Insecticide Treated Livestock and Control of Human Malaria: a Modelling Approach.

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Summary

Treatment of livestock with insecticide has been widely used as an effective strategy to control ectoparasites, flies and the diseases they transmit to livestock. This approach has recently started to be evaluated as a novel method to control human malaria, in regions where the disease vector also feeds on animals. Promising results have been observed from field trials in Pakistan. Here we develop a simple mathematical model to investigate the circumstances under which a policy that involves the systematic use of insecticides on livestock will decrease the prevalence of human malaria.

Introduction

Malaria, a re-emergent vector-borne disease, has always had a deep impact on the health and economy of a large percentage of the word population. In areas where the vectors for human malaria also feed on animals, the presence of livestock impacts the risk of disease transmission to people. Although livestock is not susceptible to malaria infection, it may act as a readily accessible source of blood meal to the host-seeking mosquitoes, thereby increasing vector population densities. Recent trials in Pakistan have showed that the treatment of livestock with pyrethroid insecticides decreased the incidence of malaria with a similar efficacy to the traditional indoor insecticide spraying but with much lower costs. Moreover, significant improvements in livestock productivity were obtained, enhancing communities uptake of the programme [1]. However, several factors underlying the effectiveness of insecticide treated livestock remain poorly understood. This study looks at the relevance of some of these factors, including vector host-feeding preference and the percentage coverage achieved each treatment round. These questions are addressed by developing a simple deterministic model of malaria transmission.

Materials and Methods

An epidemiological framework was developed that quantitatively addresses the impact of insecticide treated cattle on incidence of human malaria. The framework was built based on the classical Ross-Macdonald model of malaria dynamics (reviewed in [2]). The expanded model here presented discriminates the feeding behaviour of the vector on its alternative hosts, livestock and human populations, and incorporates the treatment of livestock with insecticide as a novel method to control human malaria. A schematic representation of the model is presented in Figure1.

Total human (N_h) and vector (N_v) populations are divided into epidemiological compartments that comprise uninfected and susceptible to infection (S_h and S_v), latently infected but not yet infectious (L_h and L_v), and infectious (I_h and I_v) sub-populations.

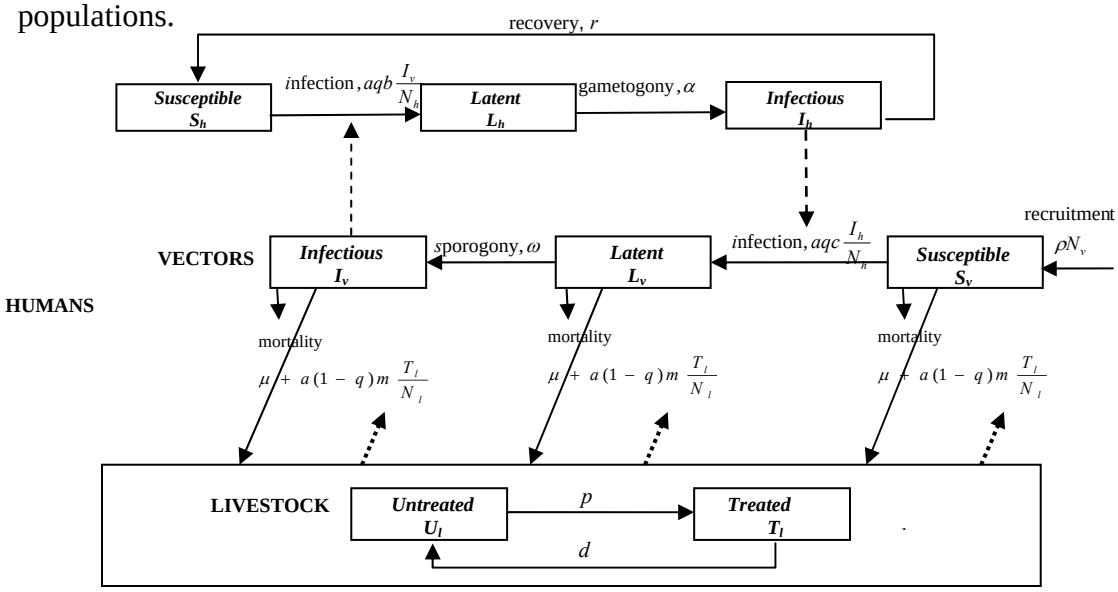

Figure 1. Diagram of the malaria model.

