

## **The Effect of Land Use Change on Exposure to and Transmission of Pathogens**

### **I. Introduction**

According to WHO, within the last 20 years, at least 30 new diseases have emerged to threaten the health of hundreds of millions of people (McMichael 2004). Most of these emerging infectious diseases (EIDs) are zoonotic, as catalogued by the Institute of Medicine (Daszak et al. 2001). Understanding the way in which human interactions with the environment facilitate vector-host interactions may improve preventative measures and public health practices. Human-induced environmental change plays a major role in the emergence of zoonotic diseases (McMichael 2004, Eisenberg et al. 2007), often because these changes increase human-vector interactions (McMichael 2004). Malaria is the world's most prevalent vector-borne parasite, and in 2011 killed an estimated 655,000 people (WHO 2011). Understanding the environmental conditions that facilitate malaria outbreaks may give insight on effective practices or initiatives to eradicate malaria.

Roads not only create the environmental conditions needed for malaria outbreaks (Chomintz & Gray 1996, Olson et al. 2010), but also facilitate the spread of disease (Arroyo et al. 2006, Heitman 2000), such as malaria (Martens & Hall 2000). In the following sections, I will discuss the impact roads and road-induced environmental change have on the emergence, transmission and spread of malaria. First I will introduce the framework required to analyze this puzzle; then I will introduce the existing research on road construction/presence and environmental change, environmental change and infectious disease, and connectivity and disease transmission; third I will briefly describe my methods; and finally I will synthesize these connections and my research into a coherent description of how roads facilitate malaria's path from parasite to vector to human host and its subsequent transmission and spread.

### **II. Framework**

A combination of land change science and epidemiology presents the most applicable framework for my research on how the socio-environmental impacts of roads collectively create environmental conditions that facilitate the emergence of microbes as

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well as the social and connectivity conditions that increase transmission rates. Eisenberg et al. (2007) present a framework called the Environmental Change and Infectious Disease (EnvID) framework that allows for such integration. This framework “uses a systems theory structure to integrate and analyze disparate information from a variety of disciplines” (p. 1216) and incorporates the knowledge obtained from EnvID-based studies into research on and control of infectious diseases (Eisenberg et al. 2007). To illustrate my aforementioned research topic, I will use this combined land change science (LCS) and epidemiology framework to explain the likelihood of progression from road construction to anopheline mosquito habitats to malaria outbreaks. These connections are present in disparate sources of current research (Turner et al. 2007, Patz et al. 2000, Patz et al. 2004, Wolfe et al. 2000, Arroyo et al. 2006), but none of these sources go so far as to connect road construction to disease emergence and transmission.

Alternative frameworks used in these and similar analyses, such as public health, frameworks for climate change, globalization and environmental health (Eisenberg et al. 2007), do not suffice to encompass all facets of my research topic. In addition, while the EnvID framework shares similarities with LCS, in that both frameworks incorporate the effects of environmental impacts into their analyses, EnvID does not consider the underlying causes of these environmental impacts in the way land change science does. Therefore, the completion of my analysis requires the use of the EnvID framework to integrate aspects of land change science and epidemiology. Furthermore, in order for the reader to understand this framework, I must explain why LCS and epidemiology are the appropriate frameworks for this analysis.

Epidemiology’s incorporation of the study of both the distribution and the determinants of infectious disease (WHO 2012) make it the appropriate framework for the microbial and transmission aspects of my analysis. Furthermore, epidemiology is important because of its application to the control of diseases (WHO 2012), which is also a potential benefit of studies that use the EnvID framework (Eisenberg et al. 2007) and thus one of the reasons I believe my research is worth pursuing. However, epidemiology’s broad interpretation (Rothman et al. 2008, World Health Organization), requires specification for the purposes of this paper. WHO’s (2012) definition of epidemiology covers a broad range of health problems including, but not limited to,

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diseases. Indeed, Rothman et al. (2008) agree, “The scope of epidemiology has become too great for a single text to cover it all in depth” (p. vii). However, due to a “surge of epidemiologic activity in the late 20<sup>th</sup> century” (Rothman et al. 2008, p. vii), ‘epidemiology’ has a somewhat coherent definition. As WHO (2012) defines the term, “Epidemiology is the study of the distribution and determinants of health-related states or events,” including disease. For the purposes of this analysis, the “health-related state” is a malarial infection, and the “event” is the prevalence and transmission of this disease. According to WHO (2012), surveillance and descriptive studies can be used to study distribution, and analytical studies are used to study determinants. My research employs case studies of both types. For example, Patz et al. (2000) and Arroyo et al. (2006) use and reference surveillance and descriptive studies to assess distribution of emerging parasitic diseases and the prevalence of the HIV virus in a defined area, respectively. On the other hand, Wolfe et al. (2000), McMichael (2004), and Daszak et al. (2001) conduct and reference analytical studies to focus on environmental change as a determinant in disease emergence. In addition, Patz et al. (2004) and Eisenberg et al. (2006) appear to combine study of determinants and distribution; this is likely why Eisenberg et al.’s (2007) paper on the EnvID framework cites both these articles. Therefore, both sides of epidemiology are vital to consider in my analysis of a microbe’s path from road-induced environmental disturbance to infection to outbreak.

