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Network analyses of transhumance movements and simulations of foot-and-mouth disease virus transmission among mobile livestock in Cameroon

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ABSTRACT

Foot-and-mouth disease (FMD) affects cloven-hoofed livestock and agricultural economies worldwide. Analyses of the 2001 FMD outbreak in the United Kingdom informed how livestock movement contributed to disease spread. However, livestock reared in other locations use different production systems that might also influence disease dynamics. Here, we investigate a livestock production system known as transhumance, which is the practice of moving livestock between seasonal grazing areas. We built mechanistic models using livestock movement data from the Far North Region of Cameroon. We represented these data as a dynamic network over which we simulated disease transmission and examined three questions. First, we asked what were characteristics of simulated FMDV transmission across a transhumant pastoralist system. Second, we asked how simulated FMDV transmission across a transhumant pastoralist system differed from transmission across this same population held artificially stationary, thereby revealing the effect of movement on disease dynamics. Third, we asked if disease simulations on well-studied theoretical networks are similar to disease simulations on this empirical dynamic network. The results show that the empirical dynamic network was sparsely connected except for an eight-week period in September and October when pastoralists move from rainy season to dry season grazing areas. The mean epidemic size across all 3,744 simulations was 99.9% and the mean epidemic duration was 1.45 years. Disease simulations across the static network showed a smaller mean epidemic size (27.6%) and a similar epidemic duration (1.5 years). Epidemics simulated on theoretical networks showed similar final epidemic sizes (100%) and different mean durations. Our simulations indicate that transhumant livestock systems have the potential to host FMDV outbreaks that affect almost all livestock and last longer than a year. Furthermore, our comparison of empirical and theoretical networks underscores the importance of using empirical data to understand the role of mobility in the transmission of infectious diseases.

1. Introduction

Foot and mouth disease (FMD) is a highly infectious disease that concerns livestock keepers worldwide (James and Rushton, 2002; Knight-Jones and Rushton, 2013) and spreads, in part, though movement of infected and susceptible hosts (Di Nardo et al., 2011; Tekleghiorghis et al., 2016; Brito et al., 2017). Livestock movement bans, like those enacted during 2001 FMD epidemic in the United Kingdom, have been found effective in slowing spread of the disease (Ferguson et al., 2001; Haydon et al., 2004) and emphasize the role of movement in FMD spread. Here, we investigate a complex series of movements made by mobile pastoralists in the Far North Region, Cameroon to determine how these movements impact foot-and-mouth disease virus (FMDV) transmission.

Mobile pastoralists are livestock herders who take their herds on transhumance, which is the practice of moving livestock between grazing areas tracking changing seasonal patterns in forage productivity (Scholte et al., 2006; Moritz et al., 2013c). A few studies have investigated correlations between mobile pastoralists and FMDV infection and found that livestock that are mobile have higher risk for FMDV infection in some systems, particularly in Adamawa, Cameroon (Bronsvoort et al., 2004) and in Ethiopia (Megersa et al., 2009). However, the association between mobility and FMDV infection is not seen in all instances and is notably missing among mobile pastoralists in Nigeria (Ehizibolo et al., 2014). To determine how transhumance affects FMDV dynamics, the connection between pastoral mobility and FMDV transmission should be studied mechanistically. Pragmatically, the lack of detailed disease data from mobile herds makes it difficult to

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assess the role of transhumance movements in FMDV transmission.

One approach to overcome the lack of disease data is to gather location data on mobile pastoralists, computationally simulate the movement patterns, and then assess the potential for sustained pathogen transmission across these herds (Buhnerkempe et al., 2014; White et al., 2017). Network models are ideal for representing spatial dynamics of mobile and heterogeneous livestock populations and disease transmission therein (Bansal et al., 2007; Brooks-Pollock et al., 2015; Riley et al., 2015). In a network, hosts are represented as nodes and contacts between hosts are represented as edges. Edges can form and break over time, representing dynamic contact patterns. If we quantify an empirical network of mobile pastoralists from location data, simulate FMDV transmission across it, and compare the empirical dynamic network results to simulations of FMDV transmission across this same population held artificially stationary, then we can determine how transhumance affects FMDV transmission.

An alternative approach, often used in the absence of both disease and location data, is to simulate disease transmission across a well-studied theoretical network. This method has led to important insights about disease dynamics in both static networks (Pastor-Satorras and Vespignani, 2001) and dynamic networks (Eames and Keeling, 2002). However, real contact networks might differ from well-studied theoretical networks and the extent to which these insights from disease simulations across theoretical networks apply to real epidemics is unclear. If we quantify an empirical network of mobile pastoralists and simulate FMDV transmission across this network, we can compare the empirical network structure and simulation results to theoretical network structures and simulation results. This would be particularly relevant for three classes of well-studied theoretical networks: random networks (Barabási and Posfai, 2016), small-world networks with node connectivity described as a power-law distribution, and small-world networks with node connectivity described as a Watts-Strogatz distribution (Watts and Strogatz, 1998; Amaral et al., 2000).

