity of plants (Donohue 2005), strategies for effective conservation biology (Boogert et al. 2006), the evolution of kin selection and altruism (Lehmann 2007), the relationship between the domestication of plants and animals and large-scale food production (Smith 2007) and countless others.

While niche construction theory can be used to analyze and

understand the evolution of all species (because all species modify they environments in any number of ways), it may be particularly effective for analyzing human evolution because humans in some cases have clear impacts on the environment (e.g., deforestation), while in other cases, they live in predominantly anthropogenic environments such as cities. Niche construction is the process which allows for this local and environmentally-determined flexibility in human evolution.

One of the most well known examples of human niche construction can be found in lactose tolerance and lactase persistence (Odling-Smee et al. 2003:248-250). Milk is high in both caloric count and protein, but for most mammals—including humans—the lactase enzyme in our bodies which allows us to break down the lactose in milk disappears soon after the end of the weaning period, ceasing our ability to digest lactose (Ingram et al. 2009:580). Current thinking suggests that in early human societies in which pastoralism developed, those with a genetic mutation which allowed them to consume milk past infancy (particularly in the highly vulnerable period between the end of weaning and puberty) were more likely to survive than those with lactose intolerance. This is because milk provided them with a high-caloric supplement to their diet, and more importantly a stable food source in between cultivation seasons or in periods of crop failure (Gerbault et al. 2011:865-866). Today, about 35 percent of the world's total population has the genes for lactose tolerance, with the populations largely descended from pastoralist societies from northern and western Europe, the northern Indian subcontinent and throughout Africa (Ingram et al. 2009:580-582, Gerbault et al. 2011:864-865). This tolerance is globally expressed in the human genome in only one place: the *MCM6* gene. The only genetic difference in lactase persistence from, say, an African population to a European population is which specific nucleotide on the gene has the mutation, but the modified gene is the same no matter the population (Gerbault et al. 2011:864). The long association between humans and cattle among these early pastoral cultures provided the opportunity for a random genetic mutation to prove adaptive, but this property was only advantageous *because of* the cultural importance of cattle, demonstrating how this is an example of niche construction and not natural selection.

While lactose tolerance and lactase persistence are a prime example of niche construction this paper argues that a far better example of human niche construction lies in one of the deadliest and most virulent pathogens in human history: Malaria. While pastoralism and its resulting genetic modifications popped up all over the world, malaria and its

resulting genetic modifications are much more localized and more of a product of highly specific environments. Although the definition of what constitutes a true niche has long been argued and yet to be settled (as Lewontin puts it, “there is a non-countable infinity of ways in which [...] to describe an ecological niche” [2001:49]), almost all definitions agree that an organism’s niche is specific to it, and precludes organisms that it could out-compete or that could out-compete it (Odling-Smee 2003:37-42). Lactose tolerance is certainly niche construction, but human’s genetic adaptations to malaria, comparatively, are much more varied in their scope and genetics, and even more strongly influenced by individual niches and niche construction.

A MALARIA PRIMER

Evidence from amber-preserved mosquitoes shows that the malaria causing parasites *Plasmodium* have been around for at least 15-45 million years (Poinar 2005:47). Some say *Plasmodium* parasites evolved as early as 150 million years ago (Escalante and Ayala 1994:11376-11377). It is thought that the disease first began infecting and killing humans in large numbers around 10,000 years ago, with the advent and rise of agriculture and irrigation (Hedrick 2011:284). Human populations eventually evolved various genetic adaptations (sickle-cell and thalassaemia being the most prevalent) giving them resistance to malaria, with the earliest found widespread evidence of this coming from the Roman Empire around 2000 BCE (Sallares et al. 2004:322-326).

Figure 1. Global distribution of malaria before modern human intervention.
Reproduced from Rees et al. 2010:2022.

Malaria remained endemic worldwide until the mid-twentieth century (see

Figure 1 above), but now is only prevalent in sub-Saharan Africa, Asia and Central/South America (Hedrick 2011:284, NIH 2000:12), with about half a billion new cases and 3 million deaths every year (Kwiatkowski 2005:171, Suh et al. 2004:1693).

Background

Malaria in humans is caused by four species of the parasite *Plasmodium*, with the most lethal being *P. falciparum*. They all, however, have similar life cycles. The cycle begins in the salivary glands of the *Anopheles* mosquito, where the parasites sexually reproduce. When a female *Anopheles* feeds off of a human, the infectious sporozoites enter the bloodstream, causing what is termed a "blood-stage infection" (Wielgosz et al. 2012:1). Once the parasite enters the human bloodstream, it migrates to the liver, where it covers itself in liver cell membranes to avoid detection by the immune system (Cormier 2011:36).

