

# Predicting the 2014 Ebola Outbreak in West Africa using Network Analysis

Final Report

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## I. INTRODUCTION AND MOTIVATION

The 2014 Ebola epidemic in the West African nations of Guinea, Sierra Leone and Liberia is the largest in history. As of writing this report, more than 17,000 suspected cases have been reported by the WHO (World Health Organization) and the numbers continue to rise. In this project, we explore the time-series data available for the current Ebola epidemic and perform analysis, model-fitting and short-term forecasting of epidemic spread at the local (country) level as well as at a world-wide scale by using economic trade network, geographical, and population data in concert with network and epidemiological theory.

Traditional epidemiological analysis is typically based on subdividing the population under consideration into different compartments based on the stage of the disease for each individual. The classic SIR model is based on three compartments - Susceptible, Infectious, Recovered. The underlying assumption of such analysis is random mixing among the population. Based on this assumption, each infected individual can infect any other susceptible individual in the population with equal probability. This compartmental model is a very useful tool for epidemiological analysis and is based on well established theory, and such analysis can provide sufficient insight into the temporal progression of an epidemic. However, due to the random mixing assumption, the application of such theory is somewhat limited when a large-scale world-wide analysis of an epidemic is required.

Network models, in contrast, allow for avoiding the random-mixing assumption inherent in compartmental models. This is done by assigning each individual a finite set of permanent contacts. Each individual in a network model can be represented as a node in a network while the edges represent the potential direct contacts between any pair of such nodes. For epidemiological analysis, clusters in a network model can be thought of as a group of individuals belonging to same local geographical areas (e.g. cities, countries etc.). Such modeling, therefore, inherently limits the number of direct contacts between individuals located in different clusters. The actual modeling of an individual's contact tracing is however a very difficult and time-consuming task and almost impossible at a large scale (for physical contact networks). The application of such contact network modeling is therefore limited to index case (patient zero) tracing in an post-epidemiological investigation. An alternative approach is to use some network with known characteristics - a random network, scale-free network, etc. - as a contact network for a population. Unlike compartmental models, however, the theory of network models for epidemiological analysis is still very young. We have explored using a simulation approach, however the theoretical underpinnings of such an approach are not yet well defined.

The third option, the current state of the art in epidemiological analysis, is a combination of the compartmental model with a network model. Such a model is useful in analyzing and forecasting large-scale spread of an epidemic. At a local scale, i.e. within a city or a country, a compartmental model is used to track the temporal progression of the city. A weighted network interconnects cities and countries to create the global model. The weight of the edges are proportional to the population inter-city/country migration. To capture the weight vector in the local compartmental model, a transportation operator is used.

We explore each of these options in this paper.

## II. RELATED WORK

### A. Compartmental Epidemiological model

The majority of research in epidemiological theory is based on the compartmental model. To model the progress of an epidemic in a large population, the individuals in the population are compartmentalized according to the state of the disease. The most widely used such model is the SIR model introduced in (Kermack and McKendrick, 1932):

- S (Susceptible): Individuals who have not yet caught the disease from contact with an infectious individual.
- I (Infectious): Individuals who have the disease. They have some probability of infecting susceptible people.
- R (Recovered): Individuals who have experienced the full infectious period, and are now non-infectious and immune.

The changes among these states over time are represented by a set of differential equations. In order to capture the dynamics of disease spread over time, a population-wide random mixing model is assumed, meaning that each individual has a small and equal chance of coming into contact with any other individual in the population. The basic reproductive number  $R_0$  is

defined as the average number of secondary cases generated by a primary case in a pool of mostly susceptible individuals, and is an estimate of epidemic growth at the start of an outbreak if everyone is susceptible. Almost all existing literatures (Chowell et al., 2004; Gomes et al., 2014; Legrand et al., 2007) on Ebola epidemic prediction are based on the modification of the basic SIR model.

In (Chowell et al., 2004), the authors model the effect of Ebola outbreaks in 1995 in Congo and in 2000 in Uganda using a variation of the original SIR model. A distinct feature of Ebola is that individuals exposed to the virus who become infectious do so after a mean incubation period. In order to reflect this feature, in the SIR model is extended with an additional “Exposed” compartment state. This SEIR model is reproduced in Figure 1:

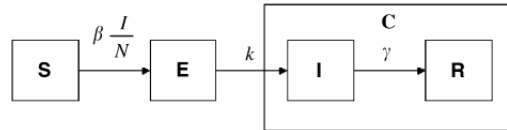


Fig. 1: SEIR model

In the SEIR model, susceptible (S) individuals in contact with the virus enter the exposed (E) state at a rate of  $\beta I/N$ . The exposed (E) individuals undergo an average incubation period of  $1/k$  days before progressing to the infectious (I) state. The exposed state is assumed to be asymptomatic as well as uninfected. Infectious (I) individuals move to the R state, either recovered or dead, at a rate of  $\gamma$ . The parameters are  $\beta$ , the transmission rate per person per day;  $N$ , the total effective population size; and  $I/N$ , the probability that contact is made with an infectious individual.

In (Legrand et al., 2007), the Ebola outbreaks in Congo in 1995 and Uganda in 2000 are also studied. However, they expand on the model from (Chowell et al., 2004) by modeling the spreading of Ebola in heterogeneous settings. In order to gain more insight into the epidemic dynamics, the infectious phase is subdivided into three stages: infection in a community setting (I), infection in a hospital setting (H), and infection after death assuming a traditional funeral (F). The resulting model is known as the SEIHDR model.

