A Network Model of Alcoholism and Alcohol Policy

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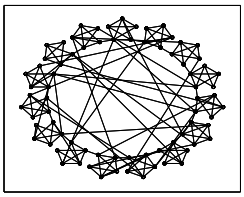

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Abstract

The evolution of alcohol dependence in populations of people on different social networks is studied. Two models are studied. One is the evolution of the states of individuals on hypothesized social structures from a rewired connected caveman model. This model spans a range of social structures (networks) from very ordered to effectively random with small world structures in between. The second model is a zip-code-level model which uses data from a recent survey in delaware. The model is a discrete model using 10 zip codes. The results show that the evolution of alcohol dependence, as governed by the simple rules that we use, depends sensitively on the network structure and a hypothetical treatment regime.

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1 Introduction

Data are available from random surveys of populations regarding alcohol abuse for relatively small geographic areas such as zip codes. This information is relatively reliable compared to information on hard drug abuse. Can these data be used to evaluate the ecology of the population and to help make effective policy decisions? We will use real data from recent surveys of the Delaware population [1]. With that data, we construct a social network consistent with those statistics and study the epidemiology of alcoholism with some known population structures (e.g., [2, 3, 4, 5, 6, 7]).

The key idea is to study the effect of previously-studied social structures on alcohol-related problems within a population. We would like to begin to answer the following question: What is the effect, if any, of different (social) connectivities on the spread and persistence of alcohol-related problems and what policy conclusions can be drawn from understanding this effect? We build on the pioneering work of Watts [3, 4] and Newman [6], who addressed similar questions in the case of infectious disease. We note that while alcohol dependence is a disease, the epidemiology is distinctly different from that of *infectious* disease. We do not intend to solve the riddle of what causes alcoholism; we aim to integrate data available for alcohol-related problems with the science of networks to provide ideas and tools for estimating policy costs.

As a beginning, we propose studying some very basic models. One possibility is to study the evolution of a fraction of a population on a network via a continuum model as discussed below. A continuum model in this case is a differential equation model for a population on the network; such models can be reduced to a single differential equation in some cases. Another possibility is to study the evolution of a population consistent with available survey data and simulate its evolution on different realizations of social networks.

In a simulation approach, we begin in the following way. A detailed survey data file is available on a sample of 2627 people with differing degrees of alcohol-related problems which can be read into MATLAB and subsets of the results can be extracted and used. A good beginning would be to try some social network configurations where some subset of the survey population is embedded into a larger population (consistent with survey and general population), and connect them with social interactions of a small world or power law form. Links are assumed because of population

characteristics: exposure to alcohol, gender, ethnicity, social stratification (occupation, education and income), and drinking norms. Then, using "rules" for changing the states of people (vertices) from an alcohol-dependent state to a healthy state or vice versa, we can see what, if any, differences occur in the population with different network realizations and rule changes. (Example rule: If a person has a majority of neighbors who are alcohol dependent, that person becomes alcohol dependent; if an alcohol-dependent person has a majority of neighbors who are healthy, then the dependent person goes into remission. If the person had an alcoholic parent, then perhaps only two alcohol dependent neighbors are needed to make healthy turn to sick, etc.). The social network must be hypothesized and sensitivity evaluated. We could also try adding a demographic model to change the age distribution of the population and then to modify the network as well (e.g., [8]); this is essentially a simulation that ages the population as it progresses. This last option is complicated, but it is a valuable ingredient for health planners.

To begin in a continuum approach, we could study the fraction of the population which is alcohol dependent, $\rho(t)$, with a single ordinary differential equation as in [5] used for an Susceptible-Infective-Susceptible (SIS) model; in this approach, the structure of the network affects the dynamics of the spread of an epidemic. We will modify the ordinary differential equation (ODE) to reflect the dynamics of alcohol dependence as opposed to an infectious epidemic. In the infectious case, the fraction of the population that is infected decays linearly, but for alcohol dependence, we could hypothesize either decay or growth.

After understanding the most basic single equation case, we can add in the effect of treatment. This can be accomplished by modifying the single equation, or by generalizing to a system where the infected population is divided into different categories. For example, we could have fractions of the infected population that are treated or untreated; we could also add mortality to the model. Policy decisions that impact initial or ongoing values of the proportion of treated individuals could be evaluated from the behavior of the models; it may then be possible to use the results to make policy decisions.

During the week of MPI2004 most of the participants focused their efforts on a network model of alcohol dependence. Overlaid on graph was Our model was a network, with each vertex representing

a person, and having a value v representing the degree of alcohol dependence. We then used a difference equation which changed a vertex's degree of alcohol dependence according to the average level of alcohol dependence in its neighbors (those vertices connected to it by graph edges). Each vertex has an associated resilience parameter. This parameter sets the exposure level that separates the dependent and healthy states; exposure exceeding the resilience value results in alcohol dependence. If the average level of alcoholism among its neighbors exceeds this threshold then the vertex increases its degree of alcoholism, otherwise it decreases. This model was studied in part because of its similarity to the logistic equation and because the dynamics of the network's behavior were relatively easy to predict from theory and numerical simulations were readily and quickly obtained.

2 A Primer on Networks

Much is known about random networks. Erdös and Rényi [9] began the study of random networks during the 1960s. They have shown that for a graph of N vertices where the probability that a pair of vertices will be linked by an edge (a connection between vertices) is $p \in [0, 1]$, the average number of edges in the graph is $N(N-1)p/2$, the average degree of a vertex is $z = (N-1)p$, and the probability that a vertex will have degree k follows the Poisson distribution $P(k) = e^{-z}z^k/k!$ when N is large. Random networks are also discussed in the books by Barabási [10] and Watts [3]. A small random network is shown in figure 6. The characteristics of random networks are that they have (1) a relatively small characteristic length L, which is the average number of links required to get from one node to another, and (2) a very small amount of clustering as measured by the average clustering coefficient G of the graph. A sample random graph with 90 vertices and 100 links is shown in figure 6.

A different graph used as a model for complete clustering is the so-called caveman graph, where there are isolated groups of vertices that are completely interlinked. The two parameters k and n describe these graphs; there are $k+1$ members in a cluster and there are n clusters. Said another way, everyone in the cave knows each other but no one knows anybody from another cave; the vertices are very clustered, and one cannot cross the graph from one cave to another at all. Figure

1 shows an example of such a graph.

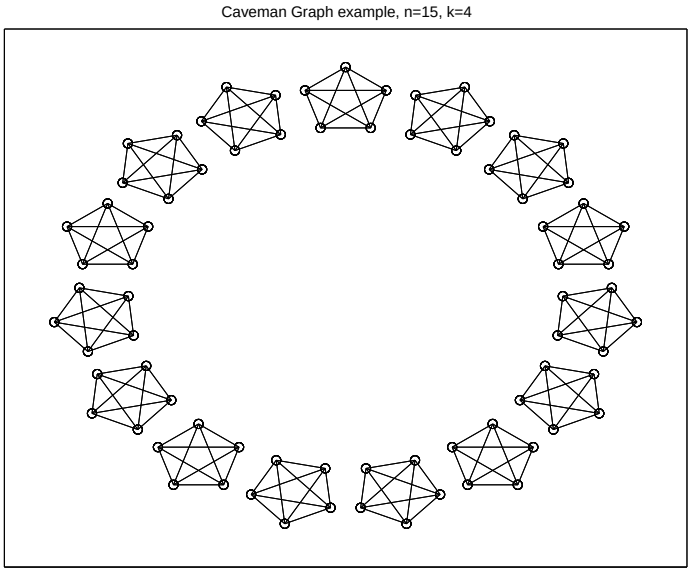

Figure 1: A caveman graph with $k = 4$ and $n = 15$; there are $k + 1$ members in each cluster ("cave") and n clusters.