The *Plasmodium* then asexually multiply in the liver cells, which burst when filled with enough of the parasites (and then can enter a mosquito's salivary glands when she feeds off an infected human). These parasites explode into the body in waves, infecting red blood cells, and in these red blood cells the process of asexually reproducing and then bursting the cell is repeated (Cormier 2011:36-37, Cowman and Crabb 2006:755-757). The periodic waves of parasites breaking out of the liver is what causes the cyclical nature of the fevers and chills characteristic of malaria (NIH 2000:13, Cormier 2011:36). Without treatment, once enough of the body's red blood cells are broken, oxygen can no longer be transported through the body in sufficient concentration, and the "corpses" of these burst cells begin to clog arteries in the brain and kidneys, further cutting off the body's essential oxygen supply. The loss of red blood cells generally results in the victim becoming anemic and hypoglycemic, which can, as stated by Cormier, "lead to renal failure, pulmonary edema, cerebral anoxia (in cerebral malaria), and death," (Cormier 2011:37, NIH 2000:13, Humphreys 2001:9-10).

As stated above, malaria became humanity's first massively lethal disease about 10,000 years ago, when human populations began becoming sedentary. It is thought that human agricultural practices made *Anopheles* mosquitoes more likely to feed on human hosts than nonhuman hosts (Porta 2014:8), which meant there were more malaria transmissions, which likely led to the more aggressive *Plasmodia* being evolutionarily selected for (Evans and Wellems 2002:404). Thus, the rise

of agriculture—a cultural adaptation—fueled population growth, which increased both the need for irrigation (providing the perfect breeding ground for mosquitoes) and the population density (allowing one mosquito to transmit malaria to more human hosts) (Etkin 2003:311-312). With this higher vulnerability to and rates of malaria, genetic adaptations providing resistance to malaria were more likely to be positively selected for. Even though a human population may experience initial decline, the selection of these adaptations will eventually lead to an increase in population, starting the whole cycle over again. This is exactly the kind of feedback loop best explained by niche construction: Human populations modified their local environment with agriculture and irrigation, and—through the rise in the mosquito population and the selection of more aggressive *Plasmodia*—the newly constructed niche then exerted an evolutionary pressure back onto the human population.

Human Genetic Adaptations to Malaria

Malaria is sometimes called one of the first—if not *the* first—selective evolutionary pressures by disease exerted on the human species (Etkin 2003, Escalante and Ayala 1994, Evans and Wellems 2002). The most common and well-known human genetic mutations caused by malaria are sickle cell disorders and thalassemia, both of which are hemoglobin[1] disorders which originated in areas of the world where malaria was endemic for much of human history (the Mediterranean, the Middle East, Southeast Asia, the Caribbean and Africa) (Kwiatkowski 2005:171, Anionwu and Atkin 2001:8).

Sickle cell disorders are a series of hemoglobinopathies[2] (which includes sickle cell anemia) that cause—under certain stressors—the hemoglobin proteins in red blood cells to undergo what is called polymerization, in which they become rigid and deoxygenated. This, in turn, causes the red blood cell to go from it's round, flexible disc shape to a rigid, crescent/sickle shape (Rees et al. 2010:2018-2021). Let us say the gene for a usual hemoglobin type is "H," and the gene for sickle cell is "S." Someone who did not inherit sickle cell from either parent would have a genotype of HH. The genotype for someone with sickle cell would be SS,

[1] Hemoglobin is the oxygen-carrying protein component of red blood cells, which also gives them their color and flexibility (allowing them to easily squeeze through small arteries) (Anionwu and Atkin 2001:11).

[2] hemoglobinopathy: "any one of a group of genetic disorders caused by or associated with the presence of one or several forms of abnormal hemoglobin in the blood" (Venes 2009:1109)

meaning they inherited one sickle cell gene from each parent. In contrast, a carrier's genotype would be SH (one regular hemoglobin gene from parent X and a sickle cell gene from parent Y). The carrier would not exhibit a sickle cell disorder, because under stressors only *some* of their red blood cells would sickle and harden (Anionwu and Atkin 2001:8-13, Rees et al. 2010:2020-2023, Wailoo and Pemberton 117).

It has been known since at least the 1950s that sickle cell traits emerged in areas where malaria was endemic 5000-10,000 years ago (the same time period agriculture came about in those areas) (Livingstone 1958:536-541, Hedrick 2011:284). In those areas today, up to 45 percent of the population has the sickle cell gene (see Figure 2 below) (Anionwu and Atkin 2001:11). Being a sickle cell carrier (SH heterozygous) has been shown to confer genetic resistance to malaria (Anionwu and Atkin 2001:11, Cormier 2011:138, Wailoo and Pemberton 2006:117, Hedrick 2010:285-286, Livingstone 1958:534). If one is homozygous either for sickle cell or not (SS or HH), there is no sickle cell resistance to malaria (Anionwu and Atkin 2001:11). Essentially, having even more sickled cells actually puts you at a disadvantage. The mechanism for why being a sickle cell *carrier* makes one's red blood cells more resistant to malaria is not quite known, but Hedrick theorizes it could be "that the growth of malarial parasites is suppressed in sickle cells" (2010:287). No matter how it works, sickle cell was selected for because of the intense evolutionary pressure malaria so quickly exerted on the human species due to our own cultural practices.