### B. Network-based epidemiological model

In the compartmental models described in the previous section, the underlying assumption is random mixing among the population. Therefore, an infected individual can infect any other individual in the population. Even though (Legrand et al., 2007) modified the SEIR model to reflect the heterogeneity of infection states, the underlying assumption is still random mixing. However, contagious diseases like Ebola spread via networks formed by physical contacts among individuals. While an individual may have the same number of contacts per unit time in either a random mixing model or a network contact model, within a static network model the set of contacts is fixed, versus a random-mixing model wherein it is continually changing. A static network model thus captures the permanence of many human relationships.

In our search for existing literature on a network-based model for the spread of Ebola, we haven’t come across any previous work that precisely does this, possibly due to the lack of detailed data in the locations historically affected by the disease. In (Newman, 2002), the authors provide the relationship between the compartmental SIR model and a random network model. The author introduces the concept of transmissibility in a network model. Transmissibility of a disease in a network model is defined as the average probability that an infectious individual will transmit the disease to a susceptible individual with whom they have contact. The authors also provide equations that connect the transmissibility and degree distribution of a random network with the basic reproductive number of an SIR model.

In (Meyers et al., 2005), the authors model the spread of the 2002-2003 outbreak of SARS in Hong Kong and Canada using a network model. A contact network model attempts to characterize every interpersonal contact that can potentially lead to disease transmission in the community. Each person in the community is represented as a node in the network and each contact between two people is represented as an edge connecting them. In (Meyers et al., 2005), the authors presented three different contact network models for SARS - (a) *Urban Network*: A plausible urban-setting contact network generated using simulation based on data from City of Vancouver, Canada. Households were randomly chosen and their members given ages, schools, occupations, hospital beds as patients, and caregivers according to statistics from public data.

### C. Combination of a compartmental and a network-based epidemiological model

As discussed in previous sections, a compartmental model is convenient in analyzing the temporal progression of a contagious disease in a localized population. However, due to the underlying assumption of random mixing among the population, such a model is not suitable for analyzing a large-scale outbreak of a contagious disease at a world-wide scale. An attractive alternative solution in analyzing the progression of disease in a world-wide scale is by combining a compartmental and network model. In such a formulation, the compartmental model is used on a local scale to track the progression of disease in individual countries

(or cities or continents). To capture the dynamics of inter-country (city or continent) spread of disease, some form of network data can be used. Such network data are usually converted to inter-country (or city or continent) transfer of population per unit time.

In (Balcan et al., 2010) the authors model the inter-country and inter-city spread of population using airport network data and commuting network data. The authors then use such a model in conjunction with a stochastic compartmental model to analyze the 2001-2002 seasonal influenza outbreak. A similar work (Gomes et al., 2014) attempts to predict the spread of Ebola to different parts of the world based on a model that incorporates both the compartmental approach and the use of world-wide air traffic flows. In this model, the world is divided into geographical regions defining a subpopulation network, modeling connections among subpopulations representing traffic flows due to transportation infrastructure.

### III. MODELING AND ANALYSIS OF INTRA-COUNTRY SPREAD OF AN EPIDEMIC

#### A. System Model and Algorithms

In the first phase of the project, to calculate the basic reproductive number of the current Ebola epidemic spread in different countries, we performed model fitting on the data we gathered from (Rivers, 2014). We considered the SEIR model described in (Chowell et al., 2004). The following set of differential equations are used to represent this model (Chowell et al., 2004):

$$\frac{dS}{dt} = \frac{-\beta SI}{N}, \quad \frac{dE}{dt} = \frac{\beta SI}{N} - kE, \quad \frac{dI}{dt} = kE - \gamma I, \quad \frac{dR}{dt} = \gamma I, \quad \frac{dC}{dt} = kE. \quad (1)$$

Here,  $S$ ,  $E$ ,  $I$ , and  $R$  denote the number of susceptible, exposed, infectious and removed individuals at time  $t$ .  $C$  is not an epidemiological state, however it is useful to keep track of the cumulative number of infected cases from the time of the onset of the outbreak. In order to model the effect of intervention on the spread of the disease, in the above model, the transmission rate  $\beta$  is modeled as a function of time. At the initial phase of the outbreak, before intervention,  $\beta$  is parameterized by  $\beta_0$ . After intervention, the value of  $\beta$  transitions from  $\beta_0$  to  $\beta_1$ ,  $\beta_0 > \beta_1$  as follows:

$$\beta(t) = \begin{cases} \beta_0 & t < \tau \\ \beta_1 + (\beta_0 - \beta_1) \exp(-q(t - \tau)) & t \geq \tau \end{cases} \quad (2)$$

where  $\tau$  is the time when interventions begin and  $q$  controls how quickly the rate of transmission changes from  $\beta_0$  to  $\beta_1$ . The SEIR model under consideration is a non-linear model with six parameters.

The current Ebola epidemic is still spreading and depending on the preventative measures taken, the underlying dynamics of the spread can change drastically any time. The Ebola data for the three most-affected countries in West Africa - Guinea, Sierra Leone and Liberia - were represented as  $(t_i, y_i)$ ,  $i = 1, 2, \dots, n$  where  $t_i$  represents the  $i$ th reporting time and  $y_i$  the cumulative number of infectious cases from the beginning of the outbreak of to time  $t_i$ . The model parameters  $\Theta = (\beta_0, \beta_1, k, q, \gamma, \tau)$  for these three countries were estimated using a non-linear least-squares procedure by fitting these data to the cumulative number of cases  $C(t, \Theta)$  in Equations (1), (2). In addition, we performed model fitting for the West Africa region by adding up the data from these three countries.

