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H. Gaff
Old Dominion University, hgaff@odu.edu

L. Gross

E. Schaefer

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Results from a mathematical model for human monocytic ehrlichiosis

H. Gaff[1], *L. Gross*[2] *and E. Schaefer*[3]

[1]Analysis and Simulation Center, School of Community and Environmental Health College of Health Sciences, Old Dominion University, Norfolk, VA, USA, [2]Department of Ecology and Evolutionary Biology, Mathematics Department, University of Tennessee, Knoxville, TN, USA and [3]Department of Mathematics, Marymount University, Arlington, VA, USA

Human monocytic ehrlichiosis (*Ehrlichia chaffeensis*), HME, is a tick-transmitted, rickettsial disease that has recently increased substantially in the USA from 142 reported cases in 2001 to 506 reported cases in 2005 [1,2]. There have been increasing surveys of tick populations over the past 10 years that have in turn supported the development of models for tick-borne disease transmission. Resulting HME models [3] suggest the importance of metapopulation structures, landscape environment parameters and periodic climatic effects in predicting the dynamics of HME transmission and the efficacy of control efforts, such as the reduction of the tick population through acaricide use. On this note, we describe a spatially-explicit model for HME transmission, and give a result illustrating the importance of migration in the dynamics of HME risk.

Our initial model is a simplified representation of the tick-host-HME system using a single host population, a single life stage and a single pathogen. The dynamics are modelled using differential equations on a spatial grid consisting of environmental patches. White-tailed deer (*Odocoileus virginianus*) serve as our generic host population and lone-star ticks (*Amblyomma americanum*) as the tick population. The population densities of the host and tick population in each patch are denoted by N_i and V_i, respectively, and the densities of the HME-infected and infectious populations are denoted by Y_i (hosts) and X_i (ticks). Both hosts and ticks are considered susceptible when not infected. Once infected with HME both ticks and hosts are assumed to be infected for life.

$$\frac{dN_i}{dt} = \beta_i\left(\frac{K_i - N_i}{K_i}\right)N_i - b_iN_i + \sum_j m_{ij}(N_j - N_i)$$

$$\frac{dV_i}{dt} = \hat{\beta}_iV_i\left(\frac{M_iN_i - V_i}{M_iN_i}\right) - \left(\hat{b}_i + \delta_i\right)V_i + \sum_j m_{ij}(V_j - V_i)$$

$$\frac{dY_i}{dt} = A_i\left(\frac{N_i - Y_i}{N_i}\right)X_i - \beta_i\frac{N_iY_i}{K_i} - b_iY_i + \sum_{N_j<N_i} m_{ij}\frac{Y_i}{N_i}(N_j - N_i) + \sum_{N_j>N_i} m_{ij}\frac{Y_j}{N_j}(N_j - N_i)$$

$$\frac{dX_i}{dt} = \hat{A}_i\left(\frac{Y_i}{N_i}\right)(V_i - X_i) - \hat{\beta}_i\frac{V_iX_i}{M_iN_i} - \left(\hat{b}_i + \delta_i\right)X_i + \sum_{V_j<V_i} m_{ij}\frac{X_i}{V_i}(V_j - V_i) + \sum_{V_j>V_i} m_{ij}\frac{X_j}{V_j}(V_j - V_i)$$

The first equation above describes the population dynamics of the host population in patch i, for which there is logistic growth with carrying capacity K_i as well as an external death rate, b_i, caused by hunting or removal. The last term in the equation models the migration from the given patch to all other patches at a rate dependent on the population differential between patches and the connectivity between patches. The second

Corresponding author: H. Gaff, Virginia Modelling, Analysis and Simulation Center School of Community and Environmental Health, 3133A Technology Building, Old Dominion University, Norfolk, VA 23529, USA
E-mail: hgaff@odu.edu

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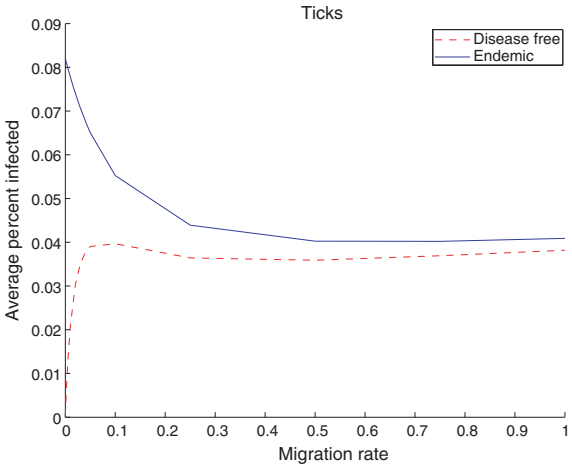

Fig. 1. Average percentage of infected ticks in the endemic (solid blue) and disease-free (dotted red) patches after 1000 months. Migration rates must be very low in order for the individual patches to retain their natural disease dynamics.

equation describes the tick population dynamics, which are similar to the host dynamics with the carrying capacity for the ticks as the product of the maximum number of ticks per host M_i and the number of hosts N_i. In addition, the external death rate for ticks includes both weather, $\hat{b}_i$, and acaricide, δ_i, components. The first terms in the third and fourth equations describe the new infections for the host and tick populations, respectively, with transmission rates given by A and $\hat{A}$. The next terms reflect the logistic growth and external deaths of the infected host and tick populations, and the last terms are the migration terms, which show the proportion of infected hosts and ticks that migrate as given with the rates in the first two equations.

In initial simulations, the model was allowed to run for 1000 months on a single patch. We found that with the absence of control, HME prevalence reached between 5 and 15% infected for ticks with hosts at about 35% infected. With the application of acaricide from month 500 forward, the tick population is significantly reduced, and HME is eliminated from both populations in the model. If the acaracide is only applied for 3 years, we find that the tick population recovers to pretreatment levels within a year, while HME doesn't return until nearly 10 years later.

We then expanded the model with multiple patches to include environmental differences, including external death rates that vary due to exposure or predation. Our initial work has two habitat classifications: grass and woods. Each geographic area is linked to others using migration that depends on distance between the two areas and existence of any barriers that would prevent movement between them. The figure shows the effect of migration rates on the average percentage of ticks that are infected in the two patches, one where HME would be endemic in isolation (solid blue) and one where it would die out in isolation (dotted red) (Fig. 1). These results show that even a small amount of connection to an endemic area will quickly increase the prevalence of HME in an area that would be free of disease in isolation.

Future work on this project will further explore the importance of metapopulation considerations in modelling of rickettsial disease. In particular, we consider the importance of environmental variability, host diversity, habitat fragmentation and targeted application of acaricide.

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