Marketing new Products: Bass Models on Random Graphs

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Abstract

We consider the problem of marketing a new product in a population modelled as a random graph, in which each individual (node) has a random number of connections to other individuals. Marketing can occur via word of mouth along edges, or via advertising. Our main result is an adaptation of the Miller-Volz model, describing the spread of an infectious disease, to this setting, leading to a generalized Bass marketing model. The Miller-Volz model can be directly applied to word-of-mouth marketing. The main challenge lies in revising the Miller-Volz model to incorporate advertisement, which we solve by introducing a marketing node that is connected to every individual in the population. We tested this model for Poisson and scale free random networks, and found excellent agreement with microscopic simulations. In the homogeneous limit where the number of individuals goes to ∞ and the network is completely connected our model becomes the classical Bass model. We further present the generalization of this model to two competing products. For a completely connected network this model is again consistent with the known continuum limit. Numerical simulations show excellent agreement with

microscopic simulations obtained via an adaptation of the Gillespie algorithm. Our model shows that, if the two products have the same word-of-mouth marketing rate on the network, then the ratio of their market shares is exactly the ratio of their advertisement rates.

1 Introduction

We are concerned with modelling the penetration of a market by one or more new products like a new type of cell phone, mattress, or item of clothing. The classical model describing this process is the well-known Bass model [2], first introduced in 1969. It assumes that a potential buyer population can be divided into a fraction which has already bought the product, $F(t)$, and $1 - F(t)$, the fraction that has not bought but consists of potential buyers (we will call this group "susceptibles", for reasons which will become clear). For this situation, Bass suggested that

$$\frac{dF}{dt} = p(1 - F) + qF(1 - F)\,, \tag{1}$$

where p represents the rate of spontaneous conversion into buyers due to advertising, and q represents the adoption rate of the product due to the word-of-mouth recruitment of a potential buyer. Interestingly, the simple model produces a good fit to empirical data [3].

The Bass model assumes a homogeneously mixed population (*i.e.*, each pair of individuals has the same chance of contact), an assumption which is clearly too simplistic. More realistic models of population contacts are social networks, where nodes represent individuals and edges connecting nodes represent contacts. It is thus of interest to understand how the underlying assumptions of the Bass model will work on social networks. Unfortunately, there currently are no models that can precisely describe dynamics of market penetration on general social networks. The purpose of our paper is to partially fill this gap.

The classical Bass model has been extended to special deterministic graphs [see, e.g. 5]. However, real populations are usually too large and too complex to be studied by this approach. Recent related research is concerned with stochastic simulations of the (accelerated) word-of-mouth propagation of information on the internet [13, 20]. Similar rumor diffusion processes on networks have been extensively studied both using stochastic simulations [see, e.g., 21] and theoretically [see, e.g. 11, 22].

A key observation is that product and rumor diffusion is similar to the spread of a disease in a population, where potential buyers are analogous to susceptible individuals, and buyers are analogous to infected individuals (this is the reason for our terminology). Indeed, most mathematical models in rumor diffusion on networks are based on a classic dynamic Susceptible-Infectious-Susceptible (SIS) epidemic model on random networks [14]. Another classic approach is the bond percolation method [12], which predicts the threshold and final epidemic size but cannot describe the dynamics of the diffusion. Lindquist et al. [9] showed that the Pastor-Satorras and Vespignani model can drastically overestimate the disease spread, and its Susceptible-Infectious-Recovered (SIR, i.e., individuals recover with lifetime immunity) model counterpart yields a larger diffusion threshold than the threshold predicted by the bond percolation method. Recently, disease models on random contact networks that agree very well with stochastic simulations have been developed [see, e.g. 9, 10, 18]. All three models yield a diffusion threshold identical to that of the bond percolation method. In this paper, we base our market diffusion model on the ideas of Miller [10] and Volz [18] because of their simplicity.

While the word-of-mouth method of promoting a product is akin to the spread of a virus via one-on-one infection, advertising provides the external influence, which can be thought of as providing "spontaneous" infection at some rate. This adds an extra twist to the modelling problem, analogous to the situation where a pathogen is present in the environment, such as the cholera bacterium in a water source. In this paper we model this external influence using a multigroup extension of the Miller-Volz model [10], due to Koch et al. [8], to the marketing of one or more products on random social networks.