LCS strives to understand, assess and predict every potential outcome and impact associated with any given type of human-generated land use change (Turner et al. 2007), and therefore is useful in examining the road-induced environmental disturbance aspect of my analysis. Land change science’s broad span of goals means the framework must inherently consider the perspectives of various different fields—biological, geographical, and social, to name a few (Turner et al. 2007). The EnvID and LCS frameworks share this interdisciplinary nature (Turner et al. 2007, Lambin 2001, Eisenberg et al. 2007), and thus the integration of LCS, an interdisciplinary framework, into the analysis, further enriches the interdisciplinary nature of the overarching EnvID framework. As part of my analysis, LCS will help identify the variety of consequences that come from road construction and presence (Lambin 2001). A thorough examination of such consequences

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will help identify which of the environmental changes that cause disease emergence (Patz et al. 2000) result from road construction.

Both LCS and epidemiology are vast frameworks and therefore difficult to surmise into individual, compact theories (Rothman et al. 2008, Turner et al. 2007). However, certain overlaps and supplementary aspects of each, as explained above, make these two frameworks seem appropriate for my analysis. The EnvID framework has parameters such that these overlaps and aspects may be combined into one applied framework and used in emerging infectious disease research. This combination is possible because the main goal of the EnvID framework is to “identify knowledge gaps” within the “plausible relationships between proximal environmental characteristics and transmission cycles” (Eisenberg et al. 2007, p. 1216). One such knowledge gap, which I intend to fill, is the path of a malarial pathogen from an initial road-related disturbance through to an established outbreak. As land change science strives to compile data on every potential consequence of anthropogenic land use, so does the EnvID framework attempt to identify and assess every potential way in which anthropogenic environmental change can affect pathogen disturbance and proliferation and disease emergence and transmission. In order to be integrative but remain somewhat focused, “the EnvID encompasses three interlocking components: environment, transmission, and disease” (Eisenberg et al. 2007, p. 1218). To articulate the breadth and flexibility of the EnvID framework, Eisenberg et al. (2007) created a matrix (Figure 1) to address all possible causes and outcomes of environmental change-driven disease emergence and transmission:

Proximal Environmental Characteristics		Population Size				Organism Properties		Human-Human Contact		Human-Vector Contact		Human Environment Contact				
		Pathogen	Vector	Host	Human	Pathogen Virulence	Host Immune Status	Sexual Contact	Close Contact	Biting rate	Host Seeking	Water ingestion	Dermal Contact	Inhalation	Food Ingestion	Biting
Water	Presence/Absence															
	Flow Rate															
	Nutrient Content															
	Chemical Composition (Inorganic Pollution)															
	Fecal Matter Content (Organic Pollution)															
Climate	Salinity															
	Turbidity															
	Temperature															
	Precipitation															
	Humidity															
Air	Cloud Cover/Sunlight Exposure															
	Wind Speed															
Animals	Particulates															
	Chemical Composition															
	Diversity (Non-Domesticated)															
	Magnitude (Non-Domesticated)															
	Density (Non-Domesticated)															
Plants	Pets															
	Livestock															
	Diversity (Non-Domesticated)															
Genetic	Magnitude (Non-Domesticated)															
	Density (Non-Domesticated)															
	Crop Cover															
Human Practice	Prevalence of antibiotic resistance genes															
	Prevalence of GMO genes															
	Prevalence of virulence factors															
Human Environment	Migration															
	Travel															
	Human Contact with Environment															
	Physical Barriers to Exposure															
Human Environment	Living Structures															
	Sanitation Infrastructure															
	Density															
	Other Structures															

Figure 1: Matrix for mapping the relationship between proximal environmental characteristics and transmission cycles (Eisenberg et al. 2007).

This matrix visually expresses the knowledge gaps in a comprehensive structure of determinants and distributors of infectious disease. Eisenberg et al. (2007) hoped the matrix and framework would “help elucidate the necessary interdisciplinary research elements and approaches needed to study environmental impacts” on disease, as it was applied in an Eisenberg et al. (2006) study to explore the effects of road development on diarrheal disease transmission in Ecuador.

Thus, this matrix and framework “can be used to assess all possible impacts of environmental factors on a single infectious disease” (Eisenberg et al. 2007, p. 1221). In addition, the EnvID framework can be used to explore “the impacts of distal environmental change on a particular disease” (Eisenberg et al. 2007, p. 1221). For the purposes of my research, I will focus on environmental factors that, according to LCS-based research, result from road construction and presence. Using epidemiology, I will evaluate how these particular environmental factors facilitate the emergence and spread of disease. This connection falls under the umbrella of an EnvID framework and will be applied to my research “so that knowledge about environmental change can be

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incorporated appropriately into the study and control of infectious diseases” (Eisenberg et al. 2007, p. 1216)—the core goal of EnvID.

