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A network-based meta-population approach to model Rift Valley fever epidemics

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HIGHLIGHTS

- A meta-population model based on weighted networks has been developed.
- Spatial and temporal evolution of Rift Valley fever virus spread has been studied.
- Parameters representing mosquito propagation and development are functions of weather.
- Bounds for the reproduction number are functions of vertical and horizontal transmission.
- The model has been applied to the case study of South Africa 2010 RVF outbreak.

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ABSTRACT

Rift Valley fever virus (RVFV) has been expanding its geographical distribution with important implications for both human and animal health. The emergence of Rift Valley fever (RVF) in the Middle East, and its continuing presence in many areas of Africa, has negatively impacted both medical and veterinary infrastructures and human morbidity, mortality, and economic endpoints. Furthermore, worldwide attention should be directed towards the broader infection dynamics of RVFV, because suitable host, vector and environmental conditions for additional epidemics likely exist on other continents; including Asia, Europe and the Americas. We propose a new compartmentalized model of RVF and the related ordinary differential equations to assess disease spread in both time and space; with the latter driven as a function of contact networks. Humans and livestock hosts and two species of vector mosquitoes are included in the model. The model is based on weighted contact networks, where nodes of the networks represent geographical regions and the weights represent the level of contact between regional pairings for each set of species. The inclusion of human, animal, and vector movements among regions is new to RVF modeling. The movement of the infected individuals is not only treated as a possibility, but also an actuality that can be incorporated into the model. We have tested, calibrated, and evaluated the model using data from the recent 2010 RVF outbreak in South Africa as a case study; mapping the epidemic spread within and among three South African provinces. An extensive set of simulation results shows the potential of the proposed approach for accurately modeling the RVF spreading process in additional regions of the world. The benefits of the proposed model are twofold: not only can the model differentiate the maximum number of infected individuals among different provinces, but also it can reproduce the different starting times of the outbreak in multiple locations. Finally, the exact value of the reproduction number is numerically computed and upper and lower bounds for the reproduction number are analytically derived in the case of homogeneous populations.

1. Introduction

Rift Valley fever (RVF) is a viral zoonosis with enormous health and economic impacts on domestic animals and humans (Linthicum et al., 2007), in countries where the disease is endemic and in others where sporadic epidemics and epizootics have occurred. An outbreak in South Africa in 1951 was estimated to have infected 20,000 people and killed 100,000 sheep and cattle (Department for Environment Food and Rural Affairs, 2010; Sellers et al., 1982). In Egypt in 1977, there were 18,000 human cases with 698 deaths resulting from the disease (Department for Environment Food and Rural Affairs, 2010; Sellers et al., 1982). While RVF is endemic in Africa, it also represents a threat to Europe and

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Western hemispheres (Chevalier et al., 2005; Gaff et al., 2007). In 1997–1998 Kenya experienced the largest recorded outbreak with 89,000 human cases and 478 death. The first recorded outbreak outside Africa occurred in the Arabian peninsula in 2000–2001 and caused 683 human cases and 95 deaths (Florida Department of Health, 2010). Tanzania and Somalia reported 1000 human cases and 300 deaths from an outbreak that was associated with above-normal rainfall in the region in 2006–2007 (Florida Department of Health, 2010). Rift Valley fever virus (RVFV) is generally distributed through regions of Eastern and Southern Africa where sheep and cattle are present (Woods et al., 2002). It can cause morbidity (ranging from nondescript fever to meningo-encephalitis and hemorrhagic disease) and mortality (with case fatality rates of 0.2–5%) in humans (Linthicum et al., 2007). The main economic losses of RVF in livestock arise due to abortion and mortality, which tends to be higher in young animals (Clements et al., 2007; Woods et al., 2002), and bans on livestock exports during an epidemic (Clements et al., 2007; Anyamba et al., 2001).

Rift Valley fever virus was first isolated from the blood of a newborn lamb in 1931 and later from the blood of adult sheep and cattle (World Health Organization, 2010; Anyamba et al., 2001). Domestic ruminants and humans are among the mammalian hosts demonstrated to amplify RVFV (Kasari et al., 2008) and *Aedes* and *Culex* are believed to be the main arthropod vectors (Chevalier et al., 2005). Rift Valley fever virus can be transferred vertically from females to their eggs in some species of the *Aedes* mosquitoes (Gaff et al., 2007; Linthicum et al., 1985). The disease has been shown to be endemic in semi-arid zones, such as northern Senegal (Zeller et al., 1997; Chevalier et al., 2005; Martin et al., 2008), and RVF epidemics often appears at 5–15 year cycles (Martin et al., 2008). As noted earlier, RVFV has already spread outside Africa, to Yemen and Saudi Arabia (Chevalier et al., 2005; CDC, 2007). The species of vectors that are capable of transmitting RVFV have a wide global distribution (Gubler, 2002) and there is therefore a distinct possibility for the virus to spread out of its currently expanding geographic range (Clements et al., 2007). A pathway analysis (Kasari et al., 2008) has shown that the RVF virus might be introduced into the United States in different ways (Kasari et al., 2008; Konrad et al., 2010) and that analysis identified several regions of the United States that are most susceptible to the RVFV introduction. It is therefore desirable to develop effective models to better understand the potential dynamics of RVF in heretofore unaffected regions and then develop efficient mitigation strategies in case this virus appears in the Western hemisphere (Gaff et al., 2007). Such preparedness can help avoid a rapid spread of the virus throughout North America, as happened with the West Nile virus during the last decade (Chevalier et al., 2005; Gaff et al., 2007).