Figure 2. Global distribution of the sickle-cell allele HbS (simplified to S in this paper). Reproduced from Rees et al. 2010:2022.

Thalassemias, on the other hand, are a condition in which the red

blood cells do not produce enough hemoglobin, which means the red blood cells cannot function properly and only live a short amount of time. This decreases the body's red blood supply overall. This can lead to anemia or even death within the first thirty years of a person's life (CDC 2014). That, however, is only if—like with sickle cell—you are homozygous for the trait. If one is heterozygous, one has a distinct advantage and resistance to malaria (Luzzi et al. 1991:785-786). Although not as well researched as sickle cell, some more recent studies have shown that actually having the homozygous forms of certain thalassemias can protect a person from certain forms of malaria (Williams et al. 2005:369). Unlike sickle cell, the thalassemias emerged in the Mediterranean, the Middle East and south Asia, and are found among populations descended from these areas 5000-10,000 years ago (see Figure 3) (CDC 2014, Hedrick 2010:285, Wailoo and Pemberton 2006:117).

Figure 3. Global distribution of thalassemia.
Reproduced from Chernoff 1959:903.

Yam and Rice Cultivation Among the Kwa

One part of the world in which the relationship between agriculture and malaria resistance provides a clear example of human genetic

adaptation to a characteristic of a modified environment is among the Kwa-speaking peoples of West Africa. There are three main groups: Yam, rice and millet cultivators, and each occupies their own ecologically unique part of the region, reflected in their linguistic differences (Durham 1991:127-129). The entire region has experienced malaria endemism for thousands of years, and thus we see sickle cell having emerged as an adaptation to this selective pressure in all three populations. The frequency, however, is different depending on the agricultural practices, as discussed in further detail below. Durham used regression analyses to show that sickle cell had come first to the yam growers, as the high levels of standing water created from clearing the forests for yam cultivation brought *Anopheles* mosquitoes and malaria to them very rapidly (1991:137, Leland et al. 2000:137). There is even a strong correlation between the rain cycles (and thus what month someone was born in) and the frequency of sickle cell genes (Durham 1991:137-139). There are, naturally, higher mosquito populations in rainier months, which is why we see the increase in sickle cell alleles among individuals born around those times of year (see Figure 4 below).

Rice growing, however, came to the Kwa-speaking people later than yam cultivation, according to studies cited by Durham completed by Frank B. Livingstone and Roland Portères (1991:135-137). Because of this, we see both a lower frequency of sickle cell alleles and a weaker correlation between monthly rainfall and said alleles among rice-growing populations (Durham 1991:139-140). Durham also realized that, genetically, the rice growers are much more isolated from their yam growing neighbors, and are "relatively exempt from the genetic selection pressures of malaria operating there." He concludes from this that it is the difference in their agricultural practices (growing yams versus rice) which "through their influence on the local density of mosquito vectors [parasite carriers], are responsible for the variable course of genetic evolution in these two groups of population" (1991:140). We see this below in Figure 4, which again shows the relationship between average monthly rainfall (positively correlated with malaria transmission) and the frequency of the heterozygous sickle cell genotype (here named "Frequency, q, of S allele" on the y-axis) of persons born in those months. We see in the graph a comparison between the yam-growing (solid circles) and the rice-growing (open circles) Kwa: The yam growers have much higher frequencies of the sickle cell gene, following a predictable pattern (shown by the line-of-best-fit overlaid on the graph) correlated to monthly rainfall, while the rice growers have a much lower and more random frequency overall.

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Figure 4. Relationship between monthly rainfall and sickle cell allele frequency among yam (solid circles) and rice (open circles) growing Kwa speakers in West Africa.. Reproduced from Durham 1991:139.

CONCLUSION

The genetic differences caused by localized agricultural practices among the Kwa are just one example of malaria as human niche construction. Both this and lactose tolerance discussed above demonstrate how niche construction cannot cause species-wide evolution over the course of millions of years. It is the faster, more localized evolutionary process showing that humans can affect their own evolution via the cultural choices we have made and continue to make. Niche construction does not discount or take away from natural selection. Rather, it fills in the gaps natural selection cannot explain as well. Natural selection is the evolutionary process in which advantageous genetic mutations best allow a species to thrive in the long term, but it occurs over the course of thousands of generations and millions of years. It is, however, an incomplete explanation for genetic mutations made advantageous by human cultural practices. These are the kind of mutations which emerge from niche construction that do not affect the

entire species, but rather pockets of it, with as much variability as the cultural practices themselves. Niche construction is both an alternative and a complement to natural selection, but it is still a separate evolutionary process. What this paper did not show, though, was how the effects of niche construction can be measured in other ways (which do require further investigation), harvest yields and measures of fitness other than survival among them. Niche construction theory is still only twenty years old and must be further studied and honed in order to be recognized on the same level as natural selection. As it gains traction outside of biology, it can become an important tool in fields such as anthropology and other social sciences.