The Random Walking Dead: Effects of urban street network topology on rates of infection spreading in zombie epidemics *

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June 21, 2013

We examine the effects of the complex structure of urban surface road transportation networks on the rate of infection spread through the human population in a hypothetical outbreak of an apocalyptic zombie epidemic. Various cities and urban spaces have diverse layouts and networks patterns (i.e. a grid layout versus a naturally evolved street network). We explore how network topology–the dynamics of a network–influences the spread of a zombie epidemic throughout an urban system. We find that a normal city street network fares about as well as a simple 2d grid, but inhibits infection-propagation as compared to random and scale-free networks.

1 Introduction

Zombies have become a cultural phenomenon in recent decades with an enormous amount of films, television shows, books, comics, and many other forms of entertainment dedicated to these undead, infinitely ravenous monsters. The rise in the popularity of zombies in modern culture has lead to an increasing amount of scholarship on the subject that has spanned multiple academic disciplines, including but not limited to mathematics, physics, computer science, political science, philosophy, and geography [9, 7, 8]. A main motivation for the use of zombies in all of these different disciplines is because zombies

*Report to the NECSI Summer School on Complex Systems Modeling and Networks, June 17–21, 2013
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are ridiculous enough to be interesting to a wide audience but are also a platform for talking about subjects and problems that might not be of immediately obvious interest to the general public. This engaging quality of zombies helps explain why the Center for Disease Control (CDC) initiated a public education campaign looking at how the nation and its citizens should respond to the zombie apocalypse [3]. Zombies can be thought of as an analogue to natural disasters, epidemics, and even terrorist attacks, allowing for the CDC to educate people on how to prepare themselves for such emergency events through the entertaining lens of the plague of the undead.

A critical aspect of preparedness for any disaster is knowing the timescales over which an agency must respond to contain the disease or the suffering of the people. With the goal of understanding this timescale in mind, we have chosen to analyze the rates of spreading of the zombie epidemic in hope that by understanding this spreading, we can also understand the timescales of other disasters. We have decided to look at the spread of zombies throughout networks as a proxy for how zombies might spread throughout a city network of streets (links) and intersections (nodes).

The rate of spreading is intrinsically dependent on the structure of the network the zombies are attacking. Social contact networks largely influence the spread of disease within a network [6]. Thus, we have chosen to explore how the topology of a network — the network architecture — affects the spread of epidemics, particularly the zombie apocalypse, within city road transportation systems.

Modeling the structural network of a city is crucial to understanding the geographic spread of a zombie epidemic. Both regular (road networks) and scale-free (airline) transportation networks have strong and weak ties to uninfected areas. We explore the road networks of various city types by introducing a single zombie into a network and analyzing the dynamic development of infection within the city types. Also, by implementing a single zombie infectant onto different kinds of networks we can analyze the relative timescales over which a population of susceptible humans will be converted to hordes of ravenous undead from which there is no recovery. We have put these times scales into a geographical context to show which types of networks and cities will be more robust and secure from the spread of an epidemic in hopes of encouraging further discussion and inquiry into how to better prepare for a catastrophic level epidemic.

2 Methods

2.1 Simulation

The epidemiological model we consider is shown schematically in Figure 1. It includes both linear diffusion of zombies and humans from node to edge (and *vice versa*, with different diffusion rate constants for the two species) and nonlinear conversion of humans