A first step toward having clustering and a characteristic length comparable to the graph size is the connected caveman model. In this case, one link from each cave is replaced so that it now connects one cave to another; figure 2 shows an example of such a graph with $n = 15$ clusters and 5 vertices per cluster ($k = 4$). This graph has two distinct scales that indicate how many links are required to go from one vertex to another. Within a cave, the average length between nodes is nearly one; another average length scale is how many links it takes to go between vertices in different caves, and this is on the order of the number of caves. This graph is highly clustered.

One can use this connected caveman graph as a "substrate" to create different graphs that span a useful range from the highly ordered world of the connected caveman (which may be too ordered) to a practically random graph. This will produce a range of graphs from very clustered with a relatively large characteristic length to graphs with small clustering and small characteristic length. Between these extremes are graphs with relatively small characteristic length (like random graphs) but relatively large clustering; these graphs are called small world graphs. Such graphs appear to be appropriate models for some sociological networks [3]. An example with a small

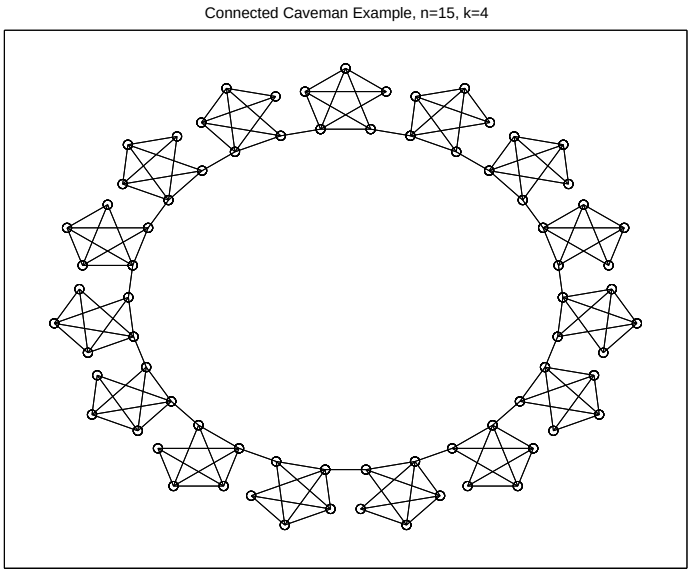

Figure 2: A caveman graph with $k = 4$ and $n = 15$; there are $k + 1$ members in each cluster ("cave") and n clusters.

amount of rewiring is shown in figure 3; more rewiring, to the extent that the graph appears to be random, is shown in figure 4. We note that Watts comments that networks that are constructed by heavily rewiring the connected caveman graph are not truly random [3], but they will serve the purpose of spanning the behavior from highly clustered (and ordered) to practically random, while covering a range of small world graphs.

We aim to use the known properties of these graphs in our modeling so that we need not study the properties of the network so much as the dynamics on it.

Barabási [10] and others cited below emphasized scale-free (or power-law) networks, e.g. the internet, but these will not be studied in this report.

3 Models for neighborhood dynamics on fixed networks

The following models for dynamics on a social network were inspired by Watts [11]. In that paper, the binary decisions are made about the status of an individual in the network based on the influence of the neighbors and on a threshold value of each individual. In that paper, the thresholds r were a probability density that is distributed randomly on $r \in [0, 1]$ (his results hold for very general

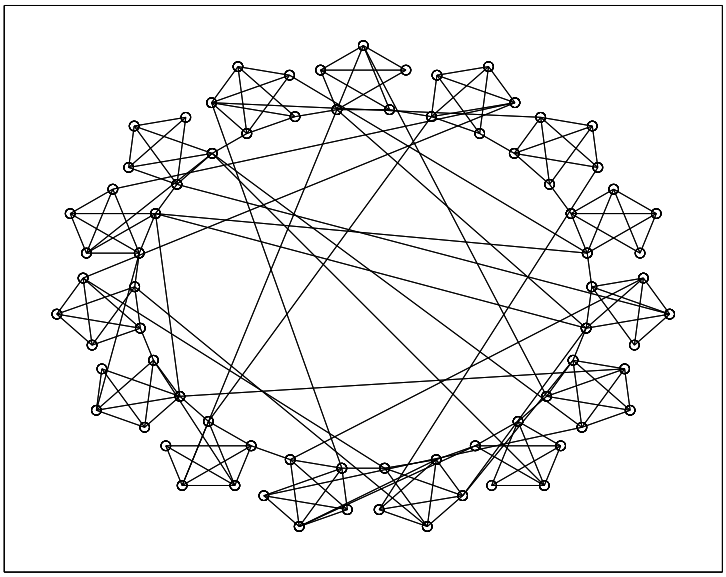

Figure 3: A rewired connected caveman graph with a small amount of rewiring; $2n$ links were rewired. Here $k = 4$ and $n = 15$; there are $k + 1$ members in each cluster ("cave") and n clusters in the original substrate.

distributions of the thresholds). The purpose of that paper was to discover conditions under which the behavior (say the unit state) would propagate from a small initial seed across a random network and the extent of the propagation. Conditions for global cascade across the network were found. This idea of a threshold value before the inception of alcohol problems occurs will be incorporated into our leading model of this type for simulation.

The class of models in this section is intended to describe the behavior of individuals in response to the behavior of other individuals in his or her neighborhood; we could therefore think of these as models for dynamics of individuals on fixed networks. The network is considered fixed because the relationships of the individuals are not altered in any case; such dynamic rewiring is not considered in this report.

3.1 Linear model

A set of people are considered to be the vertices of the graph designated by the list of values $\mathbf{v}^j$ at time level j, with $j = 0, ..., M$. An individual i is then denoted v_i^j, where $i = 1, ..., N$. $v_i^j \in [0, 1]$ is the probability that person i is alcohol dependent at time j. The structure of the population is

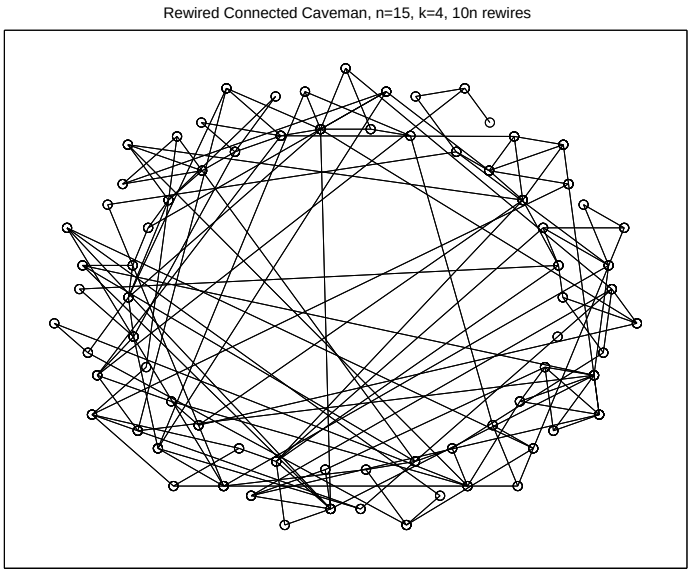

Figure 4: A rewired connected caveman graph with a large amount of rewiring; $10n$ links were rewired. Here $k = 4$ and $n = 15$; there are $k+1$ members in each cluster ("cave") and n clusters in the original substrate.

given by the symmetric matrix A with 0's on the diagonal (no self-connections) and 1's in various locations; we will call this matrix the "adjacency matrix." In a random graph, an algorithm of Chandler, *et al.* [13] is used to generate random locations for these unit values, thus creating a random graph. A rewired connected caveman graph was also used to describe the population [3]. Note that for a rewired connected caveman graph, the neighbors of a given vertex can be from both inside and outside the original cave; furthermore, with enough rewiring, the graph is practically random and distinguishing between neighbors that were originally inside or outside the cave is likely to be of little use.

The influence of the neighbors of a given person may be computed from the average of the neighbors n_i^j of individual i at time level j. The average of the neighbors at all locations may be easily computed in MATLAB by the matrix vector product $\mathbf{n}^j = BA\mathbf{v}^j$ where A is the $N \times N$ adjacency matrix and B is the $N \times N$ diagonal matrix whose elements are the reciprocals of the sum of the corresponding row or column of A (recall A is symmetric).