A RVF disease risk mapping model was developed by Anyamba et al. (2009). The authors observed sea surface temperature (SST) patterns, cloud cover, rainfall, and ecological indicators (primarily vegetation) via satellite data to evaluate different aspects of climate variability and their relationships to disease outbreaks in Africa and the Middle East (Anyamba et al., 2002, 2006). The researchers successfully predicted areas where outbreaks of RVF in humans and animals were expected using climate data for the Horn of Africa from December 2006 to May 2007. An ordinary differential equation (ODE) mathematical model was developed by Gaff et al. (2007). The model is both an individual-based and deterministic model. The authors analyzed the stability of the model and tested the importance of the model parameters. However, neither human population parameters nor spatial (or, network) aspects are explicitly incorporated in the model. Another theoretical mathematical model on RVFV dynamic transmission was proposed (Mpeshe et al., 2011). This model is also an individual based model. The most important parameters to initial disease transmission and the endemic equilibrium have been carried out.

In this paper, we present a novel model incorporating *Aedes* and *Culex* mosquito vector, and livestock and human host populations. Our model is based on weighted contact networks, where nodes of the networks represent geographical regions and weights represent the level of contact between regional pairs for each vector or host species. Environmental factors such as rainfall, temperature, wind and evaporation are incorporated into the model. For each subpopulation, a set of ordinary differential equations describes the dynamics of the population in a specific geographical location, and the transitions among the different compartments, after contracting the virus. We compute the lower and upper bounds of the reproduction number for homogeneous populations, explain their biological meaning, and numerically compare the bounds with exact values.

We test, calibrate, and evaluate the model using the recent 2010 RVF outbreak in South Africa as a case study, mapping the epidemic spread in three South African provinces: Free State, Northern Cape, and Eastern Cape. An extensive set of simulation results shows the potential of the proposed approach to accurately describe the spatial–temporal evolution of RVF epidemics.

The paper is organized as follows: (1) in Section 2, we describe our compartmentalized mathematical model, present the lower bound and upper bound of the reproduction number for homogeneous populations; (2) in Section 3, we introduce the case study using outbreak data from South Africa, 2010; and (3) in Section 4, we conclude our work. In the Appendix, we show how we derive the bounds for the reproduction number for homogeneous populations.

2. Compartmentalized mathematical model

We have constructed Compartmentalized Mathematical Models based on the principle of RVFV transmission. The parameters used in the model are shown in Table 1.

2.1. Homogeneous populations model

The principle of RVFV transmission between different species is shown in Fig. 1. The *Aedes* species and *Culex* species we consider in the model only include the mosquitoes that are competent vectors of Rift Valley fever. The main vectors, *Aedes* and *Culex* mosquitoes and the main hosts, livestock and humans are considered in the model. We use an SEI compartmental model in which individuals are either in a susceptible (S) state, an exposed (E) state, or an infected state (I) for both *Aedes* and *Culex* mosquitoes, and an SEIR. compartmental model in which individuals are either in a susceptible (S) state, an exposed (E) state, an infected state (I), or a recovered (R) state for both livestock and human populations. Infectious *Aedes* mosquitoes can not only transmit RVFV to susceptible livestock and humans but also to their own eggs (Gaff et al., 2007; Linthicum et al., 1985). *Culex* mosquitoes acquire the virus during blood meals on an infected animal and then amplify the transmission of RVFV through blood meals on livestock and humans (World Health Organization, 2010). Direct ruminant-to-human contact is the major (though not only) way for humans to acquire the infection (Anyamba et al., 2009; Davies and Martin, 2006). Accidental RVFV infections have been recorded in laboratory staff handling blood and tissue from infected animals (Anyamba et al., 2009). Usually, humans are thought of as dead end hosts that do not contribute significantly to propagation of the epidemic (Chevalier et al., 2005). There has been no direct human-to-human transmission of RVFV in field conditions recorded thus far (Kasari et al., 2008). The mosquitoes will not spontaneously recover once they become infectious (Gaff et al., 2007). Livestock and humans either perish from the infection or recover (Gaff et al., 2007). All four species have a specified incubation period (World Health Organization, 2010). The model is based on a daily time step. *Aedes*