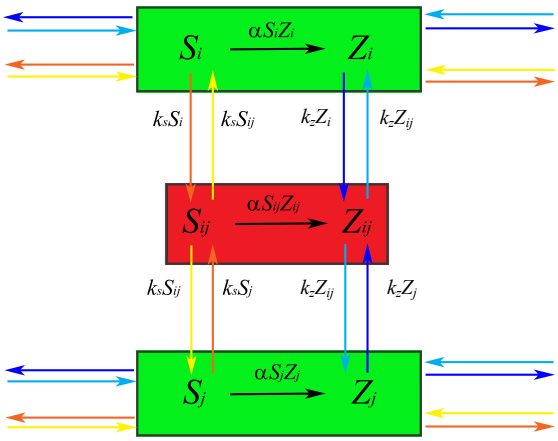

Figure 1: An epidemiological box diagram illustrating our model. The S species is the susceptible surviving humans, labeled with either a single specifying the network node or a double subscript indicating a link between two nodes. Likewise, species Z is the infected zombie population, similarly subscripted. The number of each species at each node and edge changes in time with a rate proportional to the terms indicated on the arrows. Both zombies and humans walk randomly to adjacent territories with rates proportional to their local number (simple diffusion), although zombies diffuse at a rate 5 times slower than humans. In each territory, humans turn into zombies with a rate proportional to the product SZ.

to zombies within each node and edge:

$$\dot{S}_i = -\alpha S_i Z_i - k_S d_i S_i + k_S \sum_{\text{edge}\to j} S_{ij}, \tag{1}$$

$$\dot{Z}_i = \alpha S_i Z_i - k_Z d_i Z_i + k_Z \sum_{\text{edge}\to j} Z_{ij}, \tag{2}$$

$$\dot{S}_{ij} = -\alpha S_{ij} Z_{ij} - 2k_S S_{ij} + k_S S_i + k_S S_j, \tag{3}$$

$$\dot{Z}_{ij} = \alpha S_{ij} i Z_{ij} - 2k_Z Z_{ij} + k_Z Z_i + k_Z Z_j, \tag{4}$$

where S_i and Z_i are the number of susceptible humans and zombies in node i, S_{ij} and Z_{ij} are the same for for edge connecting node i to node j, d_i is the degree of node i, and α, k_S, and k_Z are rate constants whose values define the model. We chose $\alpha = 0.1$, $k_S = 0.5$, and $k_Z = 0.1$.

The model was implemented using the PYTHON module NETWORKX and the simulation template of the PyCX dynamic simulation repository [1]. The differential equations were integrated using the Euler forward method with a time step of $\mathrm{d}t = 0.01$.