### **III. Literature Review**

In pursuit of this goal, I will review the literature on the emergence and transmission of disease in relation to environmental change. According to Patz et al. (2004), “anthropogenic land use changes drive a range of infectious disease outbreaks and emergence events and modify the transmission of endemic infections” (p. 1092). This occurs because incessant disturbance of natural ecosystems by destructive changes in land use, especially deforestation, have “modified the distribution and behaviour of parasites, their hosts and vectors” (Patz et al. 2001, p. 8). Eisenberg et al. (2007) argues for the “pressing need” required to “clearly define the causal relationships” (p. 1) between anthropogenic environmental change and disease transmission cycles. An understanding of these causal relationships may lead to new, more effective public health policies for controlling certain diseases. Further, if roads are found to be the culprit for a large part of EID-inducing environmental change, there may be implications for future road construction into and through remote areas. On an epidemiologic level, knowledge of this causal relationship could reveal implications for the prevalence, emergence and transmission of various pathogens (Daszak et al. 2001). Knowledge as to how pathogens are extracted from the environment is key to such understanding, and a large part of the answer lies in vector-host relationships (Eisenberg et al. 2006). Certain environments create a broad niche for pathogen-carrying vectors of interest (Patz et al. 2000, McMichael 2004). Awareness of how certain vectors flourish in deforested environments may lead to initiatives to control deforestation and other environmental changes that create these ideal habitats. Such initiatives may benefit not only public health, but ecological processes and ecosystems as well. Understanding of these causal relationships may greatly benefit both human and ecosystem health. To that end, I have compiled research on current knowledge regarding the impact of roads on environmental change, and the subsequent impact of environmental change on EIDs. In the following section I will introduce the literature on the environmental impacts of roads; the effect of deforestation, habitat disturbance, and climate on the malaria parasite’s vectors; the effect

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of road connectivity and proximity on disease transmission; the global malaria problem; and the characteristics of the malaria vector and its habitat.

Laurance et al. (2009), Forman & Alexander (1998), Chomintz & Gray (1996) and Patz et al. (2004) outline the various environmental and social impacts associated with road presence and road construction. New roads into forested areas lead to deforestation and habitat fragmentation (Forman & Alexander 1998, Chomintz & Gray 1996, Lambin 2001), as well as facilitate human mobility and allow increased access for humans to previously inaccessible areas (Patz et al. 2004). All three of these consequences may present the possibility for new pathogens to emerge from zoonotic sources. Road construction also increases erosion, which further degrades the surrounding environment and ecosystem and has exacerbated upsurges in malaria (Patz et al. 2004). Roads also encroach on the natural landscape and thus have the potential to increase the amount and location of human activities (Laurance et al. 2009).

Deforestation occurs with road construction, and the migrant flow that follows new road construction leads to deforestation along roadsides (Campbell 2005). According to McMichael (2004), “Deforestation, with fragmentation of habitat, increases the ‘edge effect’, which then promotes pathogen-vector host interaction” (p. 1054). Indeed, Patz et al. (2000) found that in parts of Latin America, deforestation has led to an increase in leishmaniasis. Sweat (2006) hypothesizes deforestation has destroyed the remoteness of certain locations, thus disarming our greatest defense against zoonotic pathogens—separation. Patz et al. (2000) call deforestation “one of the most disruptive changes affecting parasitic vector populations” (p. 2), likely because deforestation’s ecological effects lead to habitat destruction and fragmentation, climate change, and encroachment.

The divisive and destructive activities associated with roads lead to habitat destruction and fragmentation. Habitat disturbances are important to EIDs because “intact forests support complex ecosystems and provide essential habitat for species that are specialized to those flora and that may be relevant to our health. If these complex relationships are disrupted, there may be unforeseen impacts on human health” (Patz et al. 2000, p. 1097). Specifically, this destruction and division leads to greater lengths of forest edges, which cause certain unadapted species to vacate their niches and thus allow other, more adapted species to colonize along the edges of the forest (Patz et al. 2000).

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These species can be invasive, benign, or disease-carrying vectors. For example, “cleared lands are generally more sunlit and prone to the formulation of puddles with more neutral pH which can favor specific anopheline larvae development” (Patz et al. 2000, p. 1093). Changes in habitat can allow disease-carrying vector populations to flourish along the edges of forests, which puts them into close contact with human populations.

In addition to the physical land changes roads cause, roads contribute to both local and global increases in temperature, which can influence the geographic range of a vector population (Laurance et al. 2009, McMichael 2004). Dark asphalt attracts sunlight, while openings in forest cover attract sunlight and allow more heat to reach the surface. Canopy openings remove shade-loving species from the area and allow new species to move in. Climate change not only affects niches locally, but rising temperatures and the associated changes in weather patterns allow certain pathogens and their vectors—such as malaria and anopheline mosquitoes—to expand to new geographic ranges (Sweat 2006). The World Health Organization (WHO) (2012) found this increased and broadened presence and survivorship of mosquitoes to have a direct impact on humans—WHO estimated that in 2004, approximately “6-7% of malaria in some parts of the world is attributable to the climate change that has occurred during the past quarter of a century” (McMichael 2004, p. 1051). Therefore, road use and the deforestation associated with roads both initiate processes that result in global warming. Climate change and its ensuing effects then lead to changes in disease emergence.