Here, we quantify the network of mobile pastoralists using transhumance data from the Far North Region, Cameroon (Scholte et al., 2006; Moritz et al., 2013c). More than 200,000 cattle participate in seasonal transhumance in the Far North Region of Cameroon, which represents approximately 20% of all cattle in the region (Seignobos, 2000). Previously, this transhumance has been documented and modeled (Xiao et al., 2015; Kim et al., 2016; Ludi et al., 2016; Pomeroy et al., 2019 under review). We extend this work and analyze these mobile herds as an empirical dynamic network. We focus on camps, which consist of households and their herds of cattle. We represent camps as nodes and draw edges between camps assembled at the same location. As pastoralists move their camps, the distance between camps changes and the network structure changes.

In the Far North Region, Cameroon, five serotypes of FMDV have been detected and transmission is assumed to be endemic (Ludi et al., 2016). However, our previous analyses suggest that the nature of FMDV transmission differs by serotype: type O and SAT1 are likely to maintain endemic transmission while SAT2, SAT3, and Type A show epidemic or stuttering chains of transmission (Pomeroy et al., 2015). FMDV has been detected in cattle, sheep, pigs, and goats in this region (Ekue et al., 1990; Ludi et al., 2016), but no wildlife carriers have been detected nor implicated in this area.

Using the empirical dynamic network of mobile pastoralists, we examine three questions about pastoral mobility and FMDV transmission. First, we asked what were the characteristics of simulated FMDV transmission across a transhumant pastoralist system. Second, we asked how simulated FMDV transmission across a transhumant pastoralist system differed from transmission across this same population held artificially stationary, thereby revealing the effect of movement on disease dynamics. Third we ask if disease simulations on well-studied theoretical networks are similar to disease simulations on this empirical dynamic network. This work can help detect if mobile herds play a role in sustained FMDV transmission while also informing models used to make decisions about FMDV transmission and control.

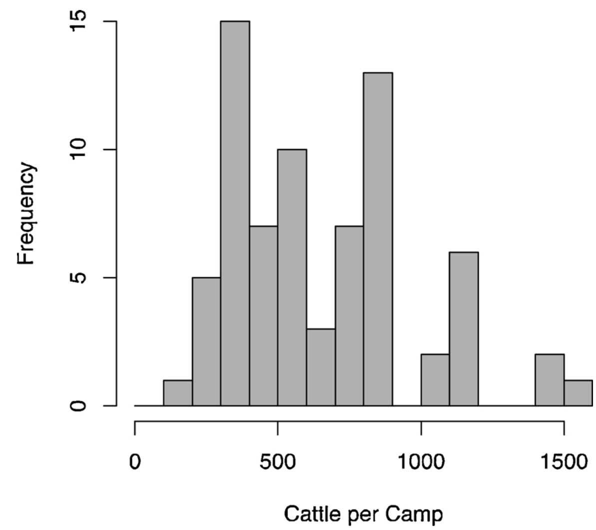

Fig. 1. Distribution of cattle among the 72 mobile camps in the census.

2. Methods

2.1. Data

We performed a complete census of mobile herds that used the seasonal grazing lands of the Logone Floodplain the Far North Region, Cameroon in 2007–2008. This generated transhumance data for 72 camps of mobile pastoralists containing 46,725 cattle. Camps contained a mean of 649 cattle, with a standard deviation of 319.6 cattle (Fig. 1).

We documented the annual transhumance of mobile pastoralists using a combination of surveys and campsite mapping using GPS. The transhumance data are available in the MoveBank Repository (Moritz, 2018). In the survey, we asked pastoralists to name all campsites they stayed at during the previous year and the duration of each stay. We also recorded the number of herds and pastoral households in each camp. Then, we obtained a single GPS coordinate at the centroid of each campsite pastoralists listed in the transhumance surveys. The full dataset consisted of a GPS location for every camp for every day between August 16, 2007 and August 15, 2008.

2.2. Building networks

To transform this dataset into a dynamic network, we created daily adjacency matrices based on physical proximity: each camp was considered connected to another camp if both had stayed at the same campsite and, therefore, had the same GPS coordinates on the same day. If the two camps were connected, the corresponding entry in the daily adjacency matrix contained a one; otherwise, the entry contained a zero. We constructed adjacency matrices in this way for each day of the year and then summarized the daily adjacency matrices into 53 weekly adjacency matrices. Entries in the weekly adjacency matrices ranged from zero to seven and represented the number of days in a given week that two camps were in the same location.

