Disease Propagation on a Network: Pandemics in the Era of Air Travel

Nabil Iqbal

We study a simple model of infectious disease propagation on a network representation of countries linked by major air routes. Each country forms a node in the network and is governed by the classic SIR model, while human traffic between countries via airline travel forms the links between nodes and allows the disease to propagate from one country to the next. We use actual population and airline traffic data and investigate a simulated pandemic starting in Mexico. A basic finding is that due to the relative insensitivity of the SIR model to initial conditions, the network dynamics affect only the rate of onset of the disease and not the eventual magnitude of the pandemic.

I. INTRODUCTION

"It is only a matter of a few hours then until death comes [...]. It is horrible. One can stand it to see one, two or twenty men die, but to see these poor devils dropping like flies [...]. We have been averaging about 100 deaths per day [...]. Pneumonia means in about all cases death [...]. We have lost an outrageous number of nurses and doctors. It takes special trains to carry away the dead. For several days there were no coffins and the bodies piled up something fierce." [1].

This is taken from a letter by a US Army physician during the 1918 Spanish Flu pandemic. This horrific chapter in human history–when more than a fifth of the world was infected with a particularly virulent strain of influenza–resulted in the death of 50-100 million people worldwide in what has been described as "the greatest medical holocaust in history." Today we find ourselves at the beginning of a new potential pandemic of a new strain of H1N1, the same subtype of influenza that caused the Spanish Flu (and indeed a large fraction of normal seasonal influenza). Dubbed the "swine flu", this new strain has already been seen to spread quickly from its origin in Mexico to other countries in the world.

In this note we develop a simple mathematical model to describe the spread of infectious disease across the globe via air travel. We will model each country in the world as a node in a network, while links between nodes are formed by major air routes. Our emphasis is on the connectivity of this network and its effect on disease propagation, and thus we use actual airline and population data in our simulations. The actual evolution of the disease in each country is given by the standard SIR model; as described below, this is very simplistic and is not truly a good approximation to reality, making our model far too rudimentary to truly allow an accurate quantitative comparison to real-life data. Nevertheless, we encounter interesting results at a qualitative level; one central theme is that the network dynamics affect only the *rate* at which the disease spreads across the globe, and not the eventual magnitude of the pandemic. An overview of network theory can be found in [2], while far more sophisticated studies of disease propagation across networks include [3, 4].

The plan of the paper is as follows: in section II we review the classic SIR model. In section III we describe our generalization of the SIR model to describe disease propagation across multiple nodes that can communicate with each other. In section IV we describe our simulation and results, while we conclude with a brief discussion in section V.

II. SIR MODEL

We begin by reviewing the classic **S**usceptible-**I**nfected-**R**ecovered model (see e.g. [5]) for describing the time dependent spread of an infectious disease within a population. The population is divided into three parts whose relative proportions vary with time: susceptibles, whose proportion of the total is denoted by $S(t)$, who have not yet caught the disease and are vulnerable to it, the infected $I(t)$ who are currently sick, and the recovered $R(t)$ who once had the disease but have since "recovered" in that they are no longer contagious. This particular model is insensitive to the mechanism of "recovery"; i.e. if a patient dies of the disease, he will no longer be contagious and thus will be counted in $R(t)$ along with those who have returned to full health. The standard SIR model is given by the ordinary differential equations

$$\frac{dS}{dt} = -\nu r I S \tag{1}$$

$$\frac{dI}{dt} = \nu(rS - 1)I \tag{2}$$

$$\frac{dR}{dt} = \nu I. \tag{3}$$

Here ν is a parameter with dimensions of inverse time that sets the timescale of the spread, and r is a dimensionless parameter that determines how infectious the disease is; we see that the rate of increase of the fraction of infected people is proportional to the product of infected and susceptible fractions, with the parameter r denoting how easily the disease spreads from individual to individual. Note that the sum

$$S(t) + I(t) + R(t) = 1 \tag{4}$$

is independent of time, and we normalize it to 1.