In addition to ecological consequences, road connectivity allows for the spread of diseases, more so today than ever before (McMichael 2004). For example, Preston (1994), Heitman (2001) and Wolfe (2000) argue the theory that the Kinshasa highway catalyzed the spread of HIV-1 throughout the sub-Saharan Africa, and subsequently to the world Arroyo et al. (2006) found populations living closer to major roads rather than secondary roads had higher rates of HIV-1 incidence and genetic complexity. The correlation of road proximity and incidence contains many implications that must be further studied due to this correlation’s potential to improve public health practices (Eisenberg et al. 2006, Arroyo et al. 2006). Meanwhile, the increased genetic complexity of HIV-1 within populations indicates the difficulty of finding ways to treat or vaccinate the virus. While HIV-1 is a prime example of road-induced increased transmission, its

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initial transference from non-human primate to human could have been a singular incident. Thus, to demonstrate the full process of how roads increase pathogen flow from the wild to other populations requires a zoonotic disease with a well-researched vector species.

One such disease is malaria. WHO (2011) identifies five species of the *Plasmodium* genus that cause malaria in humans. For humans, the most deadly of these pathogens is *Plasmodium falciparum*, which predominates in Africa (WHO 2011) and is responsible for nine out of every ten malaria deaths (Wellcome Trust 2012). *Plasmodium* pathogens spread to humans through the bite of a female Anopheles mosquito. More than 30 species of anopheline mosquitoes are capable of transmitting malarial pathogens to humans, and many attribute malaria's epidemiological success in part to the disease's high rate of transmission (Lacroix et al. 2005). Early models of epidemiology cite the parasite's life cycle and development, the life span of the mosquito, and the mosquito's biting rate as main causes of this intense transmission (Lacroix et al. 2005). Indeed, malaria is only known to be transmitted in subtropical and tropical regions where anopheline mosquitoes can survive and reproduce, and malaria parasites can complete their growth cycle in the mosquitoes (CDC 2012).

Extensive research exists on malaria, and for the purposes of this paper, ideal breeding grounds for anopheline mosquitoes are of particular interest. Research shows malaria flourishes in tropical regions, particularly Africa, where the pathogen is essentially omnipresent (Patz et al. 2000) and continually resurgent (McMichael 2004). In 2010, an estimated 81% of malaria cases and 91% of deaths occurred in sub-Saharan Africa, where people have the greatest risk of contracting the disease (WHO 2011). The global presence of malaria presents a problem, as the Center for Disease Control classifies malaria as an EID pathogen that has evolved drug resistance (Daszak et al. 2001). In addition, many malaria vectors and parasites are sensitive to changes in the ecology of their habitat (Patz et al. 2000). The environmental changes associated with anthropogenic activities facilitate the success of *r* species—small, opportunistic species that “reproduce rapidly, invest in prodigious output rather than intensive parenting, and have mechanisms to efficiently disperse their offspring” (McMichael et al. 2004, p. 1052). In contrast, *K* species, such as humans, tend to be larger, yield few offspring, and

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are long-lived (McMichael et al. 2004). Pathogens are typical to *r* species, and “live today in a world of increasing opportunity” (McMichael et al. 2004, p. 1052). Essentially, anopheline mosquitoes survive and thrive in deforested areas. Following deforestation, the most competitive mosquito species who ends up dominant depends on the type of land use activity, human settlement and ecological context of the area (Patz et al. 2000).

Furthermore, “The vegetation that supplants the previously forested area frequently promotes an increase in malaria prevalence” (Patz et al. 2000, p. 5). A case study in Malaysia found repeated resowing of rubber plants over a 50-year period coincided with cyclic malaria epidemics (Patz et al. 2000). A case study in Trinidad found, following deforestation, bromeliad plants replaced original vegetation, and the water-collecting bromeliads were preferred breeding sites for anopheline mosquitoes, and a malaria epidemic ensued (Downs & Pittendrigh 1946, Patz et al. 2000). When the bromeliads were removed, malaria prevalence decreased to its previous level (Downs & Pittendrigh 1946, Patz et al. 2000). Therefore, malaria vectors and malaria incidence are highly responsive to environmental change.