Given the limited number of data available, instead of fitting all six parameters to the model, we decided to fix some of the parameters based on studies on previous Ebola epidemics. In (Chowell et al., 2004), the incubation time of the Ebola virus  $1/k$  is found to vary between 1 and 21 days, with a mean time of 6.3 days for previous Ebola outbreaks. For ease of data fitting, we set this parameter value to the mean value of 6.3 days. We note that the dynamics of the current epidemic may differ from previous ones, and therefore fixing a value based on the prior estimate may lead to some inaccuracy. The choice of selecting initial outbreak time  $t_0$  and intervention time  $\tau$  is a difficult problem. To this end, we looked into different sources like WHO (World Health Organization) and CDC websites to learn more about the timeline of the spread. In Guinea, a 2-year-old boy died on December 2, 2013, later diagnosed as an Ebola patient. We consider this incident as the index case for Guinea and set  $t_0$  to December 2. On March 2, the Government of Guinea informed WHO regarding the possibility of an Ebola epidemic and declared a national health emergency. We considered this date as the intervention date and set  $\tau$  to 110. In Sierra Leone, one person died on April 2014. In June 12, 2014 the country declared an emergency and closed its borders with neighboring Guinea and Liberia. We consider the first date as  $t_0$  and second date as the intervention time, therefore set  $\tau$  to 50. In Liberia, on March 31, 2014, there was official confirmation of two people infected with Ebola. We set this date to  $t_0$  and set  $I_0$  and  $C_0$  in our model as 2. The government of Liberia shut down all schools on July 30, 2014. We consider this date as the date of intervention in Liberia and set  $\tau$  to 120.

The optimization problem involving the model fitting of the SEIR model is a non-linear least squares regression problem and contains a large number of local minima. Therefore, the choice of initial parameter estimate is an important consideration to get a globally optimal solution. In order to find a good initial choice of the parameters as input to the non-linear least squares solver, we first perform a Latin hypercube sampling on the 4-dimensional parameter space. We grid up the hypercube with a number of grid points in each dimension. We then choose the sample that minimizes the least squares error as the initial input. In order to calculate the 95% confidence interval of the estimated parameter, we performed bootstrapping based on residual error.