This system is nonlinear, and so exact analytic solutions are not available; the evolution of a sample system

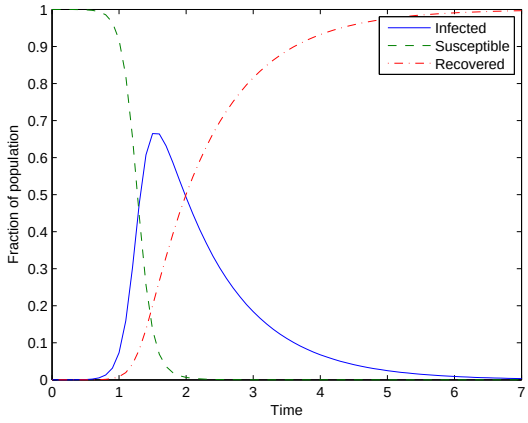

FIG. 1: Evolution of infected, susceptible, and recovered fractions of the population with time (here $r = 10$ and $\nu = 1$).

can be seen in Figure 1. Nevertheless, inspection of the equations allows a great deal of insight into the character of the model that will be helpful in understanding the behavior of the full international model studied later. First, we note that if $r < 1/S(t = 0)$, then

$$\frac{dI}{dt}(t = 0) = \nu(rS(0) - 1)I(0) \quad < 0 \tag{5}$$

and thus the number of infected people is decreasing at $t = 0$. However $S(t)$ is a strictly decreasing function of time, and thus we see that if dI/dt is negative at $t = 0$ it is also negative for all $t > 0$. The disease dies out without ever infecting anyone outside its original support base. Thus the size of the parameter r is of great importance in determining the outcome; in particular, we require $r > 1$ for a spread of the disease, and we will assume for the remainder of this paper that $r > 1$.

If $r > 1$, then the infected fraction rises before eventually beginning to fall, and it is possible to analytically determine the maximum infected fraction I_{max}. Dividing (2) with (1), we find (for $I \neq 0$)

$$\frac{dI}{dS} = -1 + \frac{r}{S}, \tag{6}$$

which can be immediately integrated to find I as a function of S and the initial conditions $I(0), S(0)$:

$$I + S - r\log(S) = I(0) + S(0) - r\log(S(0)) \tag{7}$$

Now note from (2) that we are at the maximum $dI/dt = 0$ when $S(t) = 1/r$; using this together with the fact that at $t = 0$ no one has yet recovered and thus $I(0) + S(0) = 1$, we find

$$I_{max} = 1 - \frac{1}{r}\left(1 + \log\left(\frac{r}{S(0)}\right)\right) \tag{8}$$

The important point in this expression is that the maximum infected fraction (and thus the severity of the outbreak) depends logarithmically–very weakly–on the initial infected fraction $I(0) = 1 - S(0)$, *provided that it is nonzero*. Of course if $I(0) = 0$ then the previous analysis does not apply, and $I(t) = 0$ for all t. If we imagine bringing $I(0)$ down to 0 from some nonzero value, the maximum I_{max} remains almost constant, while the time to reach this maximum grows larger and larger, eventually reaching infinity at $I(0) = 0$ (corresponding to no infection at all).

We note finally that while this may be a good model for the spread of disease in a population where all members have a reasonable chance of interacting with each other (such as a boarding school, or perhaps a very dense city), it seems that it is not appropriate it to apply this to an entire country as we have done. It is clear that in the United States an infected person in San Francisco will not infect someone in Boston; this constitutes a major failing of our model. A more careful treatment would split countries into much smaller interacting subsections (such as cities), perhaps modelling their interactions with the generalization of the SIR model described below.

III. SIR MODEL ON MULTIPLE NODES

We would now like to generalize the above construction to include many interacting populations. S, R, and I are generalized to n-component vectors, each component representing one country. Interactions between countries are included as follows:

$$\frac{dS_i}{dt} = -r\nu I_i S_i + \sum_j \frac{w_{ij}}{2N_i}(S_j - S_i) \tag{9}$$

$$\frac{dI_i}{dt} = \nu(rS_i - 1)I_i + \sum_j \frac{w_{ij}}{2N_i}(I_j - I_i) \tag{10}$$

$$\frac{dR_i}{dt} = \nu I_i + \sum_j \frac{w_{ij}}{2N_i}(R_j - R_i) \tag{11}$$

Here N_i is the total population of each country, and w_{ij} is a symmetric matrix that represents an actual flow of people per unit time between these two countries. To understand this term, we focus on (9) and imagine that of the $w_{ij}dt$ people who fly between countries i and j in time dt, $S_j w_{ij} dt/2$ of them will be susceptibles flying from j to i, and $S_i w_{ij} dt/2$ of them will be susceptibles flying from i to j. Normalizing the *change* in the number of susceptibles by N_i to obtain a fraction gives us the country-country interaction terms in (9) - (11). We note the following properties of this system:

1. If two countries with the same fraction of infected people are placed in air contact, this will result in no extra change in their relative levels of infection, in accordance with our intuition.

