A Stochastic Birth-Death Model of Information Propagation Within Human Networks

Prasidh Chhabria(✉) and Winnie Lu

Harvard University, Cambridge, MA 02138, USA
chhabria@college.harvard.edu

Abstract. The fixation probability of a mutation in a population network is a widely-studied phenomenon in evolutionary dynamics. This mutation model following a Moran process finds a compelling application in modeling information propagation through human networks. Here we present a stochastic model for a two-state human population in which each of N individual nodes subscribes to one of two contrasting messages, or pieces of information. We use a mutation model to describe the spread of one of the two messages labeled the mutant, regulated by stochastic parameters such as talkativity and belief probability for an arbitrary fitness r of the mutant message. The fixation of mutant information is analyzed for an unstructured well-mixed population and simulated on a Barabási-Albert graph to mirror a human social network of $N = 100$ individuals. Chiefly, we introduce the possibility of a single node speaking to multiple information recipients or listeners, each independent of one another, per a binomial distribution. We find that while in mixed populations, the fixation probability of the mutant message is strongly correlated with the talkativity (sample correlation $\rho = 0.96$) and belief probability ($\rho = -0.74$) of the initial mutant, these correlations with respect to talkativity ($\rho = 0.61$) and belief probability ($\rho = -0.49$) are weaker on BA graph simulations. This indicates the likely effect of added stochastic noise associated with the inherent construction of graphs and human networks.

Keywords: Evolutionary dynamics · Human networks · Birth-death process

1 Introduction

The spread of information within human networks remains a particularly complex phenomenon, of particular interest in a time when information channels are varied and numerous [2]. Today, information propagates through more media than ever before; as a result, modeling the spread of information is difficult

Supported by Harvard University.

V. V. Krzhizhanovskaya et al. (Eds.): ICCS 2020, LNCS 12141, pp. 176–188, 2020.
https://doi.org/10.1007/978-3-030-50426-7_14

and must account for complexities arising from heterogeneity in communication across different channels. The phenomenon of information propagation on a network can be seen at play in politics, public health information, and social networks, among other common scenarios. Thus, understanding the dynamics of its spread is crucial to developing systems that encourage the spread of beneficial, factual information while deterring the spread of misleading or malicious information. The evolutionary dynamics of two competing, and often contrasting, pieces of information can be meaningfully characterized in both deterministic and stochastic models of human social networks.

In this paper, we model the spread of a mutant piece of information throughout an unstructured, well-mixed population of size N. We consider the case of one-to-one communication in which a speaker is able to speak to only one individual at a time. We then consider the case of one-to-many communication in which a speaker is able to simultaneously speak to multiple receivers. We then replicate this analysis by simulating on a Barabási-Albert graph, as an approximation of a classical human network.

1.1 Observed Information Propagation Models

Several mathematical models have been constructed to explain information spread. The majority of established studies have used deterministic models [4] such as the classic Spreader-Ignorant (SI) [5] and Spreader-Ignorant-Stifler (SIS) [6] rumor models. A *spreader* is an infected, or mutated, node that can spread the rumor to its neighbors. An *ignorant* is a susceptible node, which has not been exposed to the rumor. Finally, *stiflers* are nodes which have been exposed to the rumor but do not spread it. The present study attempts to establish a continuous spectrum across these three classes of nodes, by introducing probabilistic parameters such as talkativity and belief probability, to quantify the likelihood of each node believing and or sharing a piece of information. This allows for greater flexibility in the model rather than classifying each node into one of three categories. Several existing studies using deterministic models encounter problems while fitting suitable parameter values to a system of differential equations. These are unable to account for stochastic noise or probabilistic barriers. Since the spread of information depends on probabilities at each stage, the present study presents it as a stochastic process. We use a mutation model following a birth-death process to illustrate the transmission of information through a population, in a manner similar to a mutation albeit with altered parameters.

1.2 The Moran Birth-Death Process

The Moran process is a birth-death process [7] used to study the fixation of a mutant allele in an other wild-type population of size N. There are two absorbing states, at $i = 0$: where there are no mutants in the population, and $i = N$: where the mutant has fixated, i.e. spread to every node in the population [1]. In each time step of the process, there is always a single birth and a single death, so the

number of mutants in the population can change by at most one. The present study will extend this to a one-to-many process enabling group communication.