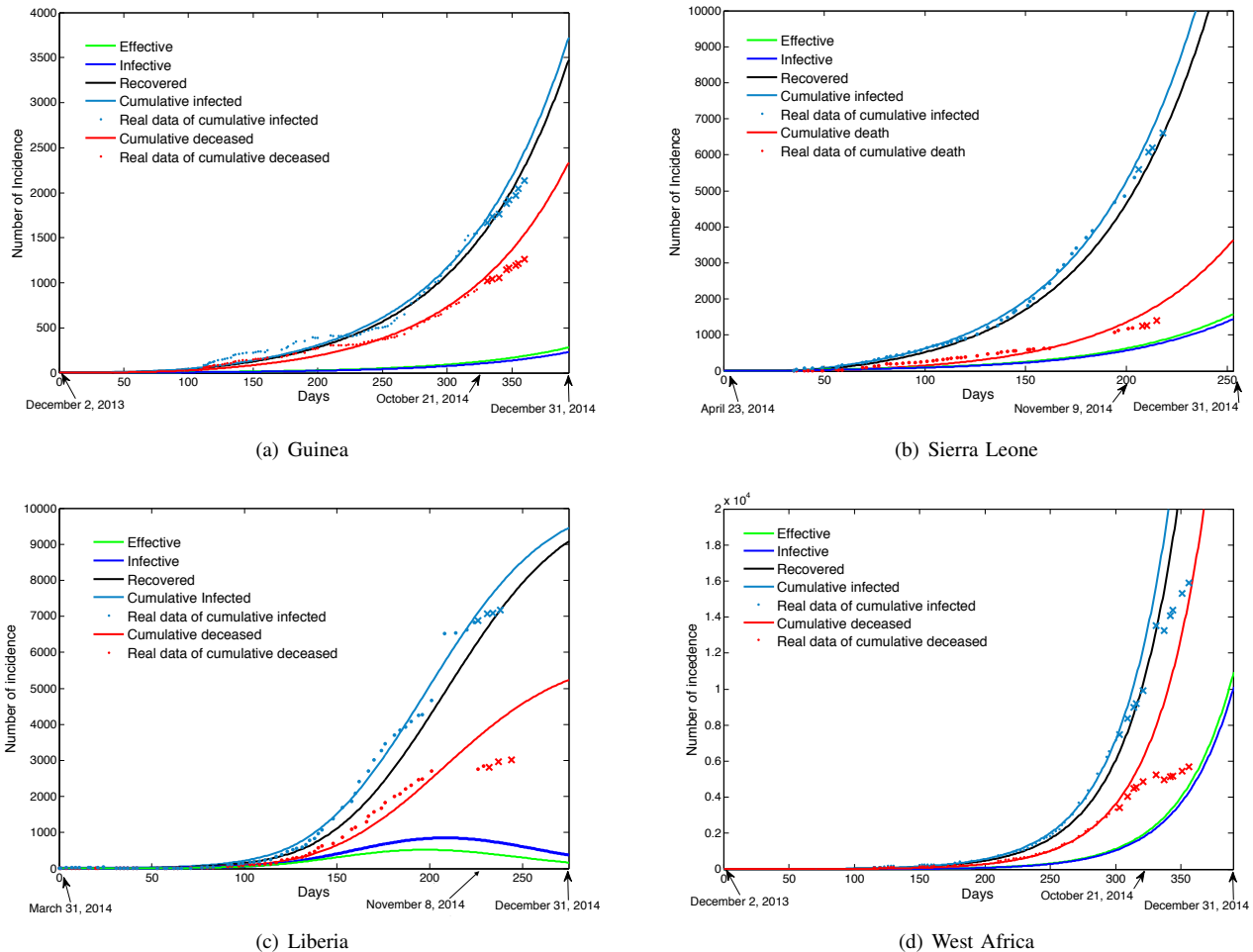


Fig. 2: SEIR model fit results for 2014 Ebola epidemic data

## B. Results and Findings

The basic reproductive number of the epidemic before and after intervention,  $R_0 \approx \frac{\beta_0}{\gamma}$  and  $R_1 \approx \frac{\beta_1}{\gamma}$  for the three countries are also presented in the tables. In Figure 2, we presented the number of incidents at different compartments of the SEIR model, as well as the cumulative number of infectious cases over time. In our project milestone report, our model fit was based on the last data we collected on Oct. 21, 2014. At the time of writing this final report, we have additional data points available. Based on data collected till November 9, 2014, we have updated our parameter estimation for Sierra Leone and Liberia. We, however, kept the same model for Guinea as in our milestone report, since we have the most amount of data available for Guinea already due to an earlier outbreak date in Guinea (Dec. 2, 2013). We extrapolated the graph up to Dec. 31, 2014. We note that forecasting future cases may not be accurate as the underlying factors of the epidemic are changing rapidly with the increase in safety measures. The unused data for Guinea (Oct. 21 - Dec. 5), Sierra Leone (Nov. 10 - Dec. 5) and Liberia (Nov. 10 - Dec. 5) are used for calculating test error. These values are also plotted in Figure 2 (points represented with ‘.’ sign are used for training, points represented with ‘x’ sign are used for testing). We observe that the predictions of the model to the cases up to Dec. 5 are mostly on par with the observed data for Guinea, Sierra Leone and Liberia. For the Guinea epidemic, we have data for the longest range of days and the estimated model, therefore, captures most of the dynamics of the spread in contrast to the other cases.

TABLE I: Estimated number of total infected people (by December 31, 2014) and root mean square error (RMSE) of prediction

Country	Estimated Number of Infected individuals	Cross-validation Error	Test Error
Guinea	3724	100.96	218.9
Sierra Leone	14040	170.14	546.2
Liberia	9500	350.3	504.24

In Table I, we presented our estimate of the total number of people who could get infected by December 31, 2014 as well

as root mean square cross-validation error of our model fit and test error on the unused data.

#### IV. ANALYSIS OF NETWORK BASED EPIDEMIOLOGICAL MODEL

##### A. Problem Definition and Algorithms

In Phase II of our project, we attempt to map the compartmental model to network based model using percolation theory to model the spread of Ebola in West Africa. The mapping between a compartmental model and a network based model is defined in (Meyers et al., 2005). Transmissibility  $T$  of a disease is defined as the average probability that an infectious individual will transmit the disease to an individual with whom they have contact. Critical transmissibility or epidemic threshold  $T_c$  is the value of transmissibility above which a population is vulnerable to large scale epidemics when the basic reproductive number  $R_0$  is 1. For our analysis, we reused the following expression from (Meyers et al., 2005):

$$R_0 = T \frac{\langle k^2 \rangle}{\langle k \rangle - 1}, \quad T_c = \frac{\langle k \rangle}{\langle k^2 \rangle - \langle k \rangle} \quad (3)$$

For simulating a contact network for Ebola in various infected countries we used a data set from a social networking site available on (UCI Machine Learning Repository, 2008). The undirected network has 4,846,609 nodes and 42,851,237 edges and average degree of 8.841. The network exhibits a power law degree distribution after a degree of approximately 50. We also generated three other datasets for a preferential attachment network, random network and a small-world network with approximately same number of nodes and average degree as the real network for comparison purposes.

Transmissibility values for each of three infected countries Liberia, Guinea and Sierra Leone in West Africa are calculated using Equations (3) and the reproductive number from Table ??, ?. These values are presented in Table II. We calculated the epidemic threshold  $T_c = 0.1275$  using  $k=8.841$  and used these values for running the simulations on the network datasets.

TABLE II: Transmissibility values for Liberia, Guinea and Sierra Leone

Country	$R_0$	Transmissibility(T)
Liberia	0.001	0.001
Guinea	1.145	0.1148
Sierra Leone	1.24	0.1243

Since our datasets were large and simulation using a percolation model is a compute intensive process, it was not possible to run the simulations in a reasonable time on a local machine so we ran our simulations on a c3.8xlarge Amazon EC2 instance. Our simulations needed to run for multiple values of transmissibility so we developed a test harness written in python to parallelize computation process-wise and leverage multiple cores on the machine. Our simulation using all four datasets and 20 different values of T completed in about 7 hrs for 200 iterations. We also ran another simulation to see the impact of minimum degree for patient zero for a given transmissibility value for two different networks.