Laurance et al. (2009), Forman & Alexander (1998), Chomintz & Gray (1996) and Patz et al. (2004) have meticulously researched the various environmental impacts of road presence and road construction. Patz et al. (2000), Eisenberg et al. (2006), Daszak et al. (2001), Sweat (2006) and McMichael (2004) have discussed the implications anthropogenic environmental change has for disease emergence. Wolfe (2000), Preston (1994) and Heitman (2001) outline the role mankind’s catalyzed connectivity has played in the spread and transmission of disease within geographic regions and between various continents. What this collection lacks is a solid, cyclical story of how a disease is brought out of the wilderness due to the environmental changes roads cause, and is subsequently spread to other areas and populations due to the connectivity the road facilitates. With my research on malaria in Africa, I will synthesize information on how roads create the ideal habitat for anopheline mosquitoes to breed and flourish, and how road connectivity allows infected individuals to move between populations and spread their disease to other vector mosquitoes and subsequently to other individuals. This demonstration of how this process can and often does begin with a road is one not seen in any singular literature source, and it is necessary to outline the vector-host transmission and subsequent spread

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of a disease through an EnvID framework. Thus far, most research in “public health scholarship has focused on the link between transmission cycles and disease burden” (p. 2) and environmental sciences have focused on ecological consequences that arise from environmental change (Eisenberg et al. 2007). Furthermore, WHO (2011) recommends insecticide and mosquito nets as methods for malaria prevention, but does not mention any initiatives to control the anthropogenic activities or environmental changes that facilitate the creation of mosquito habitats. According to Daszak (2001), understanding of impact of human environmental change on wildlife populations is clear, but to understand role of these changes in disease emergence requires an integration of diverse branches of biology. Thus, I will attempt to integrate the various branches of biology covered in my research to answer the question—how do road-induced environmental changes and road-associated social patterns and connectivity initiate and exacerbate the emergence and spread of EIDs?

#### **IV. Methods**

This semester our class set about investigating the socioenvironmental impacts of road and rail. I have an interest in epidemiology and disease, so I researched the way in which roads affect the spread and emergence of disease, with a focus on the malaria virus due to the malaria vector’s responsiveness and sensitivity to environmental change. Because malaria is a complex disease—the pathogen comes in different forms, can be drug-resistant, and occurs only under certain circumstances—and the world’s malaria prevalence is a current problem, I pursued articles that explain the ideal circumstances for malaria pathogen survival and the nature in which the disease spreads from host to host. Hopefully, my research will shed light on possible environmentally-focused preventative measures in addition to WHO’s (2011) recommendations of insecticides and mosquito nets. In this paper, I will review and analyze the sources that I found most helpful to my research while drawing from disease and epidemiology-related case studies relevant to zoonotic pathogens and viruses.

#### **V. Analysis**

This paper's review of the literature finds connections between: a) Road construction/presence and environmental change; b) Environmental change and infectious disease; and c) Connectivity and disease transmission. However, no single report or case study illustrates the path of a disease from zoonotic origin through anthropogenically-induced emergence and spread, despite Patz et al.'s (2000) statement that "Ecological disturbances exert an influence on the emergence and proliferation of malaria and zoonotic parasitic diseases" (p. 1). In this analysis, I will expand on this assertion using the connections I have found in my readings and discuss how road construction, use of roads, and road presence create the ideal environmental conditions for the vector-to-human transmission of malaria and facilitate additional spread of said disease.

### **Roads and the Mosquito Habitat**

The deforestation and habitat destruction inherent to road construction (Chomintz & Gray 1996, Forman & Alexander 1998) affect infectious disease risk (Patz et al. 2005) and play a major role in creating an ideal habitat for anopheline mosquitoes (McMichael 2004, Patz et al. 2000). According to Patz et al. (2005), road building and logging are two main causes of this deforestation and habitat destruction. Road building leads to deforestation from both its construction (Forman & Alexander 1998) and the clearing that occurs along roadsides after a road opens (Patz et al. 2000). As Patz et al. (2000) argued, this clearing is problematic for pathogen control because vegetation and habitat conditions in the newly deforested area render cleared areas a more suitable mosquito habitat than forested areas. Furthermore, the introduction of roads has been found to increase the transmission rate of diarrheal pathogens in Ecuador due in part to the subsequent increase of stagnant pools (Eisenberg et al. 2006). Stagnant pools not only provide a suitable environment for bacterial prokaryotic fission; these pools also offer a breeding ground for mosquitoes and subsequently serve as a nest for mosquito egg masses (Patz et al. 2000). Cleared areas and roadside ditches that collect water provide "far more suitable breeding sites for malaria-transmitting anopheline mosquitoes than forest" (Patz et al. 2005, p. 402) because the neutral pH puddles that form favor anopheline larvae development (Patz et al. 2000). Therefore, deforested landscapes in

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tropical and subtropical areas with heavy rainfall are more likely to provide the warm, neutral pH puddles that provide a suitable environment for the survival and reproduction of anopheline mosquitoes.