1.3 Barabási-Albert Graph [8]

The Barabási-Albert network is a widely used graph architecture for reproducing the structure of a real human social network [9]. It is a stochastic scale-free network generated by a distribution specified by the number of nodes and the average number of neighbors for each node. The present study relies on the BA graph, following its widespread use in literature for understanding the dynamic nature of human activities in social networks [10] (Fig. 1).

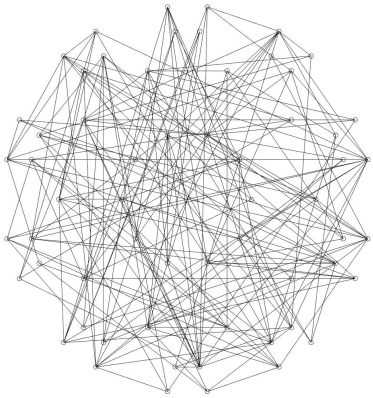


Fig. 1. A simple Barabási-Albert Graph computer-generated by a population of $N = 60$ nodes and an average number of $k = 10$ neighbors for each node.

2 Results

We outline a model for both one-to-one and one-to-many communication, beginning in an unstructured, well-mixed population. In both cases, we assume that the population is comprised of individuals (also known as 'nodes'). Within this population, we consider two competing and often contrasting pieces of information: the 'mutated' message and the 'non-mutated' message. The former is the variant of information whose propagation and fixation are of interest to our study. Following a mutation model, it begins as a rare event in the population and can either fixate, die out, or coexist with the non-mutated message. We consider only two pieces of information; each node believes one or the other, but not both. We say that the "mutant" confers a fitness of r to all believers (mutant nodes); $r < 1$ if knowledge and belief of the mutated message make an individual less likely to share the information, while $r > 1$ would suggest a heightened need to spread the mutant message.

A node that has been exposed to the mutated message and believes it is a mutant. A non-mutant is a node that has either not been exposed to or does not believe the mutated message. Past exposure to the mutant message is independent of a node's future belief probability if/when it is re-exposed to the message. A speaker is a node who communicates a piece of information, regardless of whether or not it is mutated. The speaker acts as the reproducing individual in a birth-death process. A receiver is an individual who listens to a message, representing the 'death' node, regardless of whether it is mutated or not. A node at position k has a talkativity τ_k where $0 \leq \tau_k \leq 1$. A higher τ_k here indicates that the node is more likely to be a speaker and spread the message to which it subscribes. A node at position k has belief probability β_k where $0 \leq \beta_k \leq 1$. A larger β_k here indicates that the node is more likely to believe the information it receives from a speaker.

In a well-mixed population of N individuals with i mutants, index positions 1 through i are mutants and index positions $i+1$ through N are non-mutants. When a node mutates, they are re-indexed into the i^{th} position. We disregard order within the population of mutants and within the population of non-mutants, as only the number of mutants and non-mutants is pertinent to the model.

2.1 One-to-One Communication Model

We begin modeling an unstructured, well-mixed population in which one speaker node communicates to only one recipient node at a time. The population begins with one mutant (allowing for fixation to be studied) but can have any number $0 \leq i \leq N$ of mutants at any given stage.

Population Shifts from i to $i-1$ Mutants: To facilitate this state change, a non-mutant must speak to a mutant who receives and believes the non-mutated message. The probability of this state change (see **Model Equations and Analytic Results** for derivation) is:

$$p_{i,i-1} = \frac{\sum_{k=i+1}^{N} \tau_k}{r\sum_{k=1}^{i} \tau_k + \sum_{k=i+1}^{N} \tau_k} \frac{\sum_{k=1}^{i} \beta_k}{N} \tag{1}$$

Population Shifts from i to $i+1$ Mutants: In order for this state change to occur, a mutant must speak to a non-mutant who listens and believes the mutated information. The probability of this transition is (see **Model Equations and Analytic Results** for derivation):

$$p_{i,i+1} = \frac{r\sum_{k=1}^{i} \tau_k}{r\sum_{k=1}^{i} \tau_k + \sum_{k=i+1}^{N} \tau_k} \frac{\sum_{k=i+1}^{N} \beta_k}{N} \tag{2}$$

2.2 One-to-Many Communication Model

The following explains the transition in population states in an unstructured, well-mixed population, in which one speaker node communicates to more than

one recipient node simultaneously. Similar to the one-to-one model, the population begins with 1 mutant for the sake of this paper, but the population can have $0 \leq i \leq N$ mutants at any given stage.