A random network is a network (or graph) generated by some random process. This is an important class of graphs because it is usually impossible to determine the social network of a large population, and the degree distribution (the distribution of the number of contacts, called neighbours, of a node) in such networks is usually reconstructed from statistical data. Given a degree distribution, a random graph can then be constructed by a configuration model [see, e.g. 12]: each node is randomly assigned a degree from the given degree distribution, determining the number of "half edges" coming out of the node; then two "half edges" are uniformly chosen and connected to form an edge; this edge formation process is repeated until no edges can be formed. In practice, connecting "half edges" from the same node, or nodes

that are already neighbours, is disallowed in order to avoid self-loops and multiple edges. The process works quite well for large node numbers (say, $N = 10,000$) and reasonable edge distributions, like a Poisson distribution with a moderate and realistic average edge number, say, 25. The microscopic simulations at the end of our paper were done for networks generated in this way.

Our paper is structured as follows. We first revisit the Miller-Volz model [10], a concise and very effective model to simulate the spread of an infectious disease on a random graph. We reproduce it here for completeness and because of its importance for the sequel. Section 3 contains the derivation of the generalized Bass model. By suitable modification of the ideas underlying the Miller-Volz model we arrive at a new marketing model on social networks. We show that this model precisely captures the ensemble average of the underlying stochastic marketing process, and is equivalent to the Bass model in the limit of homogeneous mixing. In Section 4 we extend this network model to the case of two competing products. In Section 5 we compare microscopic simulations with simulations based on the new models and find excellent agreement. Under some simplifying assumptions on the model parameters we make predictions of the final market shares of the competing companies in Section 6. The homogeneous limit is discussed in an appendix.

2 A brief review of the Miller-Volz model

The Miller-Volz model [10] is a Susceptible-Infectious-Removed (SIR) epidemic model on random contact networks. It describes the spread of a non-fatal disease which ends in life-time acquired immunity. Susceptible individuals may become infectious upon contact with infectious individuals, and infectious individuals recover after an infectious period and will never be infected again. The random contact network is characterized by its degree distribution $\{P_k\}$ (the probability that a random node on the network has degree k), and alternatively represented via its generating function $\Psi(x) = \sum_k P_k x^k$, where x is a dummy variable. The power of this description will become apparent. In computer simulations a random network is normally constructed as described in the introduction. Edges (contacts) are considered as directed, and can be characterized by the type of nodes they connect; for example, there are $S \leftarrow I$ edges, $I \leftarrow S$ edges, etc.

We begin by setting the terminology. The fundamental idea behind the

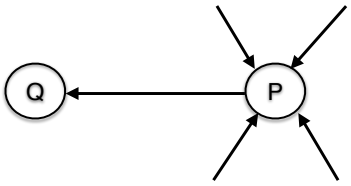

Figure 1: An illustration of the source node P, the target node Q, and the directed edges in the Miller-Volz model. The directions of the edges represent the directions of transmission. An edge in a contact network corresponds to two directed edges, the directed edge in the opposite direction is not shown here.

Miller-Volz model is to study the dynamics of the edges rather than the dynamics of the nodes. To this end, we consider a directed edge with source node P and target node Q, as depicted in Figure 1. If the source P is infectious, transmission occurs along this edge with rate β (i.e., β is a rate per edge, and independent of the number of target nodes). Transmission causes new infection only if the target node is susceptible.

Let $\theta(t)$ be the probability that a random edge has not transmitted "an infection" by time t. This θ is our first dependent variable.

A target node remains susceptible while none of its edges (contacts) has transmitted. If the node has degree k, then, assuming independence, the probability that it is susceptible is θ^k. In general, a random node is susceptible with probability

$$S(t) = \sum_{k=0}^{\infty} P_k \theta^k =: \Psi(\theta)\,,$$

where $\Psi(x)$ is the previously defined probability generating function of the degree distribution $\{P_k\}$. The probability $S(t)$ is also the fraction of susceptible nodes in the population. The fraction of infectious nodes $I(t)$ increases because susceptible nodes get infected, and decreases because infected nodes recover (with a constant rate γ per node). Thus,

$$\frac{dI}{dt} = -\frac{dS}{dt} - \gamma I = -\Psi'(\theta)\frac{d\theta}{dt} - \gamma I\,.$$