First, let us consider the dynamics of infection in the human population. Susceptible, S_h, become infected at a rate that depends on the number of infected vectors per human, I_v/N_h, the vector blood feeding rate a, the proportion q of feeds taken on humans (antropophily), and the probability b that a human will become infected following the bite of an infected vector. Infected latent (L_h) become infectious after a period for development of infective gametocytes (latent period = $1/\alpha$). Infectious individuals recover at a rate r, becoming fully susceptible to re-infection. For simplicity**,** no immunity to malaria infection is assumed and human demography is ignored, with total population size, $N_h = S_h+L_h+I_h$, set to be constant.

The disease dynamics in the vector population may be similarly described. Susceptible vectors, S_v, acquire infection at a rate that depends on the proportion of infectious humans, I_h/N_h, the vector feeding rate on humans, aq, and the probability c that a vector will become infected after biting an infectious human. Infected latent mosquitoes (L_v) become infectious following a period for sporozoites maturation (latent period = $1/\omega$). Vectors are assumed to remain infectious throughout their life. In the absence of any control intervention, the vector demography is described by the natural death rate μ, (assumed to be age independent, such that average life-span = $1/\mu$) and recruitment rate, ρ. We assume perfect density dependence compensation, by setting $\mu = \rho$, and therefore, the vector population size $N_h = S_h+L_h+I_h$ remains constant. When livestock is treated with insecticide, the vector population size is allowed to decrease due to an increase in the mortality rate, which is a function of the

vector biting rate on livestock, *a(1-q),* the proportion of insecticide treated livestock, T_l / N_l, and the vector additional mortality due to insecticide, *m.* In each round of livestock treatment, animals are treated at a daily rate *p,* set to obtain the required treatment coverage. The insecticide effect is assumed to be maximum on the day of the intervention and is subject to exponential decay, with average duration 1/*d*. The value of *d* will depend mainly on the type of insecticide formulation used, as well as on the method of application (e.g. 'spot-on' *vs.* sponging) and area of the animal covered by the insecticide. The model considers a scenario where livestock population is constant, and therefore demography can be ignored.

We start by calculating the so-called basic reproductive number R_0 for the presented model, using the next generation approach [3, 4]. This threshold quantity expresses the average number of secondary infections produced by one infected case in a fully susceptible population, and must exceed unity for the infection to persist.

$$R_o = \frac{N_v}{N_h}\frac{(aq)^2 bc}{r}\frac{\omega}{\left(\mu + a(1-q)m\frac{T_l}{N_l}\right)\left((\omega+\mu) + a(1-q)m\frac{T_l}{N_l}\right)}$$

Results

As illustrated in Figure 2, for a given livestock coverage treatment level (T_l / N_l), increases in vector antropophily (q) lead to increase in R_0. The smaller the coverage, the steeper the increase in R_0, and vice-versa. Conversely, for a given antropophily, increases in coverage generate a decrease in R_0. Further analysis will provide insights regarding the coverage treatment required to decrease R_0 below 1, in relation to the vector host feeding preference in a given setting.

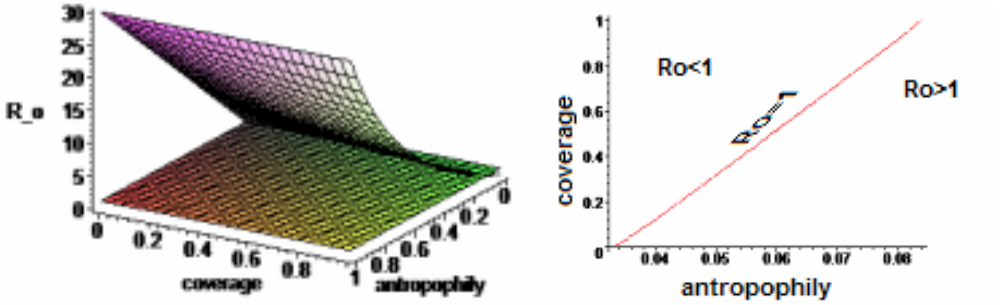

Figure 2. The basic reproduction number with respect to livestock treatment coverage and vector antropophily. (N_v/N_h=10, *a*=1/2, *b*=1, c=0.6, *r*=1/240, ω=1/8, μ=1/6, *m*=0.73).

Discussion

The main focus of this research is to understand the circumstances under which a policy that involves the systematic use of insecticides on livestock will decrease the prevalence of human malaria. The results presented in this paper are still preliminary, and further work is being conducted. Such understanding will be a major contribution to the optimization of this control strategy in a given setting. Most importantly, it will enable the impact of the strategy in different settings to be estimated. The quantitative framework developed in our study is an important step towards this direction.

References

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