The probability $p_{m,n}$ that a population goes from m mutants to n mutants where $m, n \in \mathbb{R}$ and $0 \leq m, n \leq N$ is given by an $(N+1)$ by $(N+1)$ transition matrix P. Let P consist of elements $p_{m,n}$:

$$P_{(N+1\times N+1)} = \begin{bmatrix} 1 & 0 & 0 & 0 & \dots & 0 \\ p_{1,0} & p_{1,1} & p_{1,2} & p_{1,3} & \dots & p_{1,N} \\ 0 & \dots & p_{2,2} & \dots & \dots & \vdots \\ \vdots & \dots & \ddots & \ddots & \ddots & \vdots \\ \vdots & \vdots & \vdots & \ddots & \ddots & \vdots \\ p_{N-1,0} & \dots & \dots & \dots & \dots & p_{N-1,N} \\ 0 & 0 & 0 & \dots & \dots & 1 \end{bmatrix} \tag{3}$$

The transition matrix displays two clear absorbing states in its first and last rows. The probabilities within the transition matrix P are given by the ones below.

Population Shifts from i to $i+j$ ($j \geq 0$) Mutants: In the case in which the number of mutants in the population is increasing by more than one in one time step, the probability of a population of N individuals going from a state of i mutants to $i+j$ mutants is:

$$p_{i,i+j} = \frac{r \sum_{k=1}^{i} \tau_k}{r \sum_{k=1}^{i} \tau_k + \sum_{i+1}^{N} \tau_k} \binom{N}{j} \left(\frac{p \sum_{k=i+1}^{N} \beta_k}{N} \right)^j \left(\frac{N - p \sum_{k=i+1}^{N} \beta_k}{N} \right)^{N-j} \tag{4}$$

where p is the probability of an individual being chosen to speak. This is constant for all N individuals in the population but is appropriately weighted by talkativity τ.

Population Shifts from i to $i-j$ ($j \geq 0$) Mutants: In the case in which the number of mutants in the population is decreasing by more than one in one time step, the probability of a population of N individuals going from i mutants to ij mutants, where $j \geq 0$, is:

$$p_{i,i-j} = \frac{\sum_{k=i+1}^{N} \tau_k}{r \sum_{k=1}^{i} \tau_k + \sum_{i+1}^{N} \tau_k} \binom{N}{j} \left(\frac{p \sum_{k=1}^{i} \beta_k}{N} \right)^j \left(\frac{N - p \sum_{k=1}^{i} \beta_k}{N} \right)^{N-j} \tag{5}$$

Population State Does Not Shift: We consider the final case in which the number of mutants remains unchanged. The initial and final state of the

population is i mutants. The general form of $p_{i,i}$ is given by

$$p_{i,i} = 1 - \sum_{j=1}^{N-i} p_{i,i+j} - \sum_{j=1}^{i} p_{i,i-j} \tag{6}$$

A few key elements of the transition matrix P (3) include:

$$p_{1,0} = \frac{\sum_{k=2}^{N} \tau_k}{r\tau_1 + \sum_{k=2}^{N} \tau_k} (p\beta_1)\left(\frac{N - p\beta_1}{N}\right)^{N-1} \tag{7}$$

$$p_{1,1} = 1 - \sum_{j=1}^{N-1} p_{1,1+j} - p_{1,0} \tag{8}$$

$$p_{1,N} = \frac{r\tau_1}{r\tau_1 + \sum_{k=2}^{N} \tau_k} \left(\frac{p\sum_{k=2}^{N} \beta_k}{N}\right)^{N-1} \left(N - p\sum_{k=2}^{N} \beta_k\right) \tag{9}$$

$$p_{N-1,N} = \frac{r\sum_{k=1}^{N-1} \tau_k}{r\sum_{k=1}^{N-1} \tau_k + \tau_N} (p\beta_N) \left(\frac{N - p\beta_N}{N}\right)^{N-1} \tag{10}$$