In addition, when road building and deforestation cause habitat destruction, the subsequent loss of biodiversity opens previously occupied niches that pathogen-carrying vectors, such as anopheline mosquitoes, can overtake (McMichael 2004, Patz et al. 2000, Patz et al. 2005). Anthropogenic environmental changes such as deforestation create conditions more suitable for the survival and thriving of opportunistic *r* species, rather than larger *K* species, who may control the populations of species on lower trophic levels (McMichael 2004). Often, vector populations adapt well to man-made environments and out-compete native species (Patz et al. 2000). This replacement decreases species diversity, which can have implications for disease emergence; a study on white-footed mice, a common vector for Lyme disease, suggested increasing host diversity, or species richness, might decrease the risk of disease incidence through a dilution effect (McMichael 2004). Thus, when anopheline mosquitoes occupy a vacated niche, the lack of competitors and predators could possibly further increase the mosquitoes' ability to thrive and thus the likelihood of transmitting disease.

Finally, mosquitoes thrive in warm climates with high rainfall (Sweat 2006). Roads facilitate the use of fossil fuel-burning vehicles and cause deforestation—two actions that increase carbon emissions and thereby contribute to global warming (Turner et al. 2007, Patz et al. 2000). The rising temperatures and increased rainfall that occur in association with global warming in certain areas “make it possible for mosquitoes to survive in previously inhospitable climates, thus broadening their range” (Sweat 2006). The anopheline range could expand to higher latitudes or to higher altitudes (Khasnis & Nettleman 2005). In addition, the humid climate associated with rainfall prolongs the life span of mosquitoes, and warmer temperatures increase “the rate at which mosquitoes develop into adults, the frequency of their blood feeding, the rate with which parasites are acquired, and the incubation time of the parasite within the mosquito” (Patz et al. 2000, p. 4). Therefore, roads' indirect role in global warming contributes to the expansion of mosquito populations and thus the expansion of disease.

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However, as Patz et al. (2000) alluded to, mosquito population expansion is merely part of the story. In order for anopheline mosquitoes to spread malaria, the pathogen must be able to complete its growth cycle within the mosquito host (CDC 2012). The *Plasmodium* pathogen's ability or inability to develop fully within a host depends on the interaction of the parasite's and mosquito's life span (Lacroix et al. 2005). The above paragraphs have already established that mosquitoes survive best in warm climates with heavy rainfall; *Plasmodium* is also dependent on temperature (Wilson 2000). For example, *Plasmodium falciparum*, the most deadly of malarial pathogens and the most prevalent in Africa (CDC 2012, WHO 2011) cannot complete its growth cycle within the mosquito host at temperatures below 20C° (68F°). Therefore, zones of malaria transmission only exist in certain parts of the world (Figure 2).

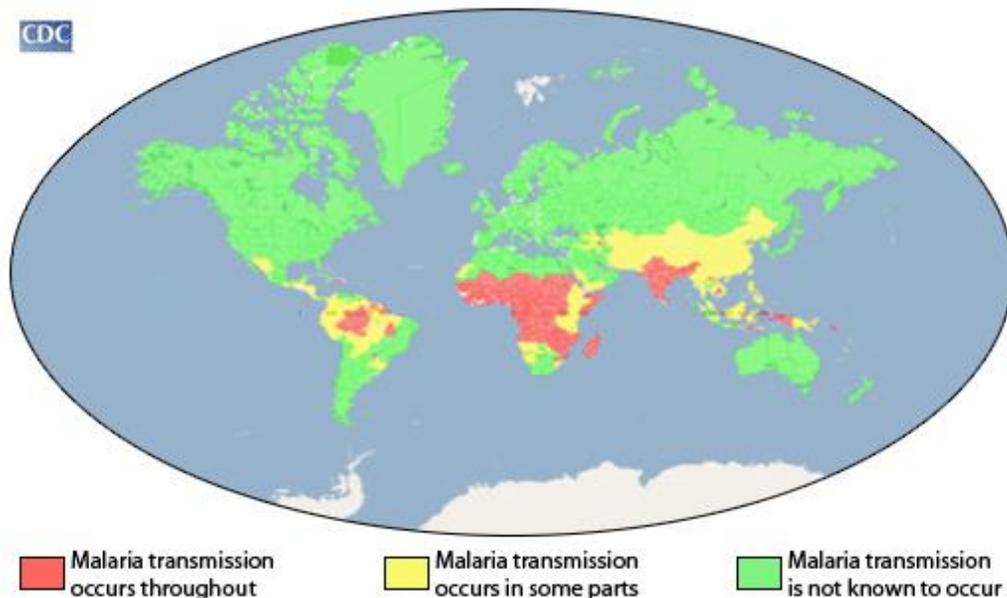


Figure 2: This map shows an approximation of the parts of the world where malaria transmission occurs (CDC 2012).

However, increasing global temperatures could expand this range, and thus broaden malaria's transmission range beyond tropical and subtropical regions (Sweat 2006, CDC 2012) and possibly increase malaria incidence worldwide.

Therefore, roads, both directly and through collateral impacts, promote the survival of both anopheline mosquitoes and the *Plasmodium* pathogen due to roads.

### **Roads and Vector-to-Human Transmission**

In addition to promoting the survival of malaria vectors and pathogens, road-induced anthropogenic change increases opportunities for vector-to-human transmission (Olson et al. 2010, Wolfe et al. 2000, Martens & Hall 2000). My research supports the claim that roads increase the rate of and opportunities for vector-to-human transmission due to the effects of deforestation, habitat destruction and habitat fragmentation.