##### B. Results and Conclusion

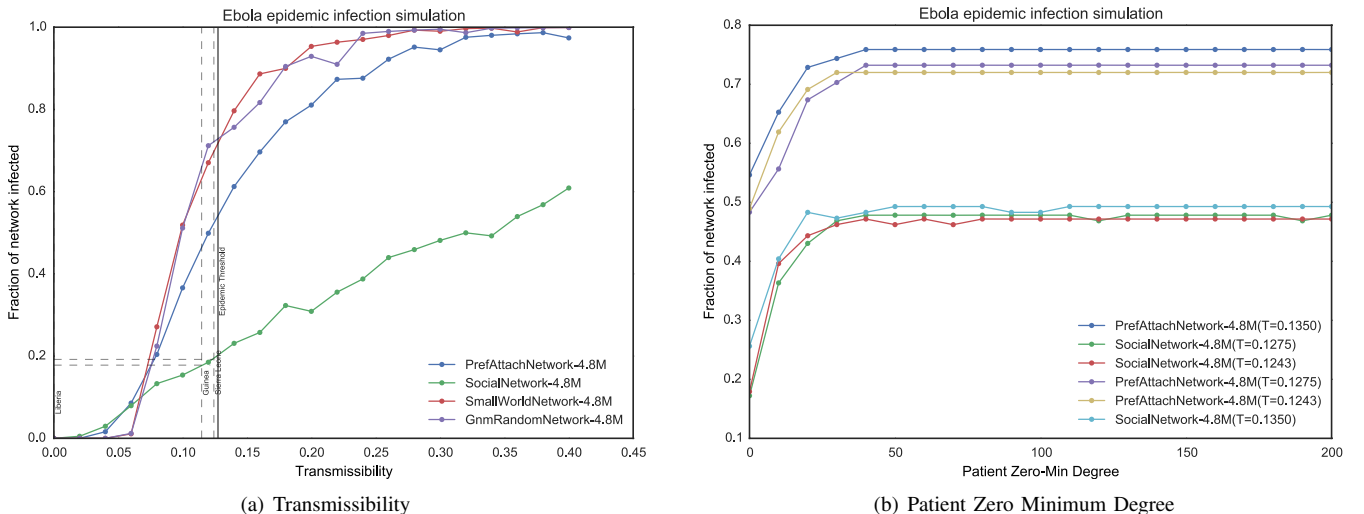


Fig. 3: Network Simulation for (a) Transmissibility, (b) Patient Zero Minimum Degree



In Figure 3(a), we show the fraction of network infected for different values of transmissibility for various network types. The simulation captures the overall fraction of network infected but does not capture any temporal progression for Ebola. Above the epidemic threshold the rate at which the fraction of the network gets infected increases for all network types and the rate is higher for other networks in comparison to power law networks which leads to a conclusion that Ebola outbreak is less likely to become an epidemic in these networks. This is contrary to the compartmental model assumption that an outbreak will always become an epidemic when reproductive number is greater than 1. For preferential attachment, small-world and random networks for value of  $T$  in the range of 0.35-0.40 nearly the entire network gets infected but not the real network.

In Figure 3(b), we show that for networks with power law degree distribution, the fraction of the network getting infected is not entirely dependent on the minimum degree of the initial node[patient zero] but also on transmissibility value. This is owing to the fact that the power law networks have majority of nodes with few edges or low degrees and small minority of nodes with high degrees or super spreaders. Since these high degree nodes are rare, outbreak at low transmissibility values may fail to reach these nodes. At higher transmissibility values above epidemic threshold, probability of reaching the super spreaders is higher leading to higher fraction of network getting impacted. The fraction become constant after a certain degree distribution, in our simulation around 50 since our real network follows the power law distribution approximately at degree 50. For predicting the total number of cases based on the network simulation we assume that a network similar to our real network exists in Guinea and Sierra Leone. Using a scaling factor proportional to the population we predict the total number of people infected at the current transmissibility value using the product of population, scaling factor and fraction of network infected. In Table III we present our results.

TABLE III: Estimated total number infected people

Country[Population]	Scaling-Factor	Fraction-of-Network-Infected	Total-Infected-People
Guinea[11.75M]	0.6545	0.178	136,889
Sierra Leone[6.2M]	0.3454	0.192	41,117

## V. PREDICTING WORLDWIDE SPREAD

### A. Problem Definition

In order to simulate how the Ebola epidemic might spread across the world, we have assembled a worldwide network based on international trade and border data.

### B. Trade network data preparation

The trade network dataset we are using is based on data retrieved from the UN Comtrade database (United Nations, 2014) via their web service API. The data queried for were country-to-country SITC-1 exports from the latest data available for each country.

Several problems with this raw dataset immediately became apparent which required working around. Many war-torn countries such as Liberia have not reported detailed export data to the UN in decades (Liberia last reported in 1984). As a result of this, the export totals are incorrect for the present day. In addition, this old data reports exports to countries that no longer exist, including East Germany and Yugoslavia. In order to make the dataset usable for modern-day predictions, we performed several transformations on the data (using Perl scripts).

We then needed to make the per-country exports sum up to the latest available export data. The United States (Central Intelligence Agency, 2013c) World Factbook contains up-to-date export totals for most countries in the world. Using the above data set, on the (admittedly strong) assumption that the export distribution from country-to-country in the UN Comtrade data set has remained the same between the last year a country has reported detailed export data and the latest totals from the CIA, we linearly renormalized each outgoing edge in our data set so that the total sum of exports equalled the latest CIA data for each country. Next we needed to map these numbers to theoretical international travelers. We could not find complete, or even nearly complete, publicly available data on the number of international outgoing travelers per country, especially for Ebola-affected countries. While it may have been possible to attempt to get this proprietary data via some other route, we chose to spend our time getting local data and running simulations instead.

