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THE INFLUENCE OF INFECTIOUS DISEASES ON POPULATION GENETICS

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Abstract. Malaria is the vector-transmitted disease that causes the highest morbidity and mortality in humans. Motivated by the known influence of sickle-cell anemia on the morbidity and mortality of malaria-infected humans, we study the effect of malaria on the genetic composition of a host (human) population where sickle-cell anemia is prevalent and malaria is endemic. The host subpopulations are therefore classified according to three genotypes, AA, AS, and SS. It is known that AA malaria-infected individuals experience higher malaria-induced mortality than AS or SS individuals. However, individuals carrying the S gene are known to experience a higher mortality rate in a malaria-free environment than those who lack such a gene. The tradeoffs between increased fitness for some types in the presence of disease (a population level process) and reduced fitness in a disease-free environment are explored in this manuscript. We start from the published results of an earlier model and proceed to remove some model restrictions in order to better understand the impact on the natural hosts' genetics in an environment where malaria is endemic.

1. **Introduction.** Efforts to understand a host's evolutionary dynamics (often slow) in the context of disease dynamics in "chemically" treated environments (e.g., chemotherapy) or the influence of diseases on the host's genetic variability or both have been carried out recently (see, for example, Andreasen [1], Beck [2], Castillo-Chavez and Feng [3], Feng et al. [5, 6], Galvani et al. [8, 9], Hsu-Schmitz [13], Kribs et al., May and Anderson [10], McKenzie [11, 12]) in the context of human diseases such as HIV, tuberculosis, malaria and others. Feng et al. [5] studied the influence of malaria dynamics (assumed to be fast) on hosts' fitness in their efforts to assess the effect of vector-borne diseases on the genetic composition of a host population. A typical setting was adopted by assuming that the human population is characterized by a two-allele single loci system. Here, we remove some of their model simplifications and show that the qualitative dynamics do not change. However, we briefly illustrate the potentially significant effect that the incorporation of additional host characteristics (age) may have on disease dynamics and on

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