One can then examine the linear model

$$v_i^{j+1} = v_i^j + \lambda(n_i^j - v_i^j) \tag{1}$$

for updating the probability that person i has alcohol dependence at time j. Here $i = 1, ..., N$ and $j = 1, ..., M + 1$. The initial condition could be viewed as a distribution of locations for $\mathbf{v}^1 \in \{0, 1\}$ or as a distribution $\mathbf{v}^1 \in [0, 1]$ around the network. The rate constant λ controls how fast the simulation evolves; choosing a relatively large value allows the simulation to reach a steady state in less steps, thereby saving computational time.

The first term of the right-hand-side of (1) is the "old" value of v and the second term measures the difference between a vertex and the average of its neighbors. The difference between a vertex and its neighbors is then used to update v to obtain v at the new time level (the left hand side).

In matrix-vector form, the same model may be written as

$$\mathbf{v}^{j+1} = \mathbf{v}^j + \lambda(BA - I) * \mathbf{v}^j; \tag{2}$$

here I is the $N \times N$ identity matrix

We may think of this model as arising from a Euler method discretization of the linear ODE system

$$\frac{d\mathbf{v}}{dt} = c(BA - I)\mathbf{v}, \tag{3}$$

where c is a scalar constant.

In either case, simulation revealed that the final state approached is a uniform, nonzero value at all vertices on the network; the final state's value was not an average of the initial state or any other such obvious value that we could deduce. We abandoned this model in favor of models with richer behavior.

Since this model is driven solely by influence from a vertex's neighbors, we could describe these as the "pure neighborhood influence" models.

3.2 Quadratic model

The second model we considered is the quadratic map

$$v_i^{j+1} = v_i^j + \lambda v_i^j (n_i^j - v_i^j) \tag{4}$$

for the same ranges of the indices as described in the linear model. The neighbor influence term is now multiplied by v_i^j. In matrix-vector form, we have

$$\mathbf{v}^{j+1} = \mathbf{v}^j + \lambda \mathbf{v}^j .* [(BA - I) * \mathbf{v}^j]. \tag{5}$$

Here $.*$ means element-wise multiplication as used in MATLAB, for example.

Equilibria occur when $v_i^{j+1} = v_i^j$ when j is increasing. In that case, the last term on the right must be 0, and examining this situation shows that $\mathbf{v} = \mathbf{0}$ is an unstable equilibrium. Thus, any small perturbation of the zero vector causes v to grow in magnitude away from this state. $\mathbf{n} = \mathbf{v}$ is a stable equilibrium at a uniform nonzero value that is not easily computed from the initial conditions (the equilibrium value depends on the initial state). Since this is the only stable steady state, the ω-limit set of any orbit beginning from nonzero initial conditions is the nonzero steady state. The behavior of this model is also not rich enough for our purposes.

We may think of this model as arising from a Euler method discretization of the logistic ODE system

$$\frac{d\mathbf{v}}{dt} = c\mathbf{v} .* (BA - I)\mathbf{v}, \tag{6}$$

where c is a constant.

3.3 Cubic model

We settled on the following cubic model as being the most desirable of this set of models:

$$v_i^{j+1} = v_i^j + \lambda v_i^j (1 - v_i^j)(n_i^j - r_i). \tag{7}$$

Here r_i is the resiliency (biological or psychological) of the ith person against an alcohol problem. In this report, the resiliency is typically uniformly distributed on [0,1]. This model, while retaining much of the simplicity of the two previous models, possesses richer dynamics. In matrix-vector form, we have

$$\mathbf{v}^{j+1} = \mathbf{v}^j + \lambda \mathbf{v}^j .* (\mathbf{1} - \mathbf{v}^j) .* (BA\mathbf{v}^j - \mathbf{r}); \tag{8}$$

here $\mathbf{1}$ is a column vector with length N consisting of unit elements.

The behavior of this model is as follows. The stable equilibrium states for each vertex are 0 and 1; the state satisfying $\mathbf{n} = \mathbf{r}$ is an unstable steady state. Each vertex will be driven toward 0 if $n_i^j - r_i < 0$ and toward 1 if $n_i^j - r_i > 0$; that is, if the average of your neighbors' average behavior exceeds your threshold, you develop an alcohol problem; if your neighbors' average behavior stays below your alcohol dependence threshold, you remain non-dependent. This qualitative description is discussed more rigorously below.

We may think of this model as arising from a Euler method discretization of the logistic-like ODE system

$$\frac{d\mathbf{v}}{dt} = c\mathbf{v} .* (\mathbf{1} - \mathbf{v}) .* (BA\mathbf{v} - \mathbf{r}), \tag{9}$$

where c is a constant and r is the set of (constant) resiliency values.

3.4 Theory for cubic model

The cubic model presented in equation 7 has three equilibrium values per vertex, thus leading to rich, heterogeneous behavior of the network. As will be proved in the theorems below, for each vertex two of the equilibrium states are unstable and the third is stable. A network governed by the cubic model can also exhibit a tipping point in which a small perturbation produces a homogeneous state throughout the network [14].

Theorem 3.1 *Consider an adjacency matrix A, a vector of resiliencies $\mathbf{r}$, and a vector of vertex states $\mathbf{v}$ in which $v_i \in \{0, 1\}$. Suppose that $n_i^j \neq r_i$ for all $i = 1, \ldots, N$. The vertex state $\mathbf{v}$ is a stable equilibrium of the cubic model (7) if and only if $H(n_i^j - r_i) = v_i$ for all i, where H is the Heaviside function.*

Proof: Regardless of the value of $H(n_i^j - r_i)$, if $v_i^j = 0$ (or 1) then according to equation (7) $v_i^{j+1} = v_i^j$. Consequently $\mathbf{v}$ is an equilibrium state for the cubic model. Let

$$\overline{\epsilon} = \min_i \frac{|n_i^j - r_i|}{\deg(i)}$$

where $\deg(i)$ is the degree of vertex i. $\overline{\epsilon} > 0$ since $n_i^j - r_i \neq 0$ for all vertices i. $\overline{\epsilon}$ will represent the largest amount by which v_i^j can be perturbed. Now let $u_i^j = v_i^j + \epsilon_i$ be a perturbation of $\mathbf{v}$ where it is assumed that $0 \leq \epsilon_i < \overline{\epsilon}$ when $v_i^j = 0$ and $-\overline{\epsilon} < \epsilon_i \leq 0$ when $v_i^j = 1$. This ensures that the algebraic sign of $n_i^j - r_i$ did not change due to the perturbation. Then using equation (7) we have

$$u_i^{j+1} - u_i^j = \lambda u_i^j (1 - u_i^j)(n_i^j - r_i) \quad \begin{cases} > 0 & \text{if } n_i^j - r_i > 0 \\ < 0 & \text{if } n_i^j - r_i < 0 \end{cases}$$

Using an induction argument on j we see that for vertices i for which $n_i^j - r_i > 0$, $\{u_i^j\}$ forms a strictly increasing sequence which is bounded above by 1 and hence converges. If $\lim_{j\to\infty} u_i^j = w_i < 1$ then

$$w_i = w_i + \lambda w_i (1 - w_i)(n_i - r_i) > w_i$$

which is a contradiction. Likewise for vertices i for which $n_i^j - r_i < 0$, $\{u_i^j\}$ forms a strictly decreasing sequence which is bounded below by 0 and hence converges. If $\lim_{j\to\infty} u_i^j = w_i > 0$ then

$$w_i = w_i + \lambda w_i (1 - w_i)(n_i - r_i) < w_i$$

which is a contradiction. □

Theorem 3.2 *Consider an adjacency matrix A, a vector of resiliencies $\mathbf{r}$, and a vector of vertex states $\mathbf{v}$ in which $n_i = r_i$ and $0 < v_i < 1$ for all i. The vertex state $\mathbf{v}$ is an unstable equilibrium of the cubic model (7).*

Proof: Let k be the index of a vertex in the graph for which $v_k < 1$. Perturb v_k by $0 < \epsilon < 1 - v_k$, *i.e.* let $v_k^1 = v_k + \epsilon$ and leave all other vertices unchanged, *i.e.* $v_i^1 = v_i$ for all $i \neq k$. Then if l is

the index of a neighbor of vertex k, then $n_l^1 - r_l > 0$ which implies that $v_l^2 > v_l^1$. We can verify from equation (7) that $v_k^2 = v_k^1$. Thus $n_k^2 - r_k > 0$ which implies that $v_k^3 > v_k^2$. We can show by induction on j that $\{v_k^j\}$ forms a strictly increasing sequence for $j > 1$. Thus $\mathbf{v}$ is unstable.