Adjacency matrices were converted into networks using the *graph.adjacency* function in the *iGraph* package (Csardi and Nepusz, 2006) in *R* version 3.1.1 (R Core Team, 2017). We used the *plot* function in *iGraph* for visualizing networks.

2.3. Connectivity

The first metric we chose for analyzing the connectivity of weekly networks is the degree, defined as “the count of the number of edges connected to the node" (Farine and Whitehead, 2015). We calculated degree as an unweighted metric. For each camp, we counted the number of non-zero entries in the corresponding column of the weekly adjacency matrix. The second metric we chose for analyzing the connectivity of the empirical dynamic network is network density, defined as "sum of edge weights divided by the number of possible edges" (Farine and Whitehead, 2015). This metric is particularly informative in measuring connectivity in relation to the potential for infectious disease transmission (Pomeroy et al., 2019). The third metric we chose for analyzing the connectivity of the empirical dynamic network is size of the largest component, defined as the number of nodes in the largest connected group of nodes present in the network. For each week, we calculated the largest connected component using the *clusters* command in the *iGraph* package in R. The size of the largest connected component was given by the *$csize* result.

2.4. Disease simulations on the empirical dynamic network

We simulated FMDV transmission using the susceptible, infected, recovered (SIR) framework adapted for transmission on a dynamic network. In this framework, individuals were designated as susceptible to disease, infected and infectious, or recovered from FMD with immunity that lasts for the duration of the simulation, effectively removing the individual from chains-of-transmission. Simulations of FMDV transmission used a weekly timestep; connectivity for each timestep was given by the relevant weekly adjacency matrix constructed from location data. Individuals remain infectious for 7 days, which is equivalent to one timestep and assumes no long-term carrier state.

We initiated epidemic simulations at timestep 1 with a single infectious individual and assumed that all other individuals were susceptible. Let x represent the unique identification number of the camp containing the initial infectious individual. We identified the set of camps at the same location as the camp containing the initial infectious individual. Let $\boldsymbol{y}$ represent a vector containing the unique identification numbers of the set of camps connected to camp. We assume that all animals in camps x and $\boldsymbol{y}$ mix homogeneously.

To simulate stochastic disease transmission, we first determined the number of new infections caused by the initial infectious individual and then determined in which camps the newly infected individuals resided. To determine the number of new infections, we generated a random deviate from a Poisson distribution with $\lambda = 11$ to represent transmission of FMDV serotype O (mean $R_t = 11$) (Pomeroy et al., 2015) using the function *rpois* in R. Let r represent the number of new infections determined by the Poisson draw. To determine which camps contained the new infections, we took a random sample, with replacement, of size r from a vector containing x and all y_i using a vector of weighted sampling probabilities.

We calculated the vector of weighted sampling probabilities based on the number of susceptible cattle remaining in each camp. Let a represent the count of susceptible cattle in camp x and let $\boldsymbol{b}$ represent the counts of susceptible cattle in all camps in $\boldsymbol{y}$. Then, we calculated the weighted probability vector, $\boldsymbol{p}$, for a and all i in $\boldsymbol{b}$ as

$$\boldsymbol{p} = \left(\frac{a}{a + \sum b_i},\ \frac{b_i}{a + \sum b_i}\right). \tag{1}$$

Simulating infection at later timesteps presents three special considerations. First, infectious individuals were distributed among multiple camps, x_i, and there were multiple infectious individuals in the same camp. In these cases, we separately simulated new infections generated by each infectious cattle and summed all of these new infections across all infectious cattle to calculate incidence. Second, the number of susceptible cattle remaining in each herd was updated at multiple times within each timestep so that this metric represented an accurate count after we simulated transmission from each new infection. Third, there were situations near the end of simulated epidemics when the number of new infections assigned to each camp might be greater than the number of susceptible animals remaining in that camp. When that was the case, we assumed that the number of new infections is equal to the number of susceptible animals remaining in the camp.

Multiple simulations were performed in which infection was initiated in each camp (72) on each week (52) for a total of 3744 simulations. Simulations progressed until there were no infectious individuals remaining in the population. Our data provided adjacency matrixes for 52 weeks. If epidemic simulations progressed such that their final epidemic size and duration would be shortened artificially if simulations ended at week 52, we allowed the simulations to proceed assuming that week 53 had the same adjacency matrix as week 1, week 54 had the same adjacency matrix as week 2, etc.