Deforestation causes many negative impacts on a landscape that promote the prevalence of malaria (Patz et al. 2004, Olson et al. 2010). For example, deforestation increases the mean biting rate of mosquitoes (Olson et al. 2010). A study in the Amazon found the mean biting rate of the malaria vector *A. darlingi* mosquito “in areas with >80% deforestation was 8.33 compared with 0.03 per night for sites with <30% deforestation” (Olson et al. 2010, p. 1108). While this difference may partially correlate to differences of human population density in forested versus deforested areas, deforestation is still a factor in the disparity (Olson et al. 2010). The study also found the likelihood of finding *A. darlingi* larvae in areas with <20% forest doubles in areas with 20-60% forest, and is seven times higher compared with areas with >60% forest (Olson et al. 2010). This could be because, as I mentioned in the previous section, anopheline mosquitoes seek breeding habitats in the neutral-pH stagnant pools of sunlit areas, and “are seldom observed in standing water bodies within undisturbed forests” (Olson et al. 2010, p. 1108).

Furthermore, Olson et al. (2010) found that after adjusting for population, access to care, and district size, malaria risk increased approximately 50% in health districts when 4% of the district’s area underwent deforestation from 1997-2000. Therefore, deforestation increases the risk of contracting malaria as well as the biting rate of malaria vectors and the number of vector larvae sites.

Deforestation also creates other negative environmental impacts. Clearing forested lands causes habitat destruction and fragmentation (Chomintz & Gray 1996). Habitat destruction also leads to growth in human-wildlife interaction and conflict (Patz et al. 2004), which has resulted in exposure to new pathogens for humans (Wolfe et al. 2000). Deforestation and habitat fragmentation increase the “edge effect” of an area—smaller, fragmented patches of forest further promote interaction among pathogens, vectors and hosts (Glass et al. 1995, Patz et al. 2004). Therefore, deforestation and habitat

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destruction increase the ability of humans to come into contact with pathogens and vectors. These consequences of road-related environmental impacts combined with the previously discussed increased abundance of anopheline mosquitoes likely result in a higher risk of vector-to-human transmission than either circumstance would on its own.

However, human migration can play a key role in the spread of disease (Martens & Hall 2000). According to Patz et al., “Deforestation, with subsequent changes in land use and human settlement patterns, has coincided with an upsurge of malaria and/or its vectors in Africa, in Asia, and in Latin America” (2004, p. 1093). According to Martens & Hall (2000), malaria incidence has increased dramatically in the Amazon since the 1960s due to new highways that linked the Amazon region to the rest of the country and facilitated massive population movements to colonize new territory, as well as attracted laborers to work on road construction. The effect of human migration on disease transmission is twofold—people who move can be categorized as either active transmitters or passive acquirers (Martens & Hall 2000). I will discuss active transmitters in the next section. Passive acquirers, however, “are exposed to [malaria] through movement from one environment to another” (p. 104), and increase their risk “through the ways in which they change the environment...for example, through deforestation” (Martens & Hall 2000, p. 103). These passive acquirers are usually nonimmune and travel from malaria-free areas to malaria-endemic areas. As vessels for human travel (Patz et al. 2000, Forman & Alexander 1998), roads play a role in these migrations.

Indeed, migrant workers from malaria-endemic areas were responsible for malaria outbreaks in California in the 1990s (Martens & Hall 2000). In addition, researchers found that malaria incidence increased after the road construction and subsequent erosion that accompanied the gold and diamond boom in Venezuela in the 1950s (Patz et al. 2005). Like deforestation, the gold and diamond boom facilitated mass immigration of new populations to a new area. Due to the previous forest cover, these areas were possibly uninhabited by humans prior to this influx of humans. This new access to resources also provides humans with new access to pathogens and vectors with new access to hosts (Patz et al. 2000). In addition, like work opportunities in California, this boom created mining jobs that drew migrant workers from many different areas. This diversified population density, combined with a large vector population, initiates and

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maintains malaria transmission at high levels (Martens & Hall 2000). Therefore, human settlement and human migration also play a role in the transmission of pathogens from vector to host. Road construction allows previously inaccessible areas to become colonized by people (Patz et al. 2004), who then may become passive acquirers (Martens & Hall 2000), and thus provides the opportunity for exposure to new pathogens (Patz et al. 2000).