P is a transition matrix for an absorbing Markov chain and can be written in canonical form where the transient states precede the absorbing states [12]. We rearrange the horizontal and vertical indices to $\{N-1, 1, ..., N-2, 0, N\}$. The canonical form of P is

$$S = \left(\begin{array}{c|c} Q & R \\ \hline 0 & I \end{array}\right)$$

$$S_{(N+1)\times(N+1)} = \begin{bmatrix} p_{N-1,N-1} & p_{N-1,1} & \cdots & \cdots & \cdots & \cdots & p_{N-1,0} & p_{N-1,N} \\ p_{1,N-1} & p_{1,1} & p_{1,2} & p_{1,3} & \cdots & p_{1,N-2} & p_{1,0} & p_{1,N} \\ \vdots & \ddots & \ddots & \ddots & \ddots & \ddots & \ddots & \vdots \\ \vdots & \ddots & \ddots & \ddots & \ddots & \ddots & \ddots & \vdots \\ p_{N-2,N-1} & \cdots & \cdots & \cdots & \cdots & p_{N-2,N-2} & p_{N-2,0} & p_{N-2,N} \\ 0 & 0 & \cdots & \cdots & \cdots & 0 & 1 & 0 \\ 0 & 0 & \cdots & \cdots & \cdots & 0 & 0 & 1 \end{bmatrix}$$

Then, the matrices Q and R are:

$$Q_{(N-1)\times(N-1)} = \begin{bmatrix} p_{N-1,N-1} & p_{N-1,1} & \cdots & \cdots & \cdots & \cdots \\ p_{1,N-1} & p_{1,1} & p_{1,2} & p_{1,3} & \cdots & p_{1,N-2} \\ \vdots & \ddots & \ddots & \ddots & \ddots & \vdots \\ \vdots & \ddots & \ddots & \ddots & \ddots & \vdots \\ p_{N-2,N-1} & \cdots & \cdots & \cdots & \cdots & p_{N-2,N-2} \end{bmatrix}$$

$$R_{(N-1)\times 2} = \begin{bmatrix} p_{N-1,0} & p_{N-1,N} \\ p_{1,0} & p_{1,N} \\ \ddots & \vdots \\ p_{N-2,0} & p_{N-2,N} \end{bmatrix}$$

To obtain the fundamental matrix $M_{(N-1)\times(N-1)}$, we must invert the $I_{(N-1)} - Q_{(N-1)\times(N-1)}$ matrix:

$$I_{(N-1)} - Q_{(N-1)\times(N-1)} = \begin{bmatrix} 1-p_{N-1,N-1} & -p_{N-1,1} & \cdots & \cdots & \cdots & \cdots \\ -p_{1,N-1} & 1-p_{1,1} & -p_{1,2} & -p_{1,3} & \cdots & -p_{1,N-2} \\ \vdots & \ddots & \ddots & \ddots & \ddots & \vdots \\ \vdots & \ddots & \ddots & \ddots & \ddots & \vdots \\ -p_{N-2,N-1} & \cdots & \cdots & \cdots & \cdots & 1-p_{N-2,N-2} \end{bmatrix}$$

$$M_{(N-1)\times(N-1)} = [I_{(N-1)} - Q_{(N-1)\times(N-1)}]^{-1}$$

$$= \begin{bmatrix} 1-p_{N-1,N-1} & -p_{N-1,1} & \cdots & \cdots & \cdots & \cdots \\ -p_{1,N-1} & 1-p_{1,1} & -p_{1,2} & -p_{1,3} & \cdots & -p_{1,N-2} \\ \vdots & \ddots & \ddots & \ddots & \ddots & \vdots \\ \vdots & \ddots & \ddots & \ddots & \ddots & \vdots \\ -p_{N-2,N-1} & \cdots & \cdots & \cdots & \cdots & 1-p_{N-2,N-2} \end{bmatrix}^{-1}$$

B is the absorption probability matrix, a t by q matrix with entries b_{ij} where t is the number of transient states $(N-1)$ and q is the number of absorbing states (two; absorbing states are $i = 0$ and $i = N$).