For all simulations, we calculated the final epidemic size and epidemic duration. The final epidemic size was defined as the total number of individuals that became infected during the simulation. We used the *image* function in R for visualizing final epidemic duration in Fig. 6. The epidemic duration was defined as the number of weeks at least one individual in any camp was infected during the simulation.

Model simulations and analyses were conducted in *R* version 3.1.1 (R Core Team, 2017). Code is available upon request.

2.5. Disease simulations on artificial stationary network

We completed additional simulations using an artificial stationary network with 72 nodes. If, for any reason, these mobile camps chose to remain stationary, they would opt to remain near the town of Mindif in the southern end of Far North Region, Cameroon. The town of Mindif, located southwest of the Logone Floodplain, is the middle of an agro-pastoral zone where many mobile pastoralists (who spend the dry season in the floodplain) spend the rainy season. It is also home to many agro-pastoralists who stay in the zone with their herds throughout the year.

Because the week 52 adjacency matrix represents the spatial arrangement of herds when they were near Mindif, we used it for the disease simulations on the artificial stationary network. Disease was initiated in each camp for a total of 72 simulations. For all simulations, we calculated the final epidemic size and epidemic duration and compared these results with output from disease simulations across the empirical dynamic network.

2.6. Disease simulations on random and small-world networks

We generated random and small-world networks with 72 nodes using commands in the *iGraph* package (Csardi and Nepusz, 2006) in *R* version 3.1.1 (R Core Team, 2017). Commands used to generate each network can be found in Table 1.

After these three theoretical networks were generated, their diagonal elements were set to one, to ensure that within herd transmission would occur and to mimic the empirical dynamic network. The parameters in the network generating commands were chosen to mimic the mean degree of the empirical dynamic network, which was 13.1. Due to constrains inherent in construction of a network with 72 nodes, we were not able to generate theoretical networks with a degree of 13.1; however, we were able to generate theoretical networks with a mean

Table 1
Generating theoretical networks in R using the *iGraph* package.

Network type	Commands
Random network	*erdos.renyi.game(72, 1/6)*
Power-law network	*barabasi.game(72, m = 6,directed = F)*
Watts-Strogatz network	*sample_smallworld(1,72, 6, 0.05)*

degree within 0.7 of the empirical dynamic network (13.3 for the random network; 12.4 for the power-law network; 13.0 for the Watts-Strogatz network).

Disease transmission was simulated across each network. Infection was initiated in each camp, which resulted in 72 simulations across the random network, 72 simulations across the power-law network, and 72 simulations across the Watts-Strogatz network. Each simulation across a theoretical network progressed until there were no infected individuals remaining. For all simulations across theoretical networks, we calculated the final epidemic size and epidemic duration in the same manner as for the simulations across the empirical dynamic network.

2.7. Ethics statement

The research protocol was reviewed and approved by the Ohio State University Institutional Review Board / Human Research Protection Program (Federal-wide Assurance #00006378 from the Office for Human Research Protections in the Department of Health and Human Services: protocol 2010B0004). We obtained verbal consent from participants after explaining the protocol and potential risks. The research was approved by the Ministère de la Recherche Scientifique et Innovation (MINRESI).

3. Results

3.1. Connectivity

The empirical network representing all mobile camps in the Far North Region, Cameroon, that visited the Logone Floodplain contained 72 nodes, representing 72 mobile camps, each consisting of one or more households and their herds of cattle. Undirected edges between nodes represented camps located at the same location. The network configuration changed throughout the ear. In August 2007, the network consisted of singletons, dyads, and multiple fully connected small groups that contained between 3 and 11 nodes (Fig. 2a). In early December 2007, all 72 nodes were connected by at least one edge (Fig. 2b). In late June 2008, the network began to break into several large groups containing many nodes and a rare small group or singleton (Fig. 2c).

Camp connectivity changed throughout the year when measured by weekly degree distributions. The shape of the weekly degree distributions varied widely (Figures S1 and S2). Three general patterns of weekly degree distributions emerged. First, there were U-shaped degree distributions: high numbers of nodes with a degree of one, smaller numbers of nodes with mid-range degrees, and high numbers of nodes with high degrees (Fig. 3a). Second, there were unimodal degree distributions that showed the opposite pattern: low numbers of nodes with small and large degrees but high numbers of nodes with mid-range degrees (Fig. 3b). Third, there were bimodal degree distributions, with two peaks at different mid-range degree values (Fig. 3c). The summed degree distribution was unimodal with a peak at the upper end of the mid-range degree values (Fig. 3d).

Camp connectivity changed throughout the year when measured by weekly network density. The empirical dynamic network changes in density throughout the year with a peak in September 2007, when pastoralists move from the rainy season to the dry season grazing areas (Fig. 4).