To understand how fast an average node becomes infectious, the Miller-Volz model focusses on the dynamics of θ. An edge in class θ loses its status

only when it transmits, *i.e.*, when a transmission occurs along it. Let $\phi(t)$ be the probability that a randomly chosen edge is of class θ (*i.e.*, has not transmitted) and has an infected source; this is exactly the fraction of edges that can attempt transmission but have not yet transmitted. Therefore,

$$\frac{d\theta}{dt} = -\beta\phi\,, \tag{2}$$

where β is the disease transmission rate along an edge. Note that by these definitions $p_I := \phi/\theta$ is the conditional probability that the source node of an edge belonging to the θ class is infected. The probability p_I is closely related to I and becomes I in a suitable limit (mentioned below).

An edge of type ϕ can change status only because of a transmission along it, or because of recovery of the infected source. An edge can enter class ϕ only if its source (which has degree k with probability $kP_k/\sum_{i=0}^{\infty} iP_i$) becomes infected. This once-susceptible source, given that it has degree k, can be infected only if at least one of its other $k-1$ edges is of class ϕ. Thus,

$$\frac{d\phi}{dt} = -(\beta+\gamma)\phi + \beta\sum_{k=0}^{\infty}(k-1)\phi\theta^{k-2}\frac{kP_k}{\sum_{i=0}^{\infty} iP_i} = -(\beta+\gamma)\phi + \beta\phi\frac{\Psi''(\theta)}{\Psi'(1)}\,. \tag{3}$$

These two differential equations for θ and ϕ form the Miller-Volz model. The fractions S and I can be recovered from θ and ϕ as shown above. The gain term in 3 can also be written as $-h'(t)$, where

$$h(t) = \sum_{j=1}^{\infty}\theta^{j-1}\frac{jP_j}{\sum iP_i} = \frac{\Psi'(\theta)}{\Psi'(1)} \tag{4}$$

is the probability that a θ-edge has a susceptible source.

We have committed a modest abuse of notation in the sense that S, I, θ and ϕ will denote fractions (or, more precisely, probabilities) as defined above, but we also talk of S-nodes, edges of type θ, etc. This practice will continue in the rest of our paper.

As already noted by Miller [10] this model is a genuine extension of the classical SIR model. This means that in a homogeneously mixed population, seen as a contact network on a complete graph, the Miller-Volz model should become the classic SIR model

$$\frac{dS}{dt} = -qSI\,,$$
$$\frac{dI}{dt} = qSI - \gamma I\,,$$

where $q = (N-1)\beta$ is the per capita transmission rate in a population of N individuals.

Treating the infectious individuals I as buyers, this SIR model is analogous to the Bass model with only word-of-mouth marketing if $\gamma = 0$, because a buyer remains a product holder and continues to pass information forever; this is equivalent to no recovery after infection in the disease model (*i.e.*, $\gamma = 0$ and $S = 1 - I$). Of course, "recovery" (e.g., an individual abandons a product and is open to buy others) is an option, and can easily be incorporated into our modeling. Here we will only consider the case $\gamma = 0$.

3 Marketing one or more products in a population modelled as a random graph

As in the classical Bass model the marketing process will include two processes: a word-of-mouth transmission on a random social network (called W in the sequel), and transmission by advertising which is assumed to reach every individual in the social network to the same extent. As above, the word-of-mouth random network W can be realized by the configuration model.

To include advertisement, we assume that there is one more node (the producer) outside W, denoted by A, which has one connection to each node in W. These connections are used for advertising purposes and can therefore transmit.

3.1 The generalized Bass model with no marketing

First, we ignore the advertising node A, and only consider the word-of-mouth network W. Let $\theta_W(t)$ and $\phi_W(t)$ be the $\theta(t)$ and $\phi(t)$ of the Miller-Volz model restricted to W, *i.e.*, $\theta_W(t)$ is the probability that a random edge in W has not transmitted "an infection" by time t, and $\phi_W(t)$ is the probability that a randomly chosen θ_W edge has a source in W that is in the buyer class. As above we commit a small abuse of notation and use θ_W and ϕ_W to denote both the fraction and the class of edges which have not transmitted. The only difference between the marketing process and an SIR infectious disease model is that a buyer (infected node) remains a buyer (will never recover). Thus, the word-of-mouth dynamics is the same as in the Miller-Volz model