$$B_{(N-1)\times 2} = M_{(N-1)\times(N-1)} R_{(N-1)\times 2}$$

$$= \begin{bmatrix} 1-p_{N-1,N-1} & -p_{N-1,1} & \cdots & \cdots & \cdots & \cdots \\ -p_{1,N-1} & 1-p_{1,1} & -p_{1,2} & -p_{1,3} & \cdots & -p_{1,N-2} \\ \vdots & \ddots & \ddots & \ddots & \ddots & \vdots \\ \vdots & \ddots & \ddots & \ddots & \ddots & \vdots \\ -p_{N-2,N-1} & \cdots & \cdots & \cdots & \cdots & 1-p_{N-2,N-2} \end{bmatrix}^{-1} \begin{bmatrix} p_{N-1,0} & p_{N-1,N} \\ p_{1,0} & p_{1,N} \\ \vdots & \vdots \\ \vdots & \vdots \\ p_{N-2,0} & p_{N-2,N} \end{bmatrix}$$

We examine fixation probability as the probability of beginning with one mutant and resulting in N mutants. Thus, the absorption probability that represents fixation, the probability of interest, is $b_{1,N}$.

For a well-mixed population, having set up the transition matrix Q in canonical form, we find the fixation probability $b_{1,N}$ as the entry in the second row and second column of the matrix MR. We go on to simulate this for well-mixed populations and extend the result to birth-death communication as per the constraints of our model on Barabási-Albert graphs.

We simulate the birth-death process with one-to-many communication on a Barabási-Albert graph of size $N = 100$ nodes and an average of $k = 16$ connections for each node. This choice of illustrative parameters was informed by a recent study revealing that on average, Americans have sixteen friends, each with whom they would willingly communicate in some capacity [13].

2.3 Simulations on a Well-Mixed Population

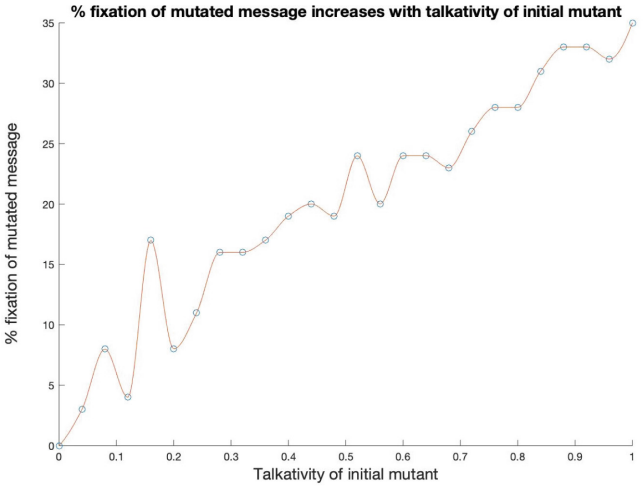
For simulations on a well-mixed population, we studied fixation probability of the mutated message given that the population begins with only one mutant. All talkativities and belief probabilities were generated as random number vectors. We examine the trend between talkativity or belief probability of the original mutant and the likelihood of its fixation. The percentage of simulations that reached fixation provides an indication of the fixation probability with respect to the talkativity and the belief probability of the initial mutant.

2.4 Simulations on a Barabási-Albert Graph

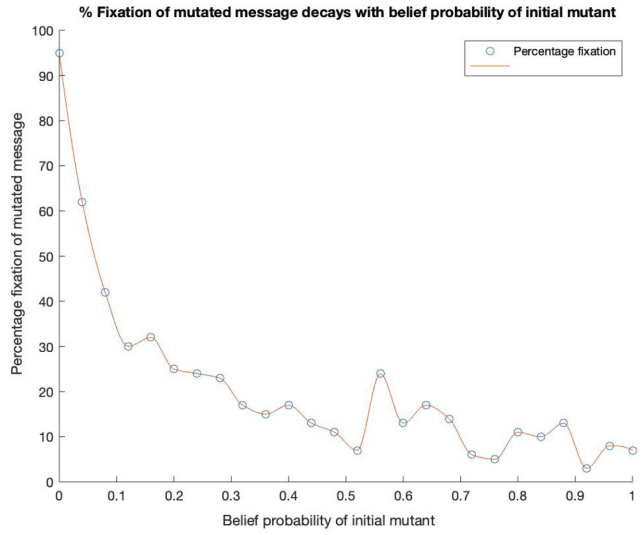
For simulations on a Barabási-Albert (BA) graph, we study the effect of talkativity and belief probability on the percentage of simulations in which the mutated message spread to at least one additional node. Fixation is fairly unlikely on a BA graph given the added stochastic noise and the probabilistic barriers created by the existence or lack of connection between two nodes. Thus, we chose to study the reduced case of simulations in which the mutated message is spread to at least one other node. The percentage of simulations that involved the mutation spreading to at least one other individual provides an indication of the fixation probability with respect to the talkativity and belief probability of the initial mutant.