Camp connectivity changed throughout the year when measured by the size of the largest connected component. Initially, the largest component was relatively small; however, in weeks 8 though 18, the largest component contained all or nearly all mobile camps. The size of the largest component decreased throughout the remainder of the study period, except for notable large component sizes in weeks 19–21, 24–27, 29, 42, 45, 47, and 49–51 (Fig. 5). These results mirror our findings on network density (Fig. 4) except for the late large component observed between weeks 40 and 50.

3.2. Disease simulations on the empirical dynamic network

The final epidemic size was equal to the total number of cattle in 91.5% of simulations. The mean final epidemic size across all simulations on the empirical dynamic network of mobile pastoralists was 46724.68 livestock, or 99.9% of all individuals.

The mean epidemic duration for all simulations on the empirical dynamic network of mobile pastoralists was 75.4 weeks (1.45 years), but varied by the date of first infection and by which camp contained the first infection. Epidemics had the shortest duration when initiated in or before week 15. Epidemics initiated between weeks 15 and 35 varied in duration. In these cases, duration was greatly affected by which camp contained the first infected cattle. After week 35, epidemics displayed duration that was consistent regardless of the first infected camps and lasted for an intermediate duration (Fig. 6).

3.3. Disease simulations on artificial static network

Simulated final epidemic sizes on the artificial stationary network with a configuration based on week 52 ranged from 325 to 22,165 individuals (47.4%). The largest epidemic size corresponds to the size of the largest connected component. The mean final epidemic size was 12,875 individuals (27.6%), which is smaller than the mean final epidemic size for all simulations across the empirical dynamic network (Table 2). The longest epidemic lasted 1.87 years; the mean epidemic duration for all simulations was 1.5 years.

3.4. Disease simulations on random and small-world networks

The mean final epidemic size for simulations across random networks, a small world network with a power-law degree distribution, and a small world network with a Watts-Strogatz degree distribution was 100% of cattle (Table 2). The epidemic duration differed by theoretical networks (Table 2; Figure S3). The random network and the small-world network with a Watts-Strogatz degree distribution had shorter mean durations than the empirical dynamic network. The small-world network with a power-law degree distribution had a longer mean duration than those on the empirical dynamic network.

Comparison of simulated epidemics on the empirical dynamic

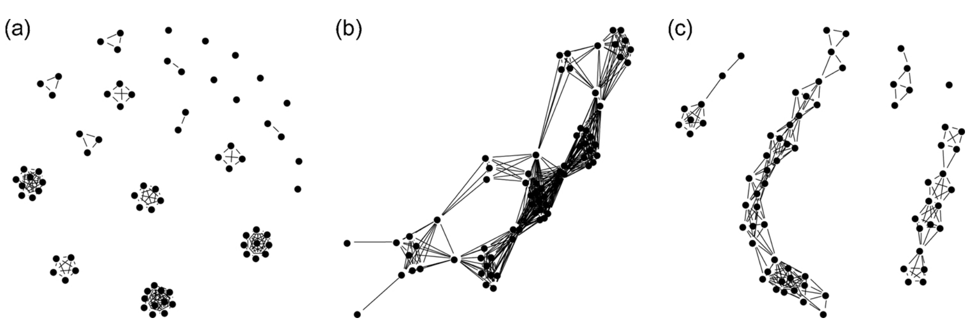

Fig. 2. Weekly configurations of the dynamic network. Edges break and form in the dynamic network because of transhumance movements with the result that the configuration of the network changes each week. Here, networks are drawn for (a) late August 2007 (week 2), (b) early December 2007 (week 17), and (c) late June 2008 (week 46) for illustration.