Our approach to map outbound dollars to outbound travelers is to assume a linear relationship between these numbers, and also to assume that the same linear relationship holds for imports to inbound tourists to a country. We currently use the United States as a model and use the ratio of imports to the United States per year to the number of tourists visiting the United States per year. Based on data from the (US Office of Travel & Tourism Industries, 2013), 69.77 million people visited the United States in 2013. According to our renormalized data set, total imports into the United States were \$2.21 trillion during the same period. Dividing imports by visitors (an admittedly simplistic approach) gives us a scaling factor of approx. 31,665. Therefore we have applied this scaling factor to all edges in our international exports network, giving us some approximation of outbound travellers from country to country based on export numbers. One benefit of this coarse method is that it may help

capture non-flight-related travel. Interestingly, the trade network-based approach turned out to be fairly useful and helped us generate some interesting results.

We show a subset of the trade network in Figure 4, specifically the top 10 trading partners for the three countries currently affected by the epidemic: Guinea, Sierra Leone, and Liberia.

### C. Supplementing the trade network with local migration

In order to model local migration for cases where the economic impact is low, we also approximate population movement across land borders. The length of the land border between each country is available from (Central Intelligence Agency, 2013a), and the population of each country is available from (Central Intelligence Agency, 2013b). We wrote scripts to normalize the country names and come up with an estimate of population movement across borders for each country due to commuting. Commute data between two given countries is not very readily available, except for certain regions with a high amount of cross-border commuting. One such data source is (SCB et al., 2013), which provides cross-border commute statistics for Nordic countries. This database shows that approximately .12% of Denmark workers commute to Norway, while approx. .01% of Norwegian commuters commute to Denmark. Due to the open borders between these countries, the number for Denmark to Norway is probably on the high side overall, and due to the disparity in numbers, the traffic seems clearly one-way. Based on looking at the generated data, we assumed that the average number of workers worldwide that commute to another country is 0.02%. We assume roughly 50% of a population of a country is employed; the rest are children, the retired, homemakers, the unemployed, etc.

Based on the above assumptions, for each country we multiply population times  $.5 * .0002 = .0001$ , giving us the number of daily commuters, and then we split that across neighboring countries proportional to their share of land border lengths. This provides an estimate of daily cross-border commuters per country, which we use to connect neighboring per-country networks with edges corresponding to that many commuters.

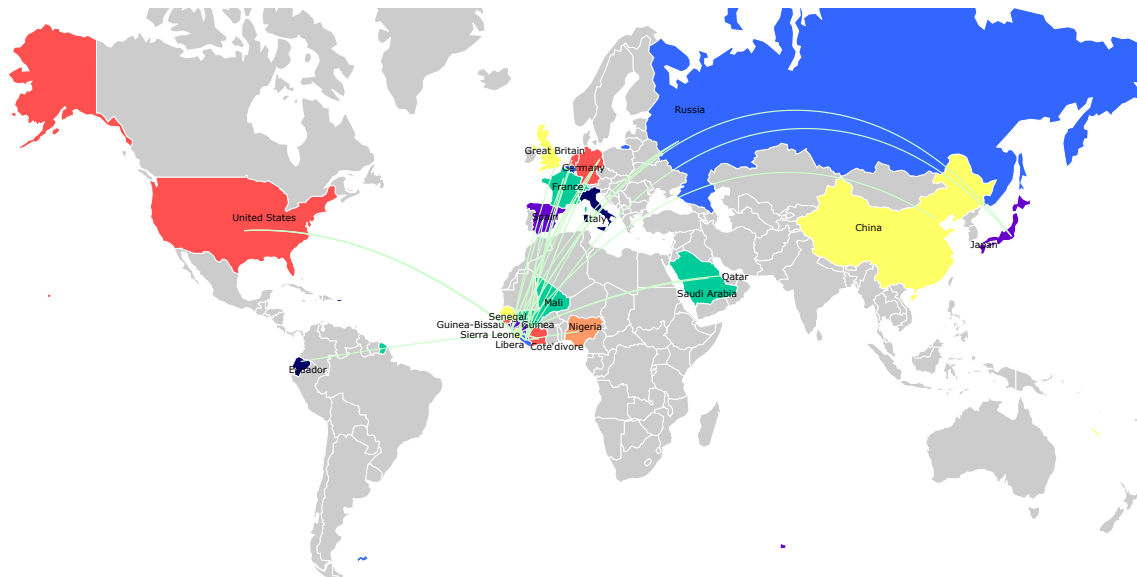


Fig. 4: World-wide trade network from Guinea, Sierra Leone and Liberia

### D. Network Simulation using the SEIR Model

We also did large-scale experiments on two 25M node "global" networks representing the population of the Earth, with subnetworks per country. This networks represent a scaled-down version of the population of the Earth, with a subgraph for each country, number of nodes per subgraph proportional to the population of that country, and number of edges connecting each subgraph based on the international migration numbers calculated in Sections V-B and V-C.

The first global network we built was based on the real-world network from (UCI Machine Learning Repository, 2008), which is a 4.8 million node undirected social network with an average degree of 10. We built each country as a subgraph based on this network. China, the most populous country with 1.36 billion people (Central Intelligence Agency, 2013b), was represented as the full social network, without removing any nodes. This scale-down factor, of 1.3 billion to 4.8 million, was 279.72. For each successive country we removed random nodes from the social network to get it to the right scale relative to China (for example, India was represented by 4.4 million nodes. The final global graph ended had 25,649,313 nodes. We then wrote scripts to renumber the nodes in these subgraphs to form a unified global graph. We added edges between each subgraph

according to the migration numbers calculated previously (the sum of the trade network and border numbers), selecting nodes from within each country’s respective subgraph randomly. Finally, we added a self-edge for each node, which allowed us to implement time-stepping and state-based node transitions without receiving a message from another node within the simulation environment’s Pregel-like algorithm (Malewicz et al., 2010). The resulting number of edges for this global network, including self-edges, was 117,537,887.