3 Discussion

The model offers an understanding of how word-of-mouth information propagation, the most common mechanism of information spread [14], works in human networks. Specifically, two significant factors, talkativity and belief probability, that affect the likelihood of information spread are incorporated to understand the stochastic barriers a message must overcome to fixate in a population. The fixation probability of mutated messages in different population structures with respect to the talkativity and the belief probability of the initial mutant illuminates several important trends. In a well-mixed population, levels of fixation increase near-linearly with the level of talkativity of the initial mutant (correlation $\rho = 0.96$) (Fig. 2a). This is an intuitive result, as an initial mutant who is more talkative is more likely to spread the mutated information, contributing to a greater likelihood of fixation. Decreasing levels of belief probability of the initial mutant ($\rho = -0.74$) correspond to decreasing fixation probabilities (Fig. 2b). One explanation for this trend is that if the initial mutant is more likely to believe information, particularly the non-mutated message, its mutated information has a reduced probability of spreading or fixating. Nevertheless, this explanation may not manifest in reality, as belief probability likely depends on the content of the information, while β in our case is independent of the message. Thus, another parameter might be necessary to explain this trend.

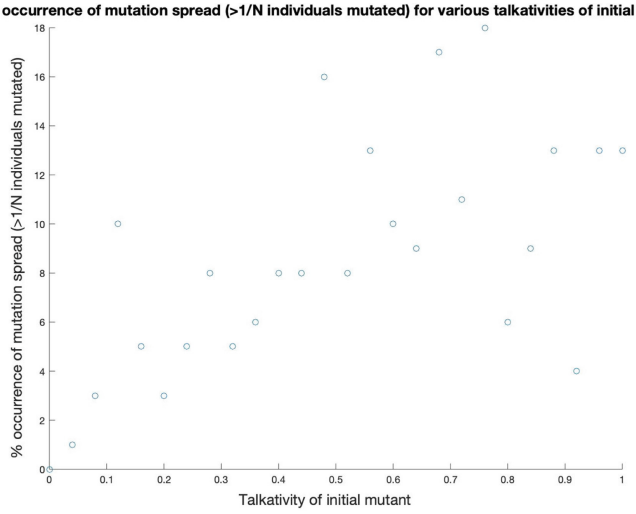


(a)

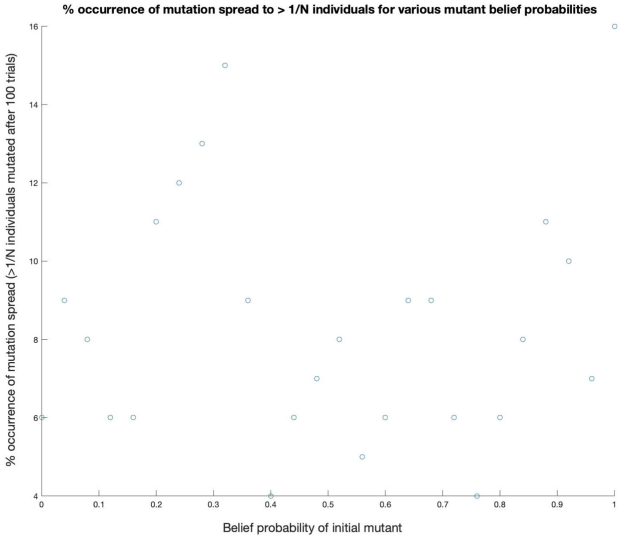


(b)

Fig. 2. Simulations of fixation of the mutated message in an unstructured, well-mixed population of $N = 100$ individuals at incremental values of talkativity and belief probability of the initial mutant. In (a), the percentage of fixation of the mutated message is measured against incremental τ values of talkativity. For each τ, 100 simulations were run, each with 10^5 time steps. In (b), the percentage of fixation of the mutated message is measured against incremental β values of the belief probability of the initial mutant. As with talkativity, 100 simulations were run for each x value of belief probability, each with 10^5 time steps. Smooth scatter lines simply connect consecutive points and do not imply any curvature or functional form of the relationship between outcome and parameters.