If $v_i = 1$ for all i, select any index k and perturb v_k by $0 < \epsilon < 1$, *i.e.* let $v_k^1 = 1 - \epsilon$ and leave all other vertices unchanged, *i.e.* $v_i^1 = 1$ for all $i \neq k$. Then if l is the index of a neighbor of vertex k, then $n_l^1 - r_l < 0$ which implies that $v_l^2 < 1$. We can verify from equation (7) that $v_k^2 = v_k^1$. Thus $n_k^2 - r_k < 0$ which implies that $v_k^3 < v_k^2$. We can show by induction on j that $\{v_k^j\}$ forms a strictly decreasing sequence for $j > 1$. Thus $\mathbf{v}$ is unstable. □

Careful perturbation of the previously described unstable equilibrium can also lead to "tipping" behavior in which all the vertices become healthy together or become sick together.

Corollary 3.3 *Consider an adjacency matrix A, a vector of resiliencies $\mathbf{r}$, and a vector of vertex states $\mathbf{v}$ in which $n_i = r_i$ and $v_i \neq 0$ for all $i = 1, \ldots, N$. Then if there exists an index k for which $v_k < 1$ then any perturbation which increases v_k leads to an asymptotic network state of $\mathbf{v}^\infty = \mathbf{1}$.*

Proof: Let k be the index of a vertex in the graph for which $v_k < 1$. Perturb v_k by $0 < \epsilon < 1 - v_k$, *i.e.* let $v_k^1 = v_k + \epsilon$ and leave all other vertices unchanged, *i.e.* $v_i^1 = v_i$ for all $i \neq k$. Then if l is the index of a neighbor of vertex k, then $n_l^1 - r_l > 0$ which implies that $v_l^2 \geq v_l^1$. Equality holds only if $v_l^1 = 1$.

The previous argument can be repeated with k replaced by every index of a vertex whose value strictly increased. If the graph consists of a single connected component, then we see that for each index i, $\{v_i^j\}$ forms a strictly increasing sequence or $v_i^j = 1$. In either case $\lim_{j\to\infty} \mathbf{v}^j = \mathbf{1}$. □

Corollary 3.4 *Consider an adjacency matrix A, a vector of resiliencies $\mathbf{r}$, and a vector of vertex states $\mathbf{v}$ in which $n_i = r_i$ and $v_i \neq 1$ for all $i = 1, \ldots, N$. Then if there exists an index k for which $0 < v_k$ then any perturbation which decreases v_k leads to an asymptotic network state of $\mathbf{v}^\infty = \mathbf{0}$.*

Proof: Let k be the index of a vertex in the graph for which $0 < v_k$. Perturb v_k by $0 < \epsilon < v_k$, *i.e.* let $v_k^1 = v_k - \epsilon$ and leave all other vertices unchanged, *i.e.* $v_i^1 = v_i$ for all $i \neq k$. Then if l is the index of a neighbor of vertex k, then $n_l^1 - r_l < 0$ which implies that $v_l^2 \leq v_l^1$. Equality holds only if $v_l^1 = 0$.

The previous argument can be repeated with k replaced by every index of a vertex whose value strictly decreased. If the graph consists of a single connected component, then we see that for each index i, $\{v_i^j\}$ forms a strictly decreasing sequence or $v_i^j = 0$. In either case $\lim_{j\to\infty} \mathbf{v}^j = \mathbf{0}$. □

The previous theorem implies that at the unstable equilibrium, if one vertex increases, then $v_i^j \to 1$ as $j \to \infty$ for all i. Likewise if one vertex decreases, then $v_i^j \to 0$ as $j \to \infty$ for all i.

3.5 Simulations for cubic model

Unless otherwise noted, the resiliency distribution over the network is normal with mean $\mu = 1/2$ and standard deviation $\sigma = 1/6$. Also, $\lambda = 0.5$ is used for all simulations; this relatively large value allows us to find the final state relatively rapidly.

3.5.1 Typical realizations of population structure

The following figures give two example realizations of the the population structures used below. A rewired connected caveman network is shown in figure 5. A representative random graph used in the simulations is shown in figure 6.

3.5.2 Rewired Connected Caveman

In figure 7, the initial state is determined by sequentially filling in clusters with alcohol-dependent people. In particular, the initial vector is set to a small value of $\mathbf{v}^0 = 0.15$; then, beginning with a single vertex, each simulation is begun with one more value $v_i^0 = 0.9$ and the result for each initial condition is a single data point on the curve. The curve was computed with 9 members of each cluster and 10 clusters; here the network had 20 edges rewired randomly. The final state doesn't stray far from the initial state in this case. A step-like curve emerges because the spread of alcoholics doesn't escape the clusters easily at this level of rewiring; once a cluster is populated with nodes expressing a high degree of alcoholism, this high average alcoholism level spreads via the inter-cluster links to neighboring clusters.

We now turn to increasing number of alcohol-dependent people on a rewired connected caveman social network when the alcohol dependent people are added randomly around the network. Results

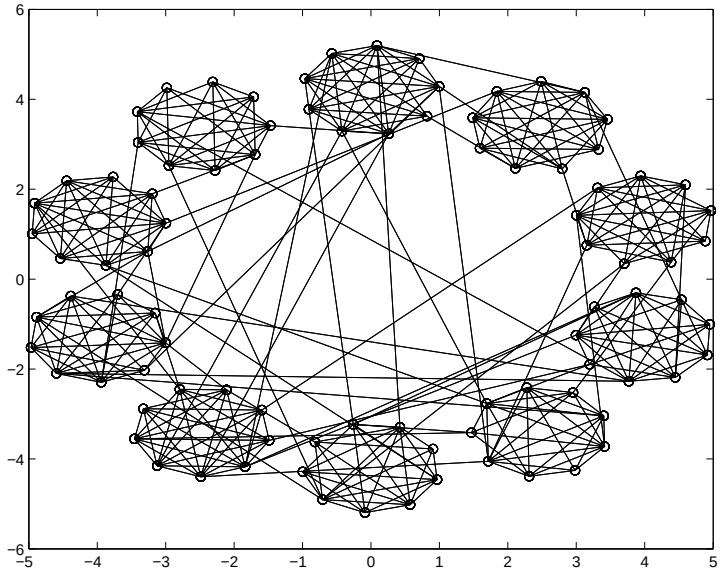

Figure 5: Example rewired connected caveman graph typically used in simulations below. $N = 90$ is the total number of people, there are $k + 1 = 9$ people per cluster and $n = 10$ clusters. 20 connections were rewired from the connected caveman graph.

for a substrate with 9 people per cluster, 10 clusters and 80 edges randomly rewired are shown in figure 9. In this case, there is a distinct phase transition in the number of dependents in the final state. There are essentially no alcoholics until $\rho_{start} \approx 1/5$ (20 initially dependent people out of 90); the fraction in the final state approaches 1 around $\rho_{start} \approx 0.8$ (70 initially-dependent people out of 90). ρ_{end} increases rapidly as $\rho_{start} \to 1$. If the initial state is changed so that the initial state has only values of 0 and 0.9, with all other parameters the same, then we obtain the results in Figure 9. In this case, the transition is shifted to the right, and the fully alcohol-dependent population only occurs if essentially all of the population is initially dependent (at 0.9). Thus changing the initial value of the healthy part of the population strongly affects the tipping point where all of the population becomes alcohol dependent.