For comparison, we built a second network, this one fully synthetic and based on preferential attachment. The primary difference in construction was that we generated each country’s subgraph from scratch (no node deletion) to achieve the desired number of nodes (same as above), while maintaining an average degree of 20. Subgraph connection and additional postprocessing was done in the same way as above. The number of edges in this graph was 233,017,479 (this is due to the preferential attachment generation script incorrectly assuming undirected edges - an oversight).

We carried out experiments on this data using a discrete time-step SEIR simulation model, where each time step represents one day. In order to minimize the number of variables considered, we chose an exposed / incubating period of 11 days and also an infectious period of 11 days. At each time step, each infectious individual has some probability of infecting each one of his susceptible contacts, which will cause that individual to transition from the susceptible to the exposed stage, after which they will eventually transition to the infectious stage. To handle this larger-scale model, we chose to implement the simulation using the GraphX library (Xin et al., 2013). These simulations were run on a 12-node Spark cluster on Amazon EC2.

### E. Global Network Simulation Results

The first thing we noticed regarding the social network-based global network was that, when seeing with an initial patient zero in Guinea, an epidemic usually did not occur. Actually less than 5% of the simulations, even run with large transmission probabilities, ended up spreading to other countries. On the one hand, we think aspects of this are realistic - Ebola occurs naturally, yet we do not often hear of an epidemic. On the other hand, it’s possible that the network became excessively fragmented due to the graph generation procedure.

The fully synthetic global network based on preferential attachment, on the other hand, became an epidemic every time. In fact, we repeatedly ran into memory errors on the cluster at larger iteration counts due to this, however that may be due to being new to GraphX. A sample of our simulation results can be found in Figures 5(a) and 5(b).

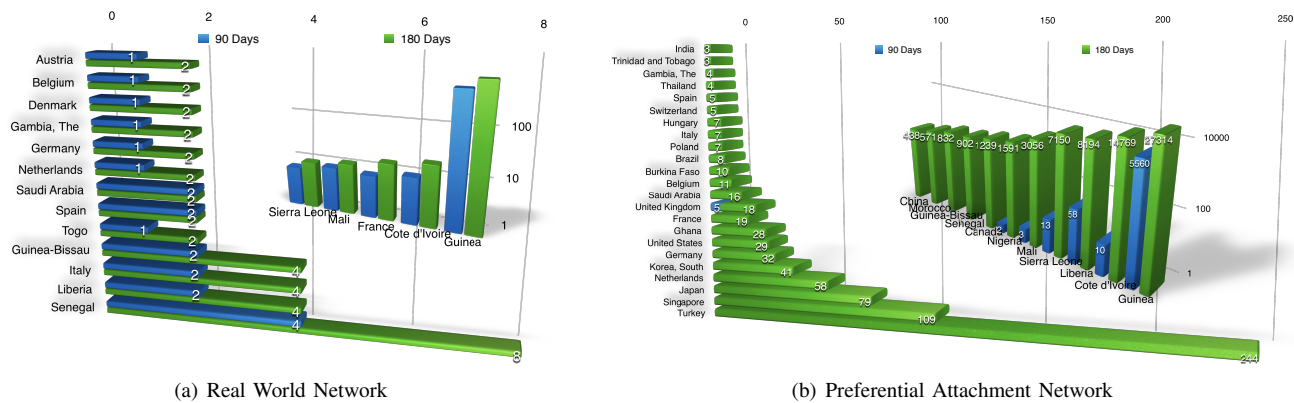


Fig. 5: 90 days and 180 days Prediction using World-wide Network Model

### F. Inter-Country Stochastic Compartmental Model

To incorporate inter-country spreading behavior, we modified the SEIR differential equations in (1) to include a transportation operator  $\Omega$  term. Similar approaches have been used in previous literature on epidemiological theory (Balcan et al., 2010; Grais et al., 2003). We assume that an individual who is in the Susceptible or Exposed stages of Ebola can travel. Due to the severe nature of Ebola, an individual who is in the Infected stage of the disease may not be able to travel. Even if such an individual travels to another country, due to strict regulations and monitoring at this time, such an individual will most likely be quarantined, and therefore will not likely spread the disease among the population of the country traveled. We therefore do not include the transportation operator in the differential equation corresponding to the infected stage. We define  $\sigma_{i,j}$  as the number of people travelling from country  $i$  to country  $j$  every day. We acquire the exact value of  $\sigma_{i,j}$  from the trade network described above. Using this value, the modified differential equations are presented below:



$$\begin{aligned}
\frac{dS_i}{dt} &= \frac{-\beta S_i I_i}{N_i} + \underbrace{\sum_{j=1, j \neq i}^K \left( \frac{\sigma_{j,i}}{N_j} S_j - \frac{\sigma_{i,j}}{N_i} S_i \right)}_{\Omega}, \\
\frac{dE_i}{dt} &= \frac{\beta S_i I_i}{N_i} - k E_i + \underbrace{\sum_{j=1, j \neq i}^K \left( \frac{\sigma_{j,i}}{N_j} E_j - \frac{\sigma_{i,j}}{N_i} E_i \right)}_{\Omega}, \\
\frac{dI_i}{dt} &= k E_i - \gamma I_i, \quad \frac{dR_i}{dt} = \gamma I_i, \quad \frac{dC_i}{dt} = k E_i, \quad \text{for } i = 1, \dots, K
\end{aligned} \tag{4}$$