### **Roads and Human Transmission**

Roads also facilitate human mobility in ways that allow humans to serve as vectors between separate human populations (Wilson 2000). The Kinshasa Highway truck stop system and its role in the transmission and prevalence of HIV-1 is a prime example of such a trend (Heitman 2001, Preston 1994). However, unlike malaria, HIV is transmissible between humans. Nevertheless, humans can transmit malaria back to mosquitoes. Therefore, human migration remains a factor in the spread of both diseases, and epidemiological studies on one disease may have implications on the other. In particular, cyclical rural-to-urban migration increases risk of spreading malaria and HIV-1 from one population to another (Martens & Hall 2000, Arroyo et al. 2006, Heitman 2000). Arroyo et al. (2006) found population proximity to roads increases incidence and genetic complexity of HIV-1 in Uganda. In this study, Arroyo et al. examined HIV-1 prevalence rates in trading hubs along a main road as opposed to rural villages up to 10 kilometers from the main road. The results showed that while infection rates were higher along main roads, identical HIV-1 strains occurred in the villages as well. This not only demonstrates an epicenter effect from the main road, but indicates transmission occurs between the villages and the trading hubs, likely due to the rural-to-urban cyclical migration Martens & Hall (2000) discussed. People traveled between an urbanized center with road access and a rural village with limited road access and thus allowed the disease to spread. Such mobility correlates to transmission of most diseases, including malaria (Patz et al. 2004, Martens & Hall 2000).

Humans become vectors capable of transmission when they become active transmitters. According to Martens & Hall (2000), “active transmitters harbor the parasite and transmit the disease when they move to areas of low or sporadic transmission” (p.

104). In addition, passive acquirers become active transmitters once they are infected (Martens & Hall 2000). Because humans can transmit malarial gametocytes to uninfected mosquitoes or mosquitoes infected with the asexual (non-infective) stage of *Plasmodium* development (Lacroix et al. 2005), humans can act as malaria vectors between populations. While this would have little effect if the destination area already had an established malaria presence, many areas where malaria has been eradicated retain a healthy population of anopheline mosquitoes, and therefore are constantly at risk to re-introduction of malaria pathogens (WHO 2011, Martens & Hall 2000). If active transmitters “return to their initial place of residence in a malaria-free but highly receptive area, they can reintroduce the parasite and initiate an outbreak of malaria” (Martens & Hall 2000, p. 105). Such was the case in Brazil, when in 1985, officials recorded 26 new active foci of malaria that were a result of highly mobile settlers in the Amazon region (Martens & Hall 2000). Active transmitters pose additional risk because mosquitoes infected with the latent stage of *Plasmodium falciparum* not only increase their biting rate, but are more likely to feed on people infected with gametocytes than uninfected individuals or those infected with the asexual stage, independent of other factors that determine human attractiveness to mosquitoes (Lacroix et al. 2005). Therefore, malaria’s impact on the behavior of its mosquito hosts increases the pathogen’s success rate. Human mobility could work in conjunction with these behavioral changes to increase malaria’s range and spread the pathogen to new areas or areas where the disease has been eradicated.

Human mobility has also spread the disease to areas outside the malaria range. Random searches of airplanes at Gatwick Airport found that 12 of 67 airplanes from tropical countries contained mosquitoes (Martens & Hall 2000). This, and other cases of airport malaria, demonstrates mosquitoes’ ability to hide in cargo or other vessels of travel. Indeed, during a hot summer in 1994, officials identified six cases of malaria in and around Roissy-Charles-de-Gaulle Airport (Giacomini et al. 1995). Two patients lived approximately 7.5 km away from the airport (Giacomini et al. 1995). The accepted explanation for these cases is, “Anopheline mosquitoes were thought to have traveled in the cars of airport workers who lived next door to two of the patients” (Martens & Hall 2000, p. 107, Giacomini et al. 1995). If cars transported mosquitoes in France, vehicles

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might easily do the same in sub-Saharan Africa and throughout the rest of the malaria range and the world. Therefore, roads not only allow the movement of humans as active transmitters of malaria, but have the potential to transport the mosquito vectors.

## **VI. Conclusion**

Roads lead to environmental change that promotes the survival, reproduction and success of *Plasmodium* malaria pathogens and malaria's anopheline mosquito vectors. Once the pathogen is established, roads can move infected persons and mosquitoes to uninfected areas, as well as move uninfected persons to infected areas. At this point in history, population movements and activities that either put people at risk for malaria or cause them to pose a threat to others cannot be stopped (Martens & Hall 2000). With or without roads, human mobility has existed throughout time; pathmaking is even an observed behavior of our primate relatives (McMichael 2004). However, researchers could pursue studies to find if building fewer roads would reduce deforestation and therefore significantly reduce the amount of viable anopheline mosquito habitats, and could possibly relocate such habitats to areas farther from human populations. Such a feat would likely require strict protection of forested areas along roadsides and preservation of large areas of forest to reduce edge effect. The evaluation of such initiatives and their potential impact on the alleviation or eradication of malaria requires further research. However, LCS researchers and epidemiologists must embrace the EnvID framework to recognize both the role of environmental change in disease emergence and spread and the specific anthropogenic actions that underlie such problems. Road building is a leading cause of deforestation—which plays a major role in disease emergence (Patz et al. 2005)—as well as a primary source of human mobility (Wilson 2000). Therefore, the environmental and social impacts of roads must be reduced and controlled in order to mediate the emergence and spread of infectious diseases.

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