In figure 10, the dependence of the final state with $\bar{r}$ is shown. Each value of $\bar{r}$ on the curve is from an average of 100 realizations of the network; the distribution of the resiliency is a truncated normal distribution with standard deviation of 1/6 with mean $\bar{r}$. The distribution is truncated in the sense that any values of resiliency below 0 or above 1 are converted to the nearest end point

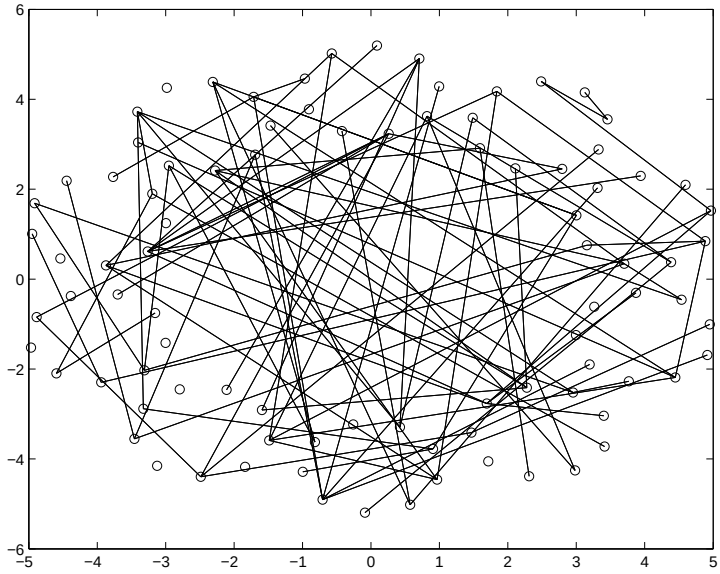

Figure 6: Typical random graph used in the calculations.

value. The initial condition is a uniform distribution $v_i^0 \in [0, 1]$. The transition from all alcoholic people (at very low average resiliency) to nearly all healthy people (at very high resiliency) occurs over a wider range of $\bar{r}$ than for random networks (next subsection). As the number of rewired edges increases, the width of the transition from all alcohol-dependent to a completely healthy population narrows, indicating the approach to effectively random-graph behavior with this trend.

3.5.3 Random Graph

Two cases are simulated on random graphs for comparison. First, we present results for an increasing number of alcohol-dependent people on a random social network. The initial condition in figure 12 is $v_i^0 = 0.15$ or 0.9, with the higher value distributed randomly around the network. In this case there is a transition in the number of alcohol-dependent people in the final state; this is illustrated in figure 11. The transition is narrower than the result for the rewired connected caveman case with 80 re-wirings. For the case with initial values of 0 and 0.9, the increase in alcohol dependence occurs at larger initial fraction of alcohol dependence in the population. This trend is similar to those from the rewired connected caveman network, though the random network exhibits a narrower transition of tipping from the completely healthy to the completely dependent

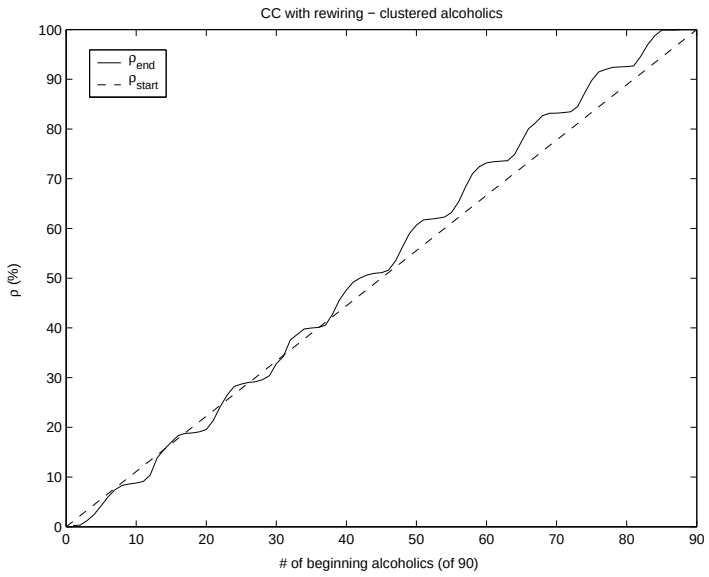

Figure 7: Initial condition: increase number of alcoholics by filling one cluster at at time. The number of stair steps equal the number of clusters. There is little change between the initial number of alcoholics ρ_{start} and final state ρ_{end}.

population.

In figure 13, the dependence of the final state with $\bar{r}$ is shown. Each value of $\bar{r}$ on the curve is from an average of 100 realizations of the network; the initial state is a uniform distribution on [0,1]. The transition from all alcohol-dependent people (very low average resiliency) to nearly all healthy people occurs over a significantly narrower range of $\bar{r}$ than for the rewired connected cavemen network with 50 re-wirings (figure 10).

3.6 Treatment model

Consider "treating" the highest 4% or 10% of v_i values in the population by resetting v_i^j to $r_i/2$ and fixing the value of this vertex i for 100 iterations (i.e., subsequent j values). This could also be viewed as detox or jail time; we do not distinguish here. In all cases, $k = 8$, $n = 10$ and so there are 90 people in this population. The seeds for both `rand` and `randn` are set to their respective initial values for each simulation; this was done for reproducibility in the results presented here. We vary the number of rewirings in the population as well as the percentage treated. The initial condition is a random distribution of $v_i \in [0, 1]$ and the resiliency is a truncated normal distribution about

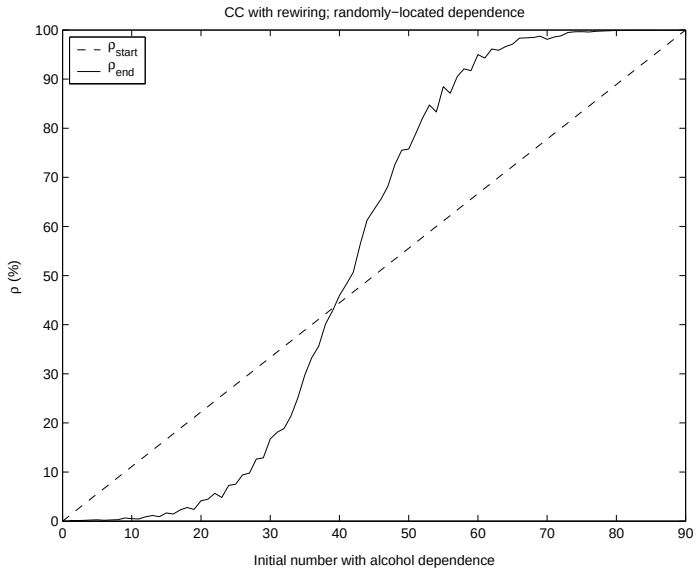

Figure 8: Initial condition: a number of alcohol-dependent people ($v_i^0 = 0.9$) are randomly placed among healthy people ($v_i^0 = 0.15$) on the network. In contrast to the filling one cluster at a time, there is a definite transition centered about 45 initially-dependent people from nearly no alcohol-dependent people in the final state ρ_{end} near 20 initially-dependent people to nearly all alcohol-dependent people when the initial number of alcohol dependents is near 70.

a mean of 0.5 with standard deviation of 1/6. Results for treating the worst 4% of the population are shown in figures 14 and 15, for 50 and 80 rewirings, respectively. From these two figures we see that increasing the amount of rewiring drives ρ, the average number of alcohol dependent people, to a significantly lower end state for this simulation. The case with less rewiring hovers around near a single value, but seems to fluctuate around it. Results from treating the worst 10% of the population for 50 and 80 rewirings, respectively, are shown in figures 16 and 17. We see that increasing the percentage of treated people drives the evolution to a healthy constant state. Increasing the rewiring as well causes the evolution to the end state to occur in fewer iterations. We conclude that the final states in the simulations are quite sensitive to the percentage of alcoholics treated.