(a)



(b)

Fig. 3. Simulations of mutation spread on a BA graph of $N = 100$ individuals at incremental values of talkativity and belief probability of the initial mutant. In (a), the percentage of mutation spread is measured against incremental τ values of talkativity. At each τ, 100 simulations were run, each with 10^5 time steps. In (b), the percentage of mutation spread is measured against incremental x values of the belief probability of the initial mutant. As with talkativity, 100 simulations were run for each β value of belief probability, each with 10^5 time steps. Smooth scatter lines simply connect consecutive points and do not imply any curvature or functional form of the relationship between outcome and parameters.

Given the convoluted nature of the BA graph generated for Fig. 3a and 3b, fixation was unlikely because of added stochastic barriers, such as the probability that two nodes are connected, thereby reducing the available number of possible channels of communication. However, a weaker trend was observed in the percentage of mutation spread against talkativity ($\rho = 0.61$) and against belief probability ($\rho = -0.49$) of the initial mutant. Although, theoretically, talkativity and belief probability still have the same effect on the graph, the effects are diluted by other random factors intrinsic to the graph such as the likelihood of successful communication between two nodes. This introduces randomness on the graph, leading to no clear trends with respect to talkativity or belief probability.

An interesting social implication of the mathematical results of our model is the sheer difficulty of information becoming common knowledge or "fixating." Even in smaller subpopulations, the stochastic nature of information propagation renders it difficult for information to not only reach every individual, but also for every individual to believe the information. Due to stochastic barriers presented by the individuals' talkativities and belief probabilities, even the most valuable of information may never reach the individuals it should or would most benefit, as it cannot surpass the various probabilistic barriers required for successful transmission. An argument for stochastic barriers proving useful against fake news and misinformation is equally valid, but can be studied further to determine the probabilistic factors that enable the spread of misinformation.

A limitation of the present model is that the parameters considered are limited to talkativity and belief probability. Particularly, β in our model is a node's belief probability independent of the content of the message, but in human networks, a node's belief probability likely depends on the information itself.

An additional factor worth consideration is the relationship strength between two individuals or nodes. While talkativity and belief probability affect the likelihood of two nodes communicating, the strength of their relationship also impacts how likely any piece of information is communicated. Two individuals with low talkativity but with a high relationship strength may have a high probability of fixation despite having low talkativity. This may necessitate the use of a weighted network in the model. Along these lines, the effect of sub-networks is also significant. Within smaller networks of nodes, one node's talkativity and belief probability may not necessarily be independent of another node's. Nodes connected in a sub-network may be of a similar level of talkativity or belief probability. Our model also does not discriminate between the nature of communication through word-of-mouth and online platforms.

Understanding how information travels through the massive network that is humans is valuable for understanding how human beliefs evolve. Insightful contributions made to the existing pool of human knowledge may not necessarily manifest in beneficial ways due to various stochastic barriers; hence, understanding the degree to which said barriers impede communication can be helpful in ensuring information achieves its intended targets, particularly on technological platforms.

4 Appendix: Assumptions and Derivations

4.1 Model Assumptions:

Communication between nodes is restricted to at most one speaker speaking at a time. Only one speaker communicates at each time step, regardless of the number of recipients. Information spread from one node to another is assumed to have a binary trait of being mutated or non-mutated information. The model assumes the default state that a population begins with all the nodes believing the non-mutated information. Any pieces of information that is not the non-mutated information is considered a mutation. In reality, there are multiple speakers communicating at once and there may be more nuances as to the classification of different information.

We model mutation state as a binary state space: there are only two states a node can be in, either it is mutated or it is non-mutated. A mutated node in this model is a node that believes the mutated information and non-mutated node is one that either does not believe the mutated information and/or believes the non-mutated information.