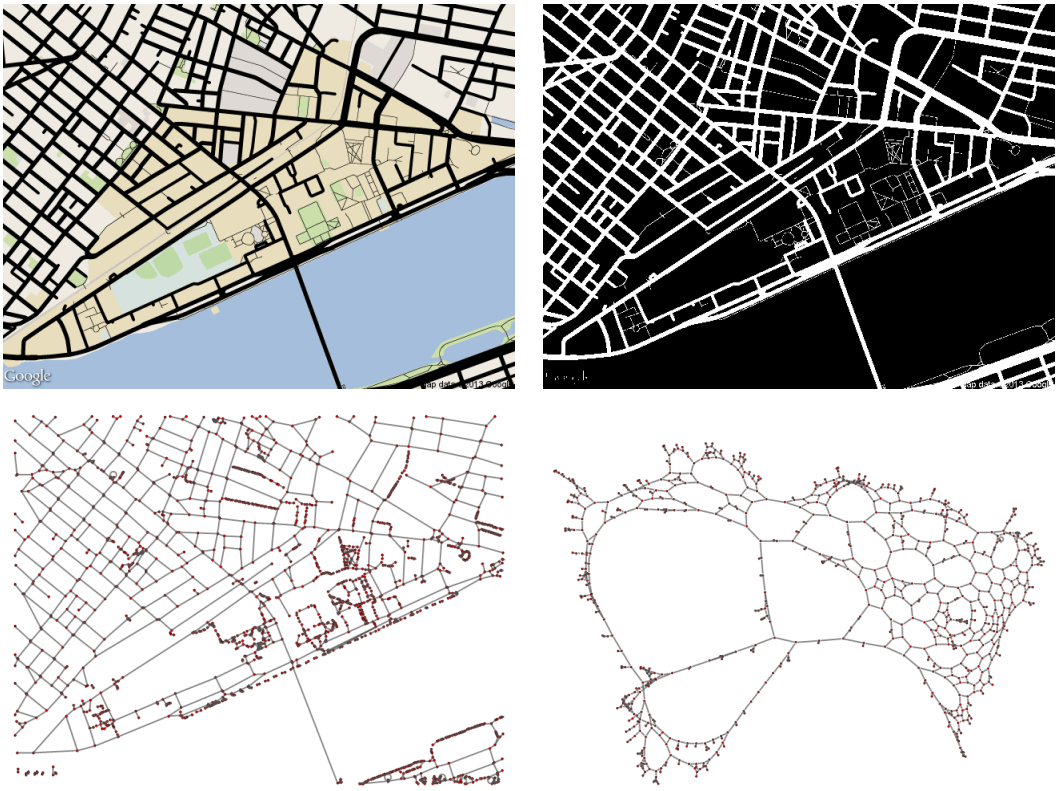

Figure 2: Road map network selection. A simplified map is generated by the Google API, binarized, and then processed for topological/connectivity information. Only the largest connected component is kept as the network representative.

2.2 Generating road map network graphs

To generate the city street networks we first generated a simplified map of the area using the *Styled Maps Wizard* of the Google Maps API.[12] The map image was then binarized (color values replaced by strictly black and white) and then processed through the MATHEMATICA function `MorphologicalGraph`, which finds the morphological branch points and endpoints and returns a graph (network) representation of the information. The resulting graphs were generally disconnected, so we selected the largest connected component to represent each city network. The result of each step is illustrated in Figure 2.

3 Results

We ran simulations on the various networks, each with populations of one zombie and 10^5 humans distributed randomly across the nodes and edges. The simulations ran until the human population dropped below one, which marked the "end of times" or "extinction

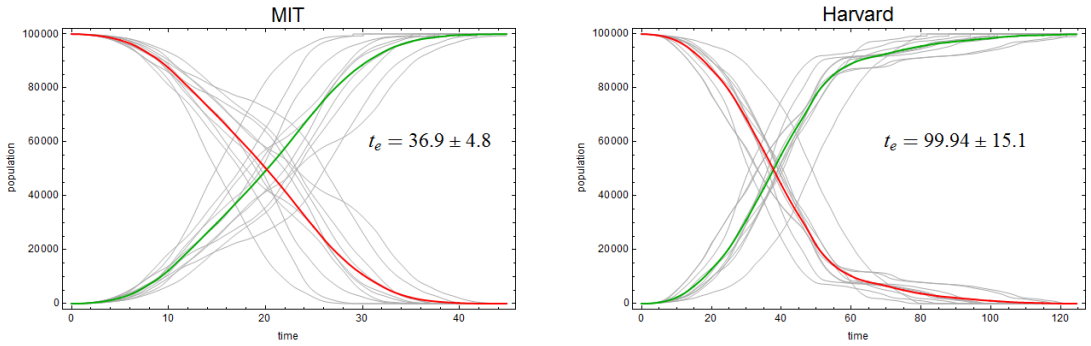

Figure 3: Extinction times for city street networks. Ten simulations were run for each city and are shown in gray. The simulation averages are show in red for humans and green for zombies.

time," denoted t_e. We ran the simulations ten times per city, with re-seeded random population distributions, to get statistics on t_e.

We first ran simulations on the MIT and Harvard street networks. The results are shown in Figure 3 and exhibit the characteristic "S" curves as the populations invert. The extinction time results were $t_e = 36.9 \pm 4.8$ for MIT and $t_e = 99.94 \pm 15.1$ for Harvard. Although the starting populations were the same, the two networks differed greatly in the number of nodes with $N_{\text{MIT}} = 849$ and $N_{\text{Harvard}} = 1578$. We speculate that since the network topologies were not too different, the enormous difference in extinction times is due to the ratio of population to number of nodes — a higher number of nodes leads to a more diffuse population and hence a longer diffusion time.

To better explore how network topology affects extinction time, we ran simulations on a few idealized networks with imposed properties similar to that of the MIT street network, in particular the same number of nodes. Again, we ran simulations for each network ten times, but before each run we re-initialized both the network (i.e. generated a new network) and the population distribution with the aim of characterizing the type of network rather than a specific instance of the network type.