2. For each country, $S_i(t) + R_i(t) + I_i(t) = 1$ for all time; thus the overall populations of each country are not changed by the addition of travel.

3. In realistic situations, we expect $I_i \ll 1$ and thus $S_i \approx R_i \approx 1$ for each country. This means that the

bracketed travel terms in (9) and (11) are essentially zero and have no effect; the only real effect of travel is in (10) to allow infected people to propagate from one country to the next.

4. Finally, since we expect $w_{ij}/N_i \ll 1$, it is clear that even in (10) the travel term is important only if initial fraction of infection I_i is very small; once I_i begins to rise the internal dynamics of the internal SIR model will quickly dominate and drive the evolution.

From these facts coupled with our understanding of the single-node SIR model, we can now imagine what happens if we begin a epidemic with a small level of infection at one specific country (say Mexico). We expect the travel terms in (10) to propagate the infection from country to country; once a country is infected, its internal dynamics will drive it to the maximum level of infection given by I_{max} in (8) with $S(0) \approx 1$; thus every country will eventually face the same level of disease. The amount of time taken t_{max} to reach this maximum level will however depend on initial conditions for that country–i.e. on how far the country is from Mexico and the rate of flow of disease-laden tourists.

We now turn to numeric simulation to verify these facts.

IV. SIMULATION AND RESULTS

We now numerically solve the system of equations given by (9)-(10). We simulate an epidemic starting from a small number (relative fraction 10^{-5}, or roughly one thousand people out of the 106 million) of infected cases in Mexico, with an eye towards a situation representing the spread of H1N1 flu in May 2009. We thus take the matrix w_{ij} from actual airline data from the IATA[7], incorportating 204 countries with a total of 4083 nonzero entries in the matrix (representing country-country air travel) and use actual population data. We pick $r = 1.5$ [6] in agreement with the current understanding of the relevant parameter for H1N1. The value of ν is then found by fitting the small-time exponential behavior of $I(t)$ to time-dependent data[1] available for the number of cases in Mexico; we find $\nu = 2.37$ week^{-1}.

Note that while we are inspired by real-life numbers in picking parameter values for the model, our model is far too rudimentary to make possible any precise quantitative comparison with the data.

[1] The data was taken from the BBC outbreak map at http://news.bbc.co.uk/2/hi/americas/8021547.stm. for the number of cases in Mexico from May 5th to 12th, 2009. Prior to May 5th the number of cases does not fit the exponential growth expected and is far smaller; we suspect this is due to an incorrect diagnosing of the disease in its early stages.

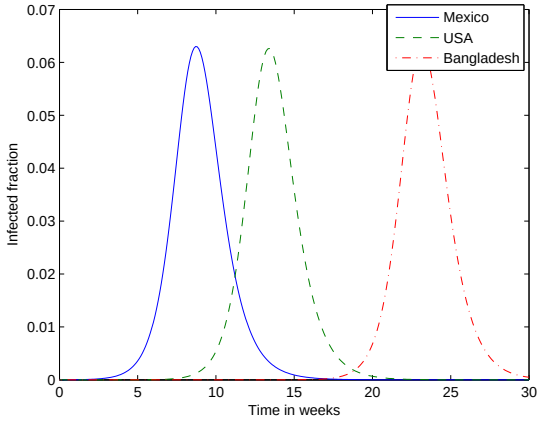

FIG. 2: The infected fraction $I(t)$ for Mexico, USA (a country with significant direct air travel from Mexico), and Bangladesh (a country with only indirect air travel links to Mexico)