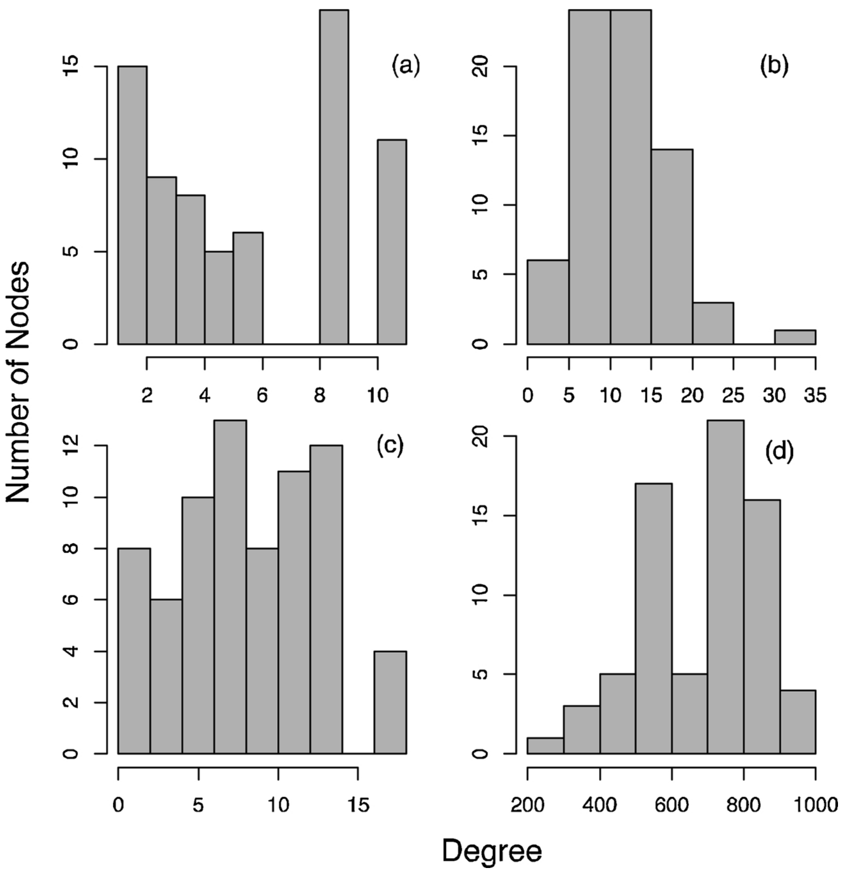

Fig. 3. Degree distributions for the networks of mobile herders. Degree distributions, which summarize connectivity by indicating how many edges each node has, varied by week. Here, we show three example degree distributions: (a) a U-shaped degree distribution in week one, (b) a unimodal degree distribution with the peak in mid-range degree values in week 22, and (c) a bimodal degree distribution in week 42. The summed degree distribution, in panel (d), is bimodal with a second peak of the distribution near, but not at, the maximum degree.

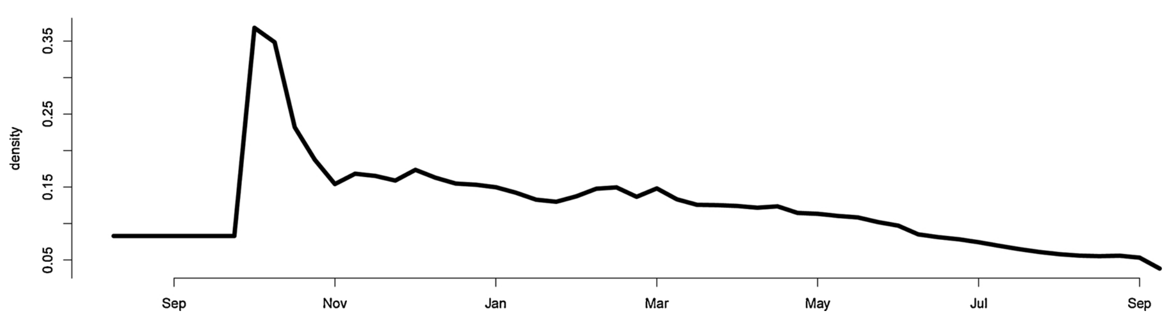

Fig. 4. Changes in network density over the year. Network density, calculated each week as the edge weight observed in the weekly network divided by total edge weight possible in a fully connected weekly network, from mid-August 2007 to mid-August 2008. Network density peaked in October 2007.

network, the artificial static network, and theoretical networks (random, small-world with power-law degree distribution, and small-world with Watts-Strogatz degree distribution) and by mean final epidemic size as a percentage of the total number of cattle and mean duration, in years.

4. Discussion

The primary goal of the paper was to examine how host movement affects foot-and-mouth disease virus transmission. Towards this goal, we analyzed transhumance data describing pastoralists' transhumance movements in the Far North Region of Cameroon as an isolated empirical dynamic network. We found that, for most of the year, camps were relatively dispersed and network density was low. However, there was a five-week period in September and October during which network density was high. Epidemics almost always infected all individuals, but those initiated before or during the high-density weeks infected swept through the population very quickly and had the shortest epidemic duration. These simulations suggest that, for the we mobility patterns observed, connectivity affects epidemic duration but does not affect epidemic size. Epidemics simulated across an artificial static network showed smaller final epidemic sizes but similar duration as those simulated across the empirical dynamic network, suggesting that transhumance affects epidemic size but not duration.