with the recovery rate $\gamma = 0$, *i.e.*,

$$\frac{d\theta_W}{dt} = -\beta\phi_W\,, \tag{5a}$$

$$\frac{d\phi_W}{dt} = -\beta\phi_W + \beta\phi_W\frac{\Psi''(\theta_W)}{\Psi'(1)}\,, \tag{5b}$$

$$S = \Psi(\theta_W)\,, \tag{5c}$$

$$\frac{dI}{dt} = \beta\phi_W\Psi'(\theta_W)\,. \tag{5d}$$

A random network may have disconnected components. For example, two degree one nodes may be connected and form an isolated pair. This is more obvious on a scale free network, which has many degree-one nodes. Thus, word of mouth may not be able to reach everyone on the social network. The expected final fraction of buyers can be computed as in Miller [10]. Specifically, as $\theta_W(t)$ is a positive and decreasing function, $\theta_W(\infty)$ exists. Thus, the fraction of susceptible nodes that never become buyers as time $t \to \infty$ is $S(\infty) = \Psi(\theta_W(\infty))$. To compute $\theta_W(\infty)$, we first simplify (5a) and (5b). Dividing ϕ'_W by θ'_W yields

$$\frac{d\phi_W}{d\theta_W} = 1 - \frac{\Psi''(\theta_W)}{\Psi'(1)}\,.$$

Integrating on both sides, with $\phi_W(\theta_W(0)) \approx \phi_W(1) \approx 0$, leads to

$$\phi_W = \theta_W - \frac{\Psi'(\theta_W)}{\Psi'(1)}\,.$$

Substituting into (5a) results in

$$\theta'_W = -\beta\theta_W + \beta\frac{\Psi'(\theta_W)}{\Psi'(1)}\,.$$

Thus, $\theta_W(\infty)$ is the interior root (i.e., strictly between 0 and 1) of

$$\theta_W = \frac{\Psi'(\theta_W)}{\Psi'(1)}\,. \tag{6}$$

3.2 Including outside marketing

Now we incorporate the marketing node A in the model. We denote by $\theta_A(t)$ the fraction of all edges with source A which have not transmitted by time t. A node in the word-of-mouth network W remains susceptible if and only if information has been transmitted neither by word of mouth nor by A-edges, so Equation (5c) must now be modified to become

$$S(t) = \Psi(\theta_W)\theta_A \,. \tag{7}$$

The edges in the class θ_A only leave the class because of transmission. Thus,

$$\frac{d\theta_A}{dt} = -\alpha\theta_A \,. \tag{8}$$

Further, in comparison to (4), the probability that the source node of a θ_W edge is susceptible should be modeled as

$$h(t) = \frac{\Psi'(\theta_W)}{\Psi'(1)}\theta_A \,.$$

The reduction of this probability by the infection of a susceptible source causes a θ_W edge to enter ϕ_W. Thus,

$$\frac{d\phi_W}{dt} = -\beta\phi_W - h'(t) = -\beta\phi_W + \beta\phi_W\frac{\Psi''(\theta_W)}{\Psi'(1)}\theta_A + \ \alpha\theta_A\frac{\Psi'(\theta_W)}{\Psi'(1)} \,. \tag{9}$$

The A-edges have no direct influence on the θ_W edges, and hence Equation (5a) remains the same.

To obtain initial conditions, we assume that, initially, every node is susceptible, and no edge has transmitted. In summary, the network marketing model is:

$$S(t) = \Psi(\theta_W)\theta_A \,, \tag{10a}$$

$$\frac{d\theta_A}{dt} = -\alpha\theta_A \,, \tag{10b}$$

$$\frac{d\theta_W}{dt} = -\beta\phi_W \,, \tag{10c}$$

$$\frac{d\phi_W}{dt} = -\beta\phi_W + \beta\phi_W\frac{\Psi''(\theta_W)}{\Psi'(1)}\theta_A + \ \alpha\theta_A\frac{\Psi'(\theta_W)}{\Psi'(1)} \,, \tag{10d}$$

with initial conditions $S(0) = 1$, $\theta_W(0) = 1$, $\phi_W(0) = 0$, $\theta_A(0) = 1$.