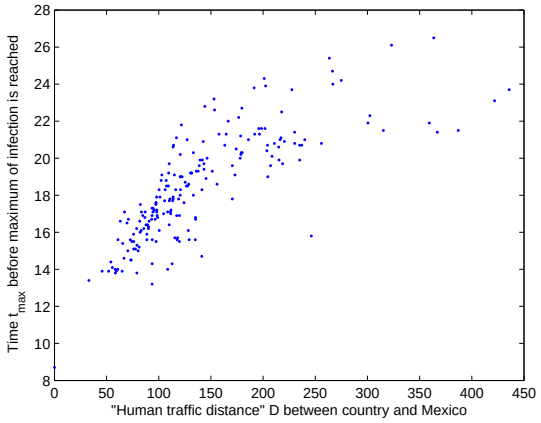

FIG. 3: Scatter plot of time to maximum infection t_{max} versus "human traffic distance" D from Mexico (see (13))

We find results that are in agreement with the qualitative picture given earlier. For example, in Figure 2 we show the time-dependence of the fraction of infected population of three different countries. We see that each country eventually peaks at a value of I_{max} which can be determined from the expression (8) with $r = 1.5$ and $S(0) \approx 1$:

$$I_{max}(r = 1.5) = 0.063, \tag{12}$$

i.e. a maximum infection level of about 6%, in perfect agreement with the simulation. We note that the USA (with a direct and strong airline connection to Mexico) peaks much earlier than Bangladesh (to fly from Bangladesh to Mexico, one must transfer at least once, leading to a correspondingly longer infection time).

In an attempt to make more precise this connection, we would like to introduce a metric on this network representing "how far" one country is from another in terms

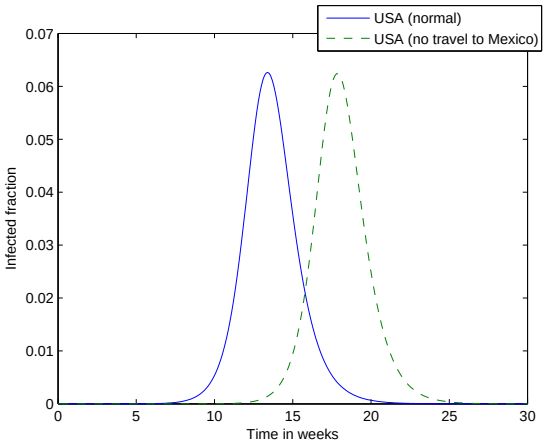

FIG. 4: Determining the effect of shutting down direct air travel between the USA and Mexico; note that the time to maximum infection is delayed.

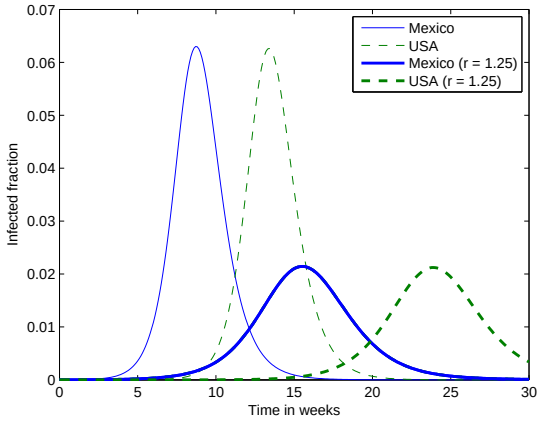

FIG. 5: Determining the effect of varying r; note significant slowdown and lessened impact of disease

of human traffic. We do this by introducing the following distance function:

$$d_{ij} = \sqrt{\frac{\sqrt{N_i N_j}}{w_{ij}}} \tag{13}$$

This is an appropriately normalized measure of inverse traffic; i.e. a large value of d_{ij} means that there is not much traffic between countries i and j, and a divergent value indicates that the countries are not directly connected by airline traffic[2]. Using d_{ij} as the cost matrix, we now use Dijkstra's algorithm (see e.g. [8]) to find the minimum "human traffic distance" D_i between each country i and Mexico. We also compute the time t^i_{max} for each country to reach its maximum level of infection, and plot t^i_{max} versus D_i. The results are shown in Figure 3; while there certainly appears to be a rough correlation between these two quantities, it does not appear that the "distance" is the only factor determining the rate of onset of the disease. We interpret this as being due to the high connectivity of the network; while the minimum distance does approximately measure how difficult it is to get from i to j, it does not tell us about the redundancy of paths, and if there are many possible paths with similar "distances" they will not significantly alter the measure D_i while they *will* have a significant effect on disease propagation. It remains an interesting open question whether there exist different measures of distance on the network that act as better measures of the rate of disease propagation.