First we chose a class of random networks, given by the Erdős-Rényi model with a link probability corresponding to an average of four links per node. Next we chose a class of scale free networks with preferential attachment, given by the Barabási-Albert model. As a benchmark for street grids, we also chose a simple 2d grid. Finally, for variety we chose a "random lobster," which is a mostly linear network with minimal branching. This network in particular exhibited a wide range of extinction times that depend strongly of the position of the starting zombie. The results are shown in Figure 4.

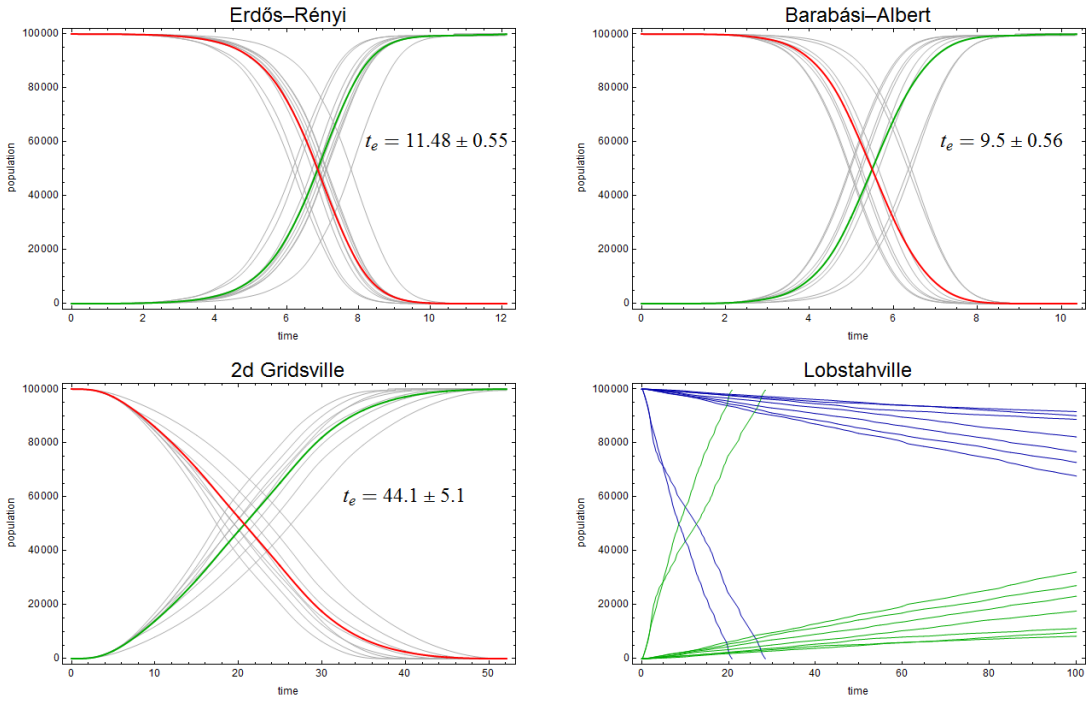

Figure 4: Zombie apocalypse on ideal networks. The extinction times for the random lobster network (Lobstahville) varied wildly depending on the starting position of the initial zombie. The simulations were stopped after $t = 100$.

4 Discussion

The simulations show a distinct difference in extinction times for the different network topologies (see Figure 5). The random (Erdős-Rényi) and scale-free with preferential attachment (Barabási-Albert) networks both exhibit relatively fast infection-spreading. This is potentially due to clustering, which allows for easy zombie saturation of the clusters and fast transport between clusters (low mean path length between clusters). The MIT street network fared about as well as a simple 2d grid, in which the human populations sustained for about four times longer than the random and scale-free networks. This suggests that the infection-propagation dynamics of city grids do not differ significantly from simple 2d grids, but inhibit diffusion more than random and scale-free networks.