4 Two competing products

In addition to the external node A (e.g., Apple) there is now a second external node B (e.g., Microsoft), also connected by an edge to each node in W, which competes with A to place their product. The susceptibles (initially all nodes in W) can therefore turn into two different kinds of buyers, I_A and I_B. The probability θ_W is defined as before, but now there are fractions ϕ_A and ϕ_B of edges in W which have not transmitted but originate in an I_A or I_B, respectively. There are also two possibly different word-of-mouth transmission rates β_A and β_B. By θ_A and θ_B we denote the fractions of edges from A into W (and B into W) which have not transmitted.

As before, we assume that, initially, every node is susceptible, and no edge has transmitted. This leads to the following equations:

$$
\begin{aligned}
S(t) &= \Psi(\theta_W)\theta_A\theta_B\,, && \text{(11a)}\\
\theta_A' &= -\alpha_A\theta_A\,, && \text{(11b)}\\
\theta_B' &= -\alpha_B\theta_B\,, && \text{(11c)}\\
\theta_W' &= -\beta_A\phi_A - \beta_B\phi_B\,, && \text{(11d)}\\
\phi_A' &= -\beta_A\phi_A + \frac{\Psi''(\theta_W)}{\Psi'(1)}\beta_A\phi_A\theta_A\theta_B + \alpha_A\frac{\Psi'(\theta_W)}{\Psi'(1)}\theta_A\theta_B\,, && \text{(11e)}\\
\phi_B' &= -\beta_B\phi_B + \frac{\Psi''(\theta_W)}{\Psi'(1)}\beta_B\phi_B\theta_A\theta_B + \alpha_B\frac{\Psi'(\theta_W)}{\Psi'(1)}\theta_A\theta_B\,, && \text{(11f)}
\end{aligned}
$$

with the same initial conditions as in (10), except $\phi_A(0) = \phi_B(0) = 0$, $\theta_A(0) = \theta_B(0) = 1$.

The gain terms in the third and fourth equations add up to $-h'$, where $h(t)$ now is given by

$$h(t) = \frac{\Psi'(\theta_W)}{\Psi'(1)}\theta_A\theta_B.$$

This h is the probability of reaching a susceptible node if one follows a W-edge from a susceptible inside W. The rate of change of h tells us how fast this susceptible turns into an infected, and we have to distinguish whether it turns into an I_A or an I_B node. The four terms in h' are split in just the right way to reflect this.

Equations (11) also enable us to compute the fractions I_A, I_B of users

who have bought products A or B, respectively. Specifically, compute

$$\begin{aligned} S' =& \Psi'(\theta_W)\theta'_W\theta_A\theta_B + \Psi(\theta_W)\theta'_A\theta_B + \Psi(\theta_W)\theta_A\theta'_B \\ =& -\beta_A\Psi'(\theta_W)\phi_A\theta_A\theta_B - \alpha_A\Psi(\theta_W)\theta_A\theta_B \\ & -\beta_B\Psi'(\theta_W)\phi_B\theta_A\theta_B - \alpha_B\Psi(\theta_W)\theta_A\theta_B\,. \end{aligned}$$

The first two terms on the right clearly generate A-buyers, the last two terms generate B-buyers. Hence,

$$I'_A = \beta_A\Psi'(\theta_W)\phi_A\theta_A\theta_B + \alpha_A\Psi(\theta_W)\theta_A\theta_B\,, \tag{12a}$$

$$I'_B = \beta_B\Psi'(\theta_W)\phi_B\theta_A\theta_B + \alpha_B\Psi(\theta_W)\theta_A\theta_B\,. \tag{12b}$$

5 Comparison with stochastic simulations

A numerical test gave perfect agreement of the behaviour predicted by this model with microscopic simulations performed using Gillespie's algorithm [6, 7]. We compare our models with the underlying stochastic marketing process on two types of networks, namely a Poisson network and a scale-free network. In a Poisson network, every pair of nodes has identical probability of being connected by an edge. Thus, such a network has a Poisson degree distribution

$$P_k = \frac{\lambda^k e^{-\lambda}}{k!}\,,$$

and

$$\Psi(x) = e^{\lambda(x-1)}\,,$$

and is similar to a homogeneously mixed population. A scale-free network has a power-law degree distribution

$$P_k \propto k^r$$

where $r < -1$, and is commonly used to model social networks [see, e.g., 1]. The random networks are generated by the configuration model as discussed in the introduction. The network parameters are listed in Figure 2.