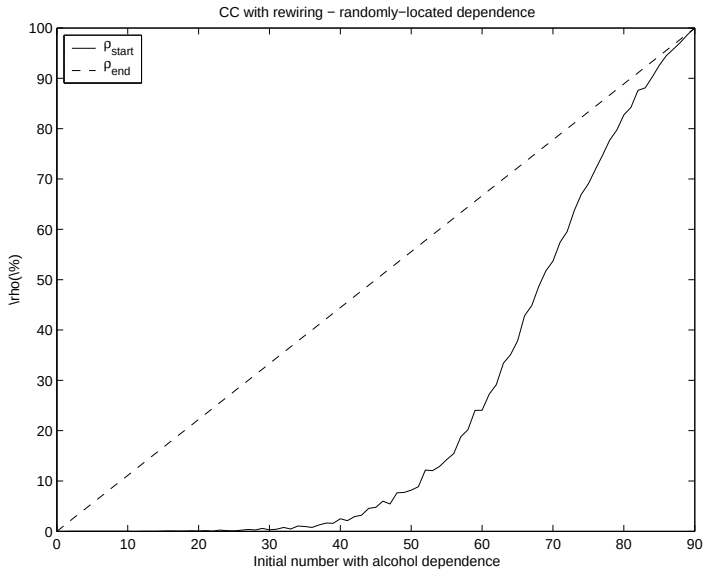

Figure 9: Initial condition: a number of alcohol-dependent people ($v_i^0 = 0.9$) are randomly placed among healthy people ($v_i^0 = 0$) on the network. In contrast to the filling one cluster at a time, there is a definite phase transition at about $\rho_{start} = 0.5$ from nearly no alcoholics in the final state ρ_{end} to a significant number. The increase in the number of alcoholics in the final state increases dramatically as $\rho_{start} \to 1$.

3.7 Discussion

The discrete cubic model coupled with a clustered graph provides a fertile testbed in which to simulate the effects of various policies for affecting the level of alcoholism within a subset of a population. We found that small world social networks have more gradual tipping behavior with respect to the parameters than comparable random social networks. Also, a model of treatment shows complex behavior including oscillations in the end state and reduction of dependence to a very low endemic state. The network substrate of the model provides a computationally and mathematically convenient framework within which the effects of treatment policies for alcoholism can be tested and predicted.

The dynamics of the cubic model are simple enough yet varied enough to study the effects of some social influences on a person's drinking behavior within the context of a larger social group.

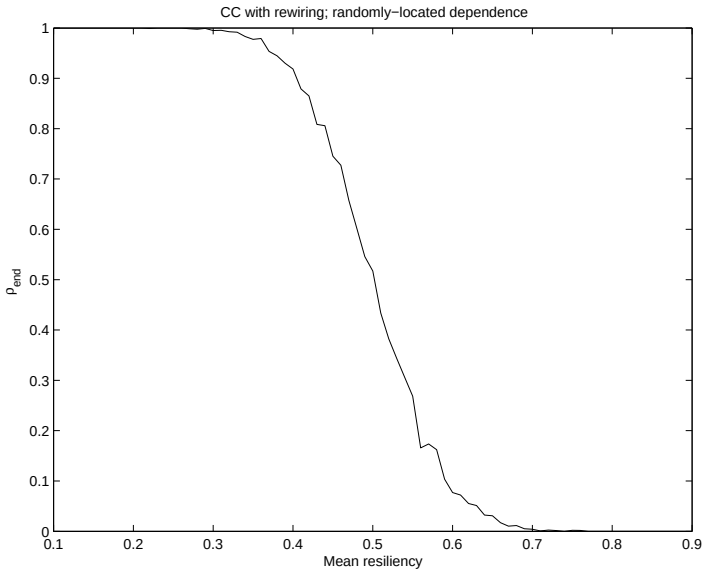

Figure 10: The final state ρ_{end} as a function of the mean resiliency of the population $\bar{r}$ on a rewired connected caveman network; here the standard deviation of the truncated normal distribution is $\sigma = 1/6$.

4 Zip Code Model

An alternative mathematical model to the one described in section 3 was proposed and briefly explored. The main goal of this alternative was to take into account both long and short term effects of surrounding population densities (given by zip codes) on the level of alcoholism in a specific zip code. By specifying the initial conditions and assigning the appropriate weights to certain social influences in this new model, projections can be made of the total number of alcoholics in the model as well as the density of alcoholics in any given zip code.

4.1 Formulation

This model of alcoholism is, like the previous one, of the category of discrete, difference equations. The difference in the proportion of alcoholics in the population of a cluster (thought of as a zip code) between time t and time $t+\Delta t$ is increased by the sum of the long term influence of social activities, the short term influence of peers of alcoholics, and the influence of alcoholism in other clusters, and decreased by the recovery rate of alcoholics in the cluster. Thus the local level of alcoholism

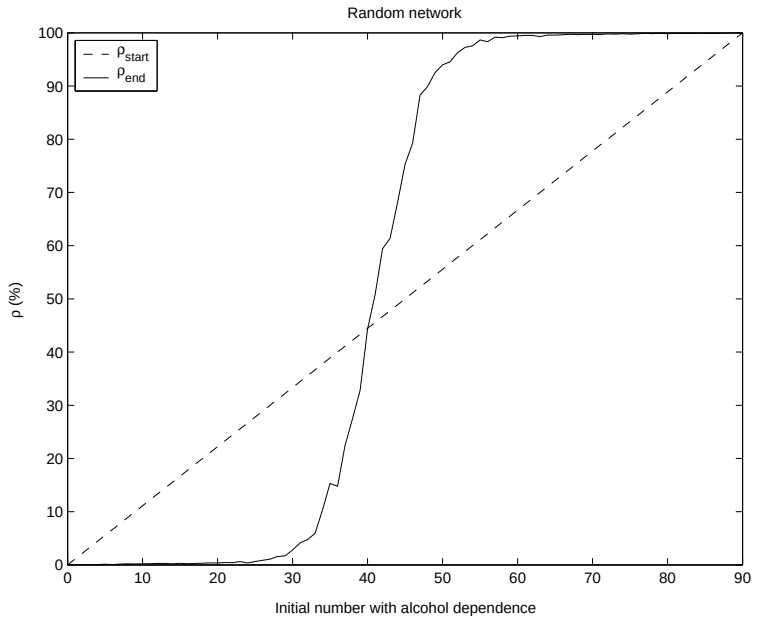

Figure 11: In contrast to the rewired connected caveman case, where the initial values are 0.15 or 0.9, there is a narrower transition from the case when the final state has $\rho_{end} \approx 0$ (the healthy population) and the final state $\rho_{end} \approx 1$ (the fully alcohol-dependent population).

is affected by society-wide influences, such as cultures' attitudes toward drinking alcohol, local influences, such as the level of alcoholism in geographically nearby regions, and person-to-person influences, such as peer pressure among acquaintances.

Before presenting the model the following notation should be understood.

Z_k**:** the k^{th} zip code

$P^k(t)$**:** the population of the k^{th} zip code at time t

$i^k(t)$**:** the proportion of alcoholics in the k^{th} zip code at time t.

$i^k_m(t)$**:** the proportion of alcoholics of age m in the k^{th} zip code at time t.