The infection-propagation dynamics of the "random lobster" network, however, exhibit a rather different behavior. The extinction time depends strongly on the starting position of the initial zombie. Due to the linear structure of the network, a (fortunate for humans?) placement of the initial zombie toward the end of the network requires linear diffusion in one direction across most nodes and, due to the slow shambling of the zombies, takes a long time. In the simulations, we stopped the runs after $t = 100$,[1]

[1]This was mostly due to limitations of the authors' patience, but there were also instances of numerical

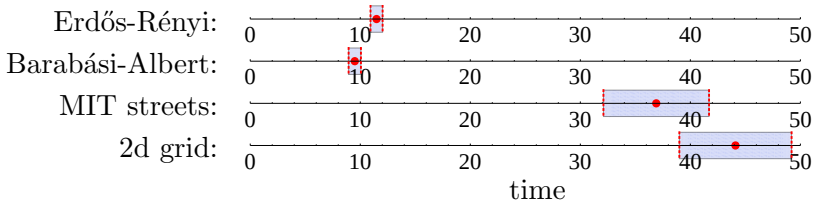

Figure 5: Network extinction times. Note the fast infection-spreading times for the random and scale-free networks and the similarity of the city street and 2d grid networks.

but these preliminary results show a more linear inversion of populations instead of the usual "S" curves. These results suggest that, although the extinction time is difficult to pin down, a random lobster network would allow humans a better chance against infection-spreading than normal city grids.

4.1 Model Limitations

We integrated the differential equations using the Euler forward method. Despite keeping a time step small compared to any relevant time scale in the system ($\mathrm{d}t = 0.01$ *vs.* time scales of several time units), the Euler method is susceptible to numerical instabilities. One manifestation of this in our model was that while the differential equations predict that the total population (human plus zombie) in the city should remain constant, our total population could drop by as much as 1% over thousands of integration steps. This simple, technical, shortcoming could be overcome with further research by simply switching from the Euler forward method to a more sophisticated integration scheme, like the fourth-order Runge-Kutta method.

There are a number of aspects of population dynamics which our model explicitly ignores: natural birth and death of humans; decay of zombies; season (temperature) variability of zombie mobility and mortality; specialization of the human population into labor pools with different zombie interactions, including zombie hunters and medical personnel; a refractory or latency period for infected humans; and so on. All of these model limitations are ameliorated by constraining the modeled scenario to the early stages of a "high contagion, slow zombie" outbreak, and simply asking that the model show relative rates of outbreak in different network topologies, rather than absolute times. Similarly, in a real street network, human or zombie bodies can only be packed into a crowd so tightly until they hit a maximum density at which no more individuals can be added, and diffusion out of an area is greatly reduced due to jamming. This model limitation is avoided by only considering total population densities which fall well below the crowding limit.

There are other limitations and simplifications of the model which are more serious

anomalies in which the populations reached an artificial oscillatory steady state (which is impossible for our model) such that the human population never dropped below the threshold.

shortcomings. One of these is hinted at in the title of this paper: our humans and zombies are uncorrelated random walkers whose motion on the network is characterized by only a pair of rate constants. Even within the simplified two-species epidemiological model considered here, correlated motion of the populations would likely be non-negligible. That is, instead of a number of individuals moving out of a territory during each time step in proportion to their number in the territory, humans would more likely run away from areas of high zombie number — moving down the gradient in the zombie population — while zombies would chase humans — moving up the gradient in the human population. This important effect is unaccounted for in our model.

The most serious limitation is perhaps the treatment of all roads and intersections as if they have the same size and shape: only the connectivity of nodes varies. In a more realistic model, diffusion out of an edge only occurs for beings close to the end of the roads, so the rate constant for diffusion out of edges should be suppressed relative to that of nodes by a factor of the ratio of the typical area of an intersection to the area of a street. Ideally, the model would allow this rate constant to vary from edge to edge. Similarly, the human-to-zombie transition terms are treated as being proportional to the product of population numbers, but this form for these terms should be derived as a product of population densities, and should again vary from edge to node by factors of area.

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