In Figure 4, we present the world-wide network under consideration. Here, for each of the three major Ebola affected countries in West Africa - Guinea, Sierra Leone and Liberia, we have an weighted edge to the selected countries with most amount of population movement. Although not shown in the figure, in our simulation, we also consider the weighted edges between the countries which are reachable from any of the three countries - Guinea, Sierra Leone, Liberia. The parameter values  $\Theta = (\beta_0, \beta_1, k, q, \gamma, \tau)$  for Guinea, Sierra Leone and Liberia are set to the estimates from our Phase 1 results. The parameters for the rest of the countries are unknown. For these countries, we reuse the estimated value of West Africa from Phase 1. We set  $t_0$  to December 2, 2013 - the initial outbreak date in Guinea and initial number of infected individual at Guinea to one and to the rest of the countries to zero. We then run the simulation based on the differential equations in (4) for the duration up to December 31, 2014.

### G. Results and Findings

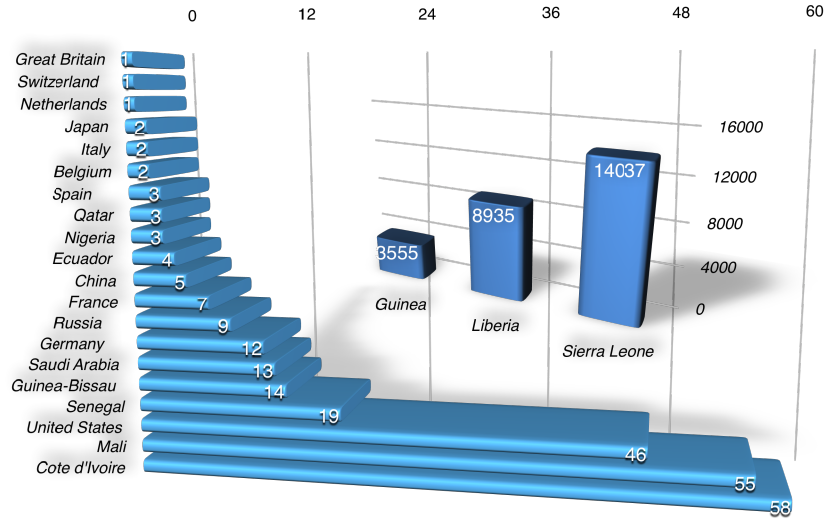


Fig. 6: Potential countries that could get infected and potential number of infected by December 31, 2014

In Figure 6, we present the potential number of infected people by December 31, 2014 in countries other than Guinea, Sierra Leone and Liberia based on our world-wide simulations. The number for Guinea, Sierra Leone and Liberia are similar to the result presented in Table I in Phase 1 as is expected. We note that, the modified trade network data that we used is a simplistic realization of the very complex human migration pattern and does not take into account intricacies involving border restrictions, enhanced monitoring, religious and cultural missions, etc. Therefore, results obtained from our simulation will rather represent an upper bound on the potential outbreak of the disease. Alternatively, we can interpret the results as a measure of relative risk of disease spread in different countries. It is, however, interesting to see that the result obtained from such a simplistic model represents the reality fairly well. So far, in addition to Guinea, Sierra Leone and Liberia, Ebola has spread to Mali, Senegal, Nigeria, United States and Spain. Our simulation result identified all of these countries successfully. In our simulation, Cote d'Ivoire is identified as the country with the highest risk of spread apart from Guinea, Sierra Leone and Liberia. However, so far Cote d'Ivoire is Ebola-free, even though it shares a border with all of these three infected countries. The reason for this is not clear, but it is reported in the media that very severe restrictions on border movement are in place there. Our simulation result indicated 3 people to be infected in Nigeria, whereas 20 people got infected in reality. The reason behind such small number is the lack of sufficient trades as well as no shared border between Guinea, Sierra Leone, Liberia and Nigeria. So far, the Lagos region of Nigeria has been affected the most by Ebola. The airport located in the Lagos region is the busiest hub

in West Africa. It is possible that migration through the Lagos airport may be related to the spread of disease in that region, which is not captured in our model.

## VI. CONCLUSION

In this project, we analyzed the 2014 Ebola epidemic in West Africa using epidemiological and network theory, as well as experimental simulation. We divided our work into three phases. In phase 1, we analyzed the data sets of a number of Ebola infected individuals to fit an SEIR model for Guinea, Sierra Leone, and Liberia as well as the West African region. We then used these models to perform short-term prediction of the temporal progression of Ebola epidemic in Guinea, Sierra Leone, Liberia. In phase 2 of our project, we analyzed the spread of disease in four large scale networks - a preferential attachment network, real world social network, small world network, and random network with the aid of Percolation Theory. In addition to that, we created a very large scale world-wide contact network and attempt to see the progression of disease among different countries from a single infected initial node in Guinea. In phase 3 of our project, we created a world-wide human migration network using a combination of economic trade data and country border information. We then combined this migration network in an experimental time-step simulation model, in addition to the SEIR models from phase 1. For the combined SEIR model, we then ran the combined network to watch the temporal progression of the disease over the course of one year using a single initial infection in Guinea and provided short-term prediction of world-wide outbreak of Ebola. These final simulation results match the real-world data fairly well.

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