$s^k_m(t)$**:** the proportion of non-alcoholics of age m in the k^{th} zip code at time t. This quantity may be defined as

$$s^k_m(t) = 1 - i^k_m(t).$$

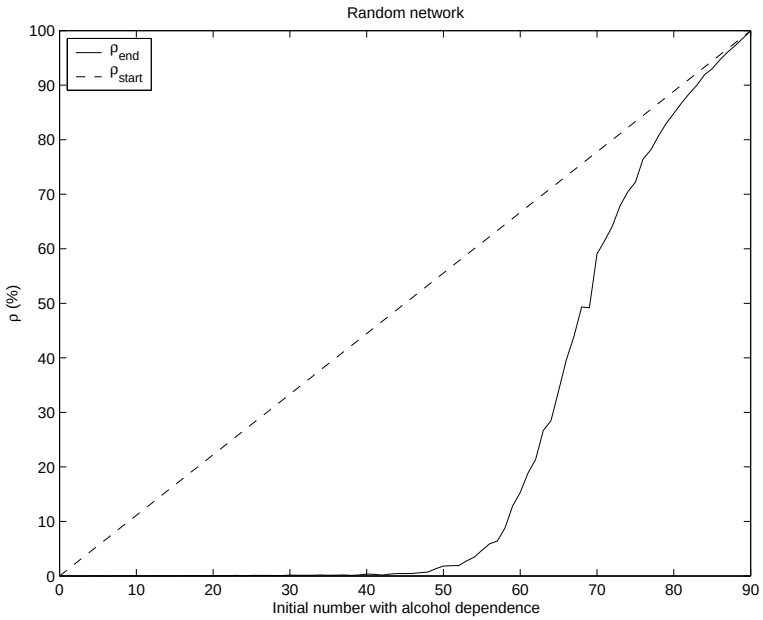

Figure 12: In contrast to the rewired connected caveman case, with initial values of 0 and 0.9, the transition is shifted to the right compared to the other random network case.

$y^k(t)$**:** the population density of the k^{th} zip code at time t.

$\rho^{k,l}(t)$**:** the proportion of population in the zip codes adjacent to the l^{th} zip code that lives in the k^{th} zip code at time t. This quantity is defined as

$$\rho^{k,l}(t) = \frac{P^k(t)}{\sum_{n \in adj(Z_l)} P^n(t)}.$$

The reader should note that the k^{th} zip code is a member of the set of zip codes adjacent to itself.

$\alpha_{n,m}$**:** a matrix of real numbers representing the degree to which an age group within a cluster contributes to the overall cultural attitude toward alcoholism.

$\beta_{n,m}(y^k(t))$**:** a matrix of real-valued functions representing the degree to which an age group within a cluster influences drinking behavior. The elements of this matrix are functions of the population density of the k^{th} cluster at time t.

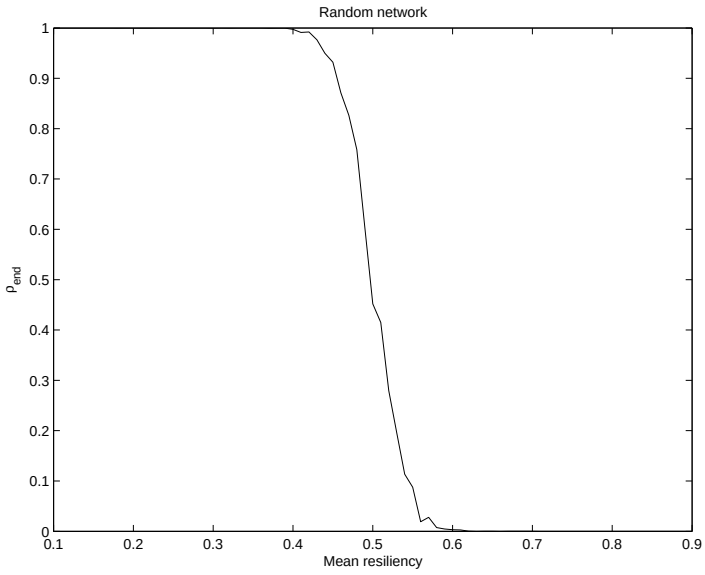

Figure 13: The final state ρ_{end} as a function of the mean resiliency of the population $\bar{r}$ on a random graph; here the standard deviation of the truncated normal distribution is $\sigma = 1/6$. Tipping from all dependent to all healthy occurs over a narrower range of $\bar{r}$ than for the rewired connected caveman graph of figure 10

$\mu(\rho^{k,l}(t))$**:** a matrix of real-valued functions representing the inter-cluster influence of each age group on alcoholism.

η_m^k**:** recovery rate for alcoholics of age m in the k^{th} zip code.

Using the quantities described above the change in the proportion of alcoholics of age m in the k^{th} zip code between times t and $t + \Delta t$ is modeled as

$$i^k_{m+\Delta t}(t+\Delta t) - i^k_m(t) = s^k_m(t)\left(\sum_n \left[\alpha_{n,m} + \beta_{n,m}(y^k(t))\right] i^k_n(t) + \sum_{n\in adj(Z_k)} \mu(\rho^{k,n}) i^n(t)\right) - \eta^k_m i^k_m(t).$$

The left hand side is the change in the proportion of alcoholics from t to $t + \Delta t$. On the right-hand side, the first term is the long term influence from the alcoholics within the cluster. The second term is the short term influence from the alcoholics within the cluster. The third term is the influence from the alcoholics from neighboring clusters. The fourth term is the proportion of recovered alcoholics.

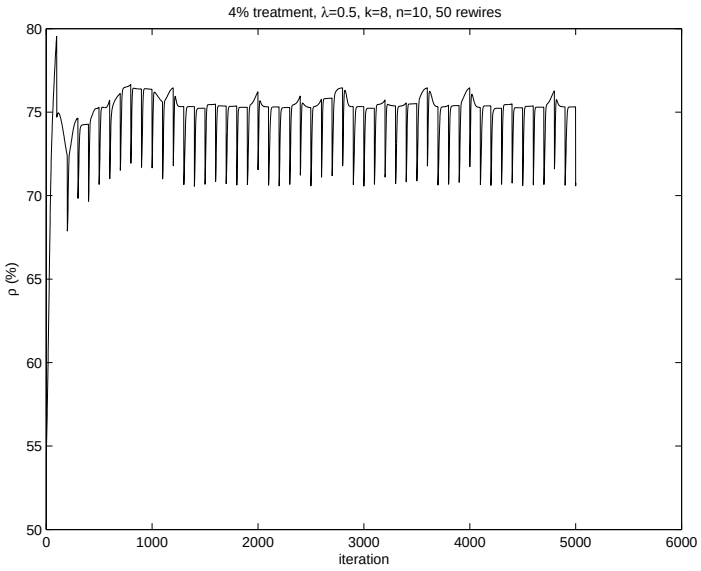

Figure 14: Simulation treating the top 4% of $\mathbf{v}$; the parameters are $\lambda = 0.5$, $k = 8$, $n = 10$, and there were 50 rewirings in this 90 person graph. The simulation doesn't seem to settle down to a constant value of ρ_{end} in this case, though the simulation does seem to be attracted to a neighborhood of a narrow range of ρ values.

4.2 Simulations

Using the governing difference equation, we can build a system of difference equations which are coupled by zip code adjacency and age group. Measured demographic data is used to initialize the coefficient matrices $[\alpha]$, $[\beta]$, and $[\mu]$. In general, $\beta_{n,m}$ should be much larger than $\alpha_{n,m}$, since long term influence changes the drinking habit slowly. Forward iterates of the model provide forecasts of the prevalence of alcoholism within the population. To verify that the model is computationally feasible, a simulation of the forward iterates of the difference equation shown above was implemented in MATLAB. The initial percentage of alcoholics in ten zipcode regions is shown in the bar chart in Figure 18. These values are obtained from the survey data collected in [1]. The model-predicted percentage of alcoholics in the same ten zipcodes after 50 iterations of the model is shown in Figure 19.