Another interesting question that one can ask is whether attempts to contain the disease by curtailing air travel are effective. We investigate this by examining what would happen if the USA, upon learning of the epidemic, immediately shut down all air travel between the USA and Mexico. In the model above this draconian measure can be effected simply by setting $w(\text{Mexico}, \text{USA}) = 0$. In Figure 4 we display the results of such a practice on the infection levels of the USA; as we had expected, the eventual level of outbreak is not affected at all, but curtailing travel did have the effect of delaying t_{max} by approximately 4.5 weeks. The optimal disease-carrying path as defined above is now Mexico-Canada-USA.

Finally, we investigate the effects of varying r. We show in Figure 5 the effect of bringing r down to $r = 1.25$ from its previous value of 1.5; we see that this both lessens the magnitude of the epidemic and slows down its timescale, having a significant positive impact.

V. CONCLUSION AND DISCUSSION

Our model is very rudimentary, and thus we have not attempted a detailed quantitative match with existing data. However one clear qualitative finding of our study is that the dynamics of the network (strength of the connections, etc.) affect only the *rate* of onset of the disease. The *magnitude* of the eventual infection is determined by the internal dynamics of each node and not the connectivity of the network. We feel that this feature is likely to survive in a more detailed model.

This can help illuminate the wisdom of the World Health Organization [9], who explain: "*WHO is not recommending travel restrictions related to the outbreak of the influenza A(H1N1) virus... returning travelers who fall ill should seek appropriate medical care.*" This is in agreement with our findings–travel restrictions have a limited effect, and efforts should really be made not to alter the connectivity of the network but instead to reduce r, i.e. quickly treat ill people so that they do not

[2] The extra external square root is to help soften the large differences in this number from country to country

infect others.

Directions for future work are clear: a better understanding of the dynamics inside each country (rather than simply using the SIR model for each) is very important to obtain accurate results. Also, our model does not take into account the discrete nature of a single individual, focusing instead on a continuous flow of population; while these are equivalent in the limits of large population, for realistic (finite) population numbers the difference may well play an important role in the dynamics [3].

Finally, we conclude by noting that it appears that in our model there is little way to stop any disease from sweeping across the world. While in the currently relevant case of H1N1 this is probably not a serious problem as the disease appears to have a low fatality rate, we note that a more deadly disease with a similar capacity for spread could easily result in a global disaster; looking at Figure 3 we see that a mere six months after the beginning of the pandemic the disease has peaked in the most remote corners of the globe. The 1918 pandemic occured from March 1918 to June 1920, or more than two years; it is likely that a modern pandemic will require much less time to wreak its havoc.

Acknowledgments

We would like to think M. Kiermaier and M. Z. Iqbal for helpful discussions.

[1] S. L. Knobler, A. Mack, A. Mahmoud, S. M. Lemon, "The Threat of Pandemic Influenza: Are We Ready?" National Academies Press, Washington D.C. (2005), p59.

[2] D. J. Watts, "The "New" Science of Networks," Annu. Rev. Sociol. **30**, 243-70.

[3] V. Colizza and A. Vespignani, "Epidemic modeling in metapopulation systems with heterogeneous coupling pattern: Theory and simulations," J. Theor. Bio, **251**, 450-467 (2008).

[4] V. Colizza, A. Barrat, M. Barthelemy, A. Vespignani, "Predictability and epidemic pathways in global outbreaks of infectious diseases: the SARS case study," BMC Medicine, **5** 34 (2007)

[5] J. D. Murray, "Mathematical Biology," Springer, New York (2001)

[6] C. Fraser *et al*, "Pandemic Potential of a Strain of Influenza A (H1N1) : Early Findings," Science [DOI: 10.1126/science.1176062].

[7] International Air Transport Authority Passenger Forecast 2007-2011, http://www.iata.org

[8] T. H. Cormen, C. E. Leiserson, R. L. Rivest, C. Stein, "Introduction to Algorithms," MIT Press and McGraw-Hill, Cambridge (2001).

[9] World Health Organization website, http://www.who.int/csr/don/2009_05_13/en/index.html