Our previous models indicated that mobile herders in the Far North Region of Cameroon maintained dispersed or ideal-free distributions when choosing their camp (Moritz et al., 2013a) and that epidemics among mobile herders showed multiple peaks in the same year but did not progress to endemicity (Kim et al., 2016). Our current study extended our previous work to investigate how these patterns changed seasonally. Similar to the earlier movement study (Moritz et al., 2013a), we also found that mobile herders maintained dispersed distributions for most of the year; however, our current study demonstrates strong seasonality in grouping behavior such that a period in September and October showed different – and higher – density. This seasonality in contact networks lead to seasonality in final epidemic sizes resulting from simulations of FMDV transmission across the networks. Similar seasonality in both contacts and disease simulations have been

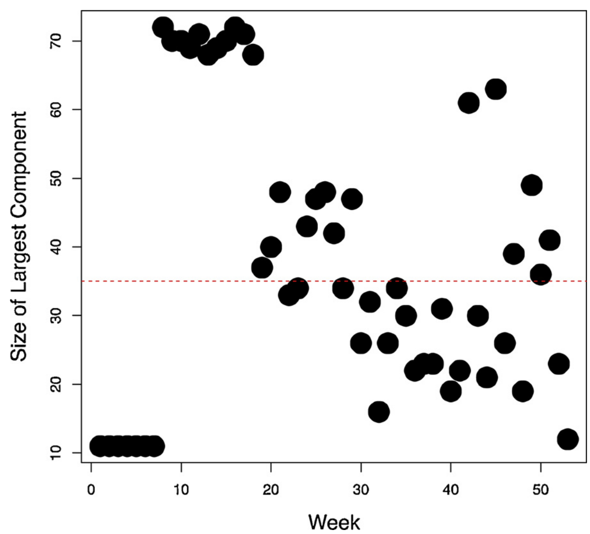

Fig. 5. Size of the largest connected component each week. The points represent the size of the largest connected component each week and the dotted red line represents the size a point would be if 50% of camps were connected in a single connected component. The largest connected component was small except for weeks 8 through 18, when almost all camps were connected. Notable large connected components with sizes greater than 50% of camps were observed in weeks 19–21, 24–27, 29, 42, 45, 47, and 49–51 (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article).

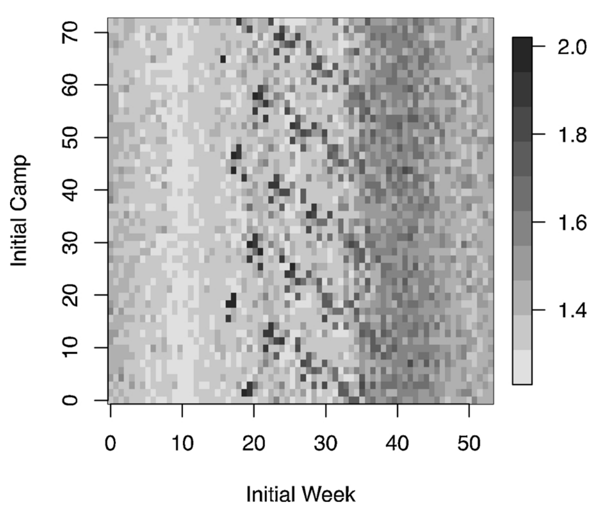

Fig. 6. Epidemic duration for simulated FMDV transmission across the dynamic empirical network of mobile pastoralists. Epidemic simulations were conducted for each combination of initially infected camp and week of initial infection; the duration of infection (in years) is depicted with the darker colors representing longer durations and the lighter colors representing shorter durations. Epidemic duration ranged from 1.27 years to 1.98 years. A seasonal pattern emerged, such that disease initiated in any individual in any camp before week 16 produced shorter epidemics than when disease was initiated in after week 35.

observed in other pastoralist systems (VanderWaal et al., 2017).

There is a period of high network density that occurs when pastoralists move from their rainy season grazing areas to their dry season grazing areas. During this time, pastoralists follow relatively fixed transhumance routes that take them through agricultural areas with millet and sorghum fields that have not yet been harvested. This bottleneck is not repeated at the end of the dry season, when pastoralists move to rainy season grazing areas, because an absence of standing crops in the agricultural fields means that they do not have to follow these fixed transhumance routes. It is bottlenecks that create situations where epidemics can sweep quickly through the mobile livestock population. This suggests that restrictions on livestock movements rather than the movements themselves can be a problem for livestock health and pathogen transmission and that it is thus important to protect the free movements of livestock within this transhumance system (Moritz et al., 2013b).

Table 2
Characteristics of infectious disease simulations across the empirical dynamic network of mobile pastoralists, the artificial static network, and the theoretical networks.