The two forms of communication we model are one-to-one and one-to-many communication. In the one-to-many case, we assume that the number of receivers of a piece of information follows a binomial distribution $\text{Bin}(N, p)$. N here is the total population size in a well-mixed population. On a graph, N reduces to the number of neighbors of the speaker node. p is the probability of speaking to any one individual, independently.

4.2 Model Equations and Analytic Results

Analytic Results for One-to-One Communication:

- *Population shifts from i to $i-1$ mutants:*
  (1) is the product of the following probabilities:

$$\frac{\sum_{k=i+1}^{N} \tau_k}{r\sum_{k=1}^{i} \tau_k + \sum_{k=i+1}^{N} \tau_k} \tag{11}$$

$$\frac{\sum_{k=1}^{i} \beta_k}{N} \tag{12}$$

  (11) is the probability that a non-mutant speaks, weighted by the ratio of its talkativity to the sum of all individuals' talkativity and (12) is the probability that a mutant is the receiver and believes the non-mutated information.
- *Population shifts from i to $i+1$ mutants:*
  (2) is the product of the following probabilities:

$$\frac{r\sum_{k=1}^{i} \tau_k}{r\sum_{k=1}^{i} \tau_k + \sum_{k=i+1}^{N} \tau_k} \tag{13}$$

$$\frac{\sum_{k=i+1}^{N} \beta_k}{N} \tag{14}$$

(13) is the probability that a mutant speaks, weighted by talkativity, and (14) is the probability that a non-mutant is the receiver and believes the mutated information.

Acknowledgments. We thank Martin A. Nowak and Alexander Heyde of the Program for Evolutionary Dynamics (PED) at Harvard University for their guidance and support.

References

1. Nowak, M.A.: Evolutionary Dynamics: Exploring the Equations of Life, 1st edn. Harvard University Press, Cambridge (2006)
2. Yan, Q.: Information propagation in online social network based on human dynamics. Abstr. Appl. Anal. (2013). https://doi.org/10.1155/2013/9534068
3. Frias-Martinez, E., Williamson, G., Frias-Martinez, V.: An agent-based model of epidemic spread using human mobility and social network information. In: Proceedings of the 2011 IEEE Third International Conference on Privacy, Security, Risk and Trust and 2011 IEEE Third International Conference on Social Computing (2011)
4. Haeupler, B.: Simple, fast and deterministic gossip and rumor spreading. J. ACM (2015). https://doi.org/10.1145/2767126
5. Li, D., Ma, J.: How the government's punishment and individual's sensitivity affect the rumor spreading in online social networks. Phys. A (2017). https://doi.org/10.1016/j.physa.2016.11.033
6. Rabajante, J.F.: Investigating the propagation and death of information in human subpopulation networks. Int. J. Appl. Math. Res. **1**(4) (2012). https://doi.org/10.14419/ijamr.v1i4.336
7. Moran, P.A.P.: Random processes in genetics. Math. Proc. Cambridge Philos. Soc. (1958). https://doi.org/10.1017/S0305004100033193
8. Barabasi, A.L.: Network Science The Barabasi-Albert Model (2014)
9. Mazzoli, M., Re, T., Bertilone, R., Maggiora, M., Pellegrino, J.: Agent Based Rumor Spreading in a scale-free network (2018)
10. Rezvan, A., Meybodi, M.R.: Stochastic graph as a model for social networks. Comput. Hum. Behav. (2016). https://doi.org/10.1016/j.chb.2016.07.032
11. Souza, E.P., Ferreira, E.M., Neves, A.G.M.: Fixation probabilities for the Moran process in evolutionary games with two strategies: graph shapes and large population asymptotics (2018)
12. Ermon, S., Gomes C.P., Sabharwal A., Selman B.: Designing fast absorbing Markov chains. In: Proceedings of the Twenty-Eighth AAAI Conference on Artificial Intelligence (2014)
13. The reasons American adults find it hard to make new friends. https://www.independent.co.uk/life-style/friends-adults-american-how-to-friendship-difficulty-a8906861.html. Accessed 7 Nov 2019
14. Aramendia-Muneta, M.E.: Spread the word - the effect of word of mouth in e-marketing. Comm. Commun. Digital Age (2017). https://doi.org/10.1515/9783110416794-013