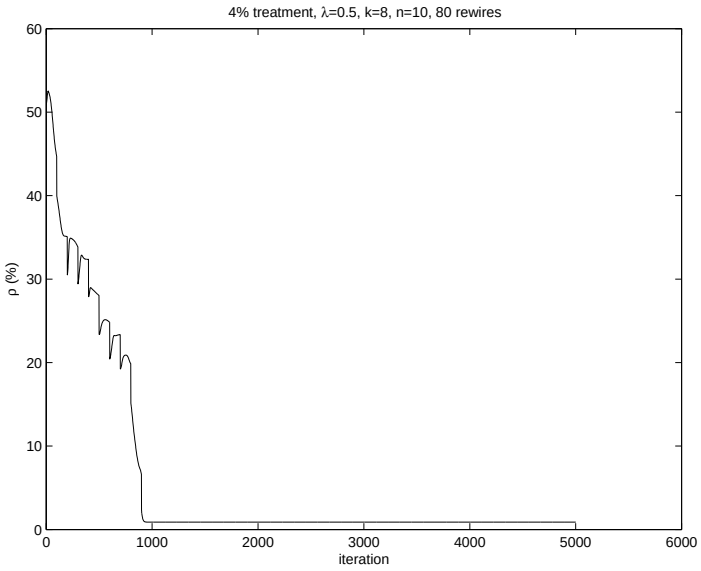

Figure 15: Simulation treating the top 4% of $\mathbf{v}$; the parameters are $\lambda = 0.5$, $k = 8$, $n = 10$, and there were 80 rewirings in this 90 person graph. The simulation is attracted to a very healthy steady state in this case.

4.3 Discussion

This model separates the influence on drinking behavior into three categories: long term influences, short term influences, and inter-cluster influences. The prevalence of alcoholism within ages groups of the population is a feature of the model. The extra detail provided by this model comes at the expense of requiring more demographic data to estimate model parameters.

5 Conclusion

Sensitivity to the population structure and a treatment regimen were established. This can be take as proof of concept.

Further work needed in discovering dependence of the the results on network size and properties; for example, more detailed understanding of the behavior as random rewiring is increased as well as dependence on initial conditions and the resiliency parameter. Investigation of other treatment modes, e.g., 'rewiring' the relationships of a sample of the alcoholics (AA-style treatment), are also needed. Finally, more detailed study of the zip-code-level model could be fruitful as well.

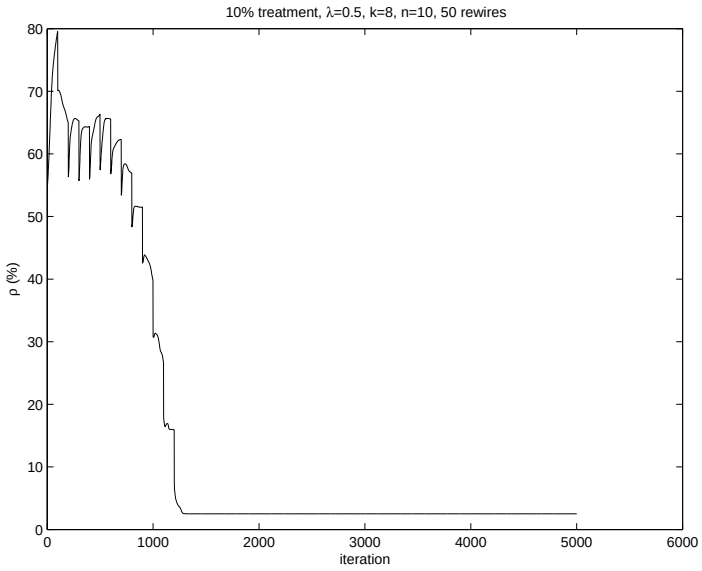

Figure 16: Simulation treating the top 10% of $\mathbf{v}$; the parameters are $\lambda = 0.5$, $k = 8$, $n = 10$, and there were 50 rewirings in this 90 person graph. "Treating" a sufficiently high percentage (in this simulation 10%) of the population seems to dramatically affect the percentage of alcohol-dependent people at late times in the simulation; the final state is a very healthy (a low ρ value).

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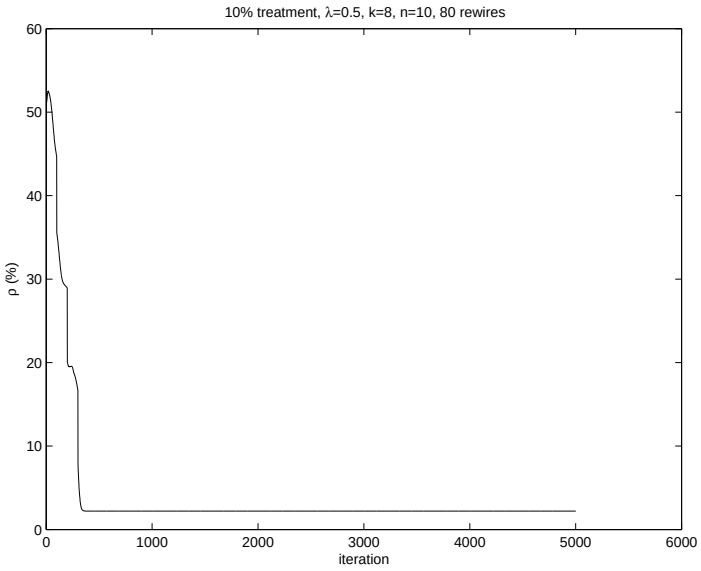

Figure 17: Simulation treating the top 4% of $\mathbf{v}$; the parameters are $\lambda = 0.5$, $k = 8$, $n = 10$, and there were 80 rewirings in this 90 person graph. The simulation is attracted to a very healthy steady state in this case faster than for 50 rewirings.

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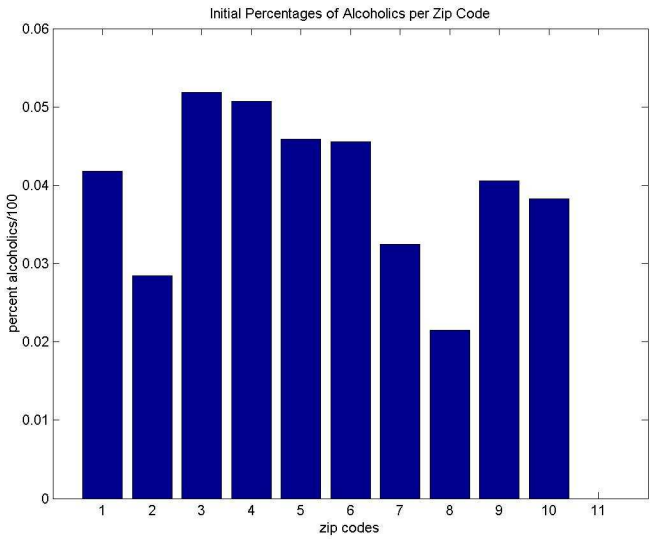

Figure 18: The initial percentages of alcoholics among the population of ten zipcodes in Delaware. These data are summarized from that contained in [1]. These data were used as the initial conditions for the difference equation model.

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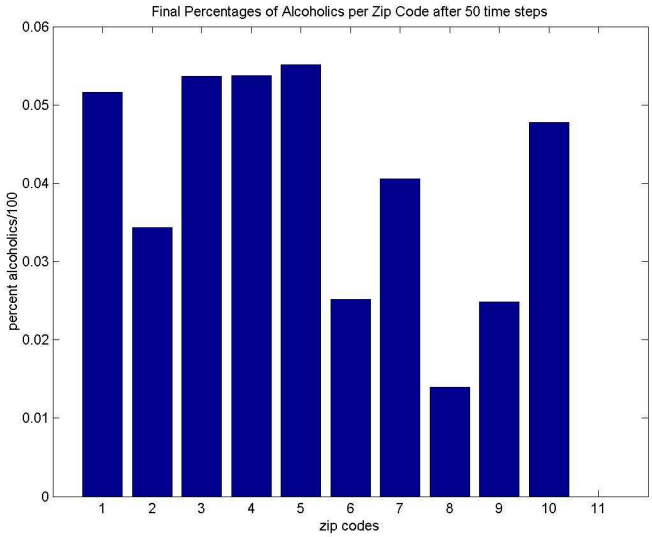

Figure 19: The predicted percentage of alcoholics in ten zipcodes in Delaware after 50 iterations of the model.