Network type	Mean final epidemic size	Mean epidemic duration
Empirical dynamic network	99.9 %	1.45 years
Artificial static network	27.6%	1.5 years
Random network	100 %	1.24 years
Power law network	100 %	1.64 years
Watts-Strogatz network	100 %	1.26 years

Our model was a simplified version of movement and transmission dynamics in the Far North Region, Cameroon because we assumed that all animals were susceptible. Because FMDV serotype O circulates consistently in our study population (Pomeroy et al., 2015), there might be individuals in each camp that are already immune. If this occurs, it would cause a smaller susceptible population than what our models assumed, causing our models to perhaps overestimate the final epidemic size and the duration of sustained transmission. For FMDV serotypes A, SAT 2, or SAT 3, which do not consistently circulate in our study population (Pomeroy et al., 2015), most individuals are likely to be susceptible, which aligns with our immunity assumptions.

Another simplifying assumption in our model was to assume that FMDV transmitted only among mobile camps in the absence of new births. Mobile camps comprise approximately 20% of the livestock in the Far North Region, Cameroon (Seignobos, 2000), indicating that most livestock are raised in a sedentary production system. Even though our data represented a network of mobile livestock, these cattle will likely encounter neighboring sedentary livestock or transboundary trade cattle, which are other risk factors for disease transmission (Dean et al., 2013) and might provide an opportunity for bidirectional transmission. Our model omits these possible sources of infection. If mobile camps acquire infection from sedentary or transboundary trade cattle, our model might not correctly estimate the duration of sustained transmission. Additionally, cattle are likely to be born during the 1.5 years that the simulated outbreaks lasted, providing additional susceptible animals that might increase final epidemic size or lengthen duration.

Nevertheless, we were still able to address the primary goal of this paper, which was to examine whether and how host movement contributes to transmission of infectious diseases. Host movement has long been implicated in disease invasion by contributing to a metapopulation rescue effect in which an infectious individual travels from an endemic location to a disease free location and (re)introduces infection that sparks an epidemic (Grenfell and Harwood, 1997; Metcalf et al., 2013). Movement throughout a metapopulation can lead to disease maintenance (Bolker and Grenfell, 1995) and shape temporal epidemic dynamics (Marguta and Parisi, 2015). Instead of modeling host movement and disease transmission in a metapopulation framework, we modeled host movement and disease transmission using spatially explicit dynamic networks drawn from empirical movement data. Across this empirical dynamic network, we also found that movement shapes connectivity and disease dynamics.

The secondary goal of this paper was to determine if the empirical dynamic network and disease simulations across it resembled three well-studied theoretical networks and disease simulations across them. We found that the duration of simulations across the empirical dynamic network differs from simulations across random and small-world networks. Random networks have been well studied; however, they are not observed in natural systems (Barabási and Posfai, 2016) and we were prepared for this natural system to display different connectivity patterns. However, small-world networks have been observed in a number of systems including metabolic and cellular organization (Jeong et al., 2000; Albert, 2005), the world wide web (Barabási et al., 2000), cattle movements in Uruguay (VanderWaal et al., 2016), and contacts among lions in the Serengeti (Craft et al., 2010). Other empirical studies of wildlife populations have found that contact networks vary in structure (Craft and Caillaud, 2011) and differ from theoretical network structures. Wildlife contact networks range from the giant connected component observed among Tasmanian devils (Hamede et al., 2009), to the dynamic contact patterns driven by female estrous events in wild chimpanzee contacts (Rushmore et al., 2013), to the changes in network structure driven by translocation events in Mojave Desert tortoise contact networks (Aiello et al., 2014). Our work shows that the empirical dynamic network of mobile pastoralists in the Far North Region of Cameroon also differs from small-world networks with either power-law or Watts-Strogatz degree distribution and is highly temporally variable, displaying strong seasonal patterns. We conclude that these well-studied theoretical networks act as a poor proxy for empirical dynamic networks and caution against making decisions about veterinary interventions in this population of mobile pastoralists based on anything other than location data.

Paradoxically, the majority of modeling studies that focus on FMD are based on livestock populations that are disease-free or experience infrequent epidemics and often in sedentary systems (Pomeroy et al., 2017). Movement, connectivity, and transmission in settings like the United Kingdom might not apply to disease processes among mobile pastoralists in endemic settings like Cameroon. In order to identify the role of mobility in FMDV transmission, multiple movement patterns need to studied directly using networks generated from empirical data.

Competing interests statement

The authors have no competing interests.

Author contributions

LWP, MM, and RBG conceived the ideas and designed methodology; MM collected the data; LWP, MM, and RBG analyzed the data; LWP visualized, quantified, analyzed the networks; LWP designed and coded disease simulations across the network; LWP led the writing of the manuscript. All authors contributed critically to the drafts and gave final approval of the version submitted.

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Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.epidem.2019.02.005.

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