Each node in the social network is labeled as either a susceptible or a buyer. If it is a buyer, it is also labeled with the product it buys. To model marketing, a marketing node is created for each product. This marketing node is also labeled as a buyer, and is connected to every node in the social

network. Buyers transmit to their neighbors at rate β per edge, and convert their neighbors to buyers of the same product. Each marketing node transmits at rate α per edge, and converts its neighbors to a buyer of its product. Once a node becomes a buyer, it is always a buyer, and cannot be converted to the other product.

A fixed network is generated, and simulations are repeated on this network. The ensemble average of the simulations are computed and compared with our marketing models. The degree distribution of the generated network is used for the computation of Ψ in our models.

Figures 2 and 3 show the comparison between the single-product model (10) and the two-product model (11) with the corresponding stochastic simulations, respectively. Both models agree with the ensemble average of stochastic simulations very well. We do not average over multiple realizations of random networks. However, as these averages should give the provided degree distribution for the configuration model, the average over both the random networks and the simulations on each random network should agree with the solutions of our models with this provided degree distribution.

Figure 4 shows that in a scale-free network, which typically has many nodes in disconnected components, the product first diffuses quickly through word of mouth and advertisement in the connected components, then diffuses slowly in disconnected components through advertisement only. The expected fraction of nodes in disconnected components is $\Psi(\theta_W(\infty))$ where $\theta_W(\infty)$ is determined by (6).

6 Market share and advertisement

As explained in (12), the two-product competition model gives the following equations for the fractions of buyers:

$$I_A' = \beta_A \Psi'(\theta_W)\phi_A\theta_A\theta_B + \alpha_A\Psi(\theta_W)\theta_A\theta_B\,,$$
$$I_B' = \beta_B \Psi'(\theta_W)\phi_B\theta_A\theta_B + \alpha_B\Psi(\theta_W)\theta_A\theta_B\,.$$

For the case where the transmission rates $\beta_A = \beta_B$ are equal, it is rather straightforward to predict the market shares companies A and B will achieve:

Theorem 6.1. *Let $\beta_A = \beta_B = \beta$ and $\phi_A(0) = \phi_B(0) = 0$. Then, for all $t > 0$,*

$$\frac{I_A}{I_B} = \frac{\alpha_A}{\alpha_B}. \tag{13}$$

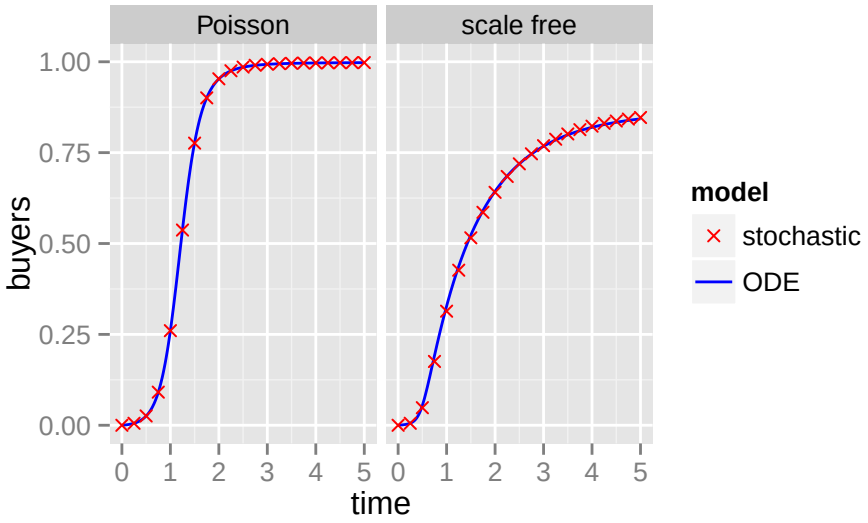

Figure 2: The comparison of the single-product model (10) with stochastic simulations (average of 500 runs) on a Poisson and a scale-free network. The network size for both networks is $N = 20,000$. For the Poisson network, the average degree $\langle k \rangle = 6$. For the scale-free network, the degree distribution is $P_k \propto k^r$ where $r = -2$, with a maximum degree $k_{\max} = 66$. The word-of-mouth transmission rate $\beta = 1$, the advertisement rate $\alpha = 0.01$. The long term dynamics on the scale free network is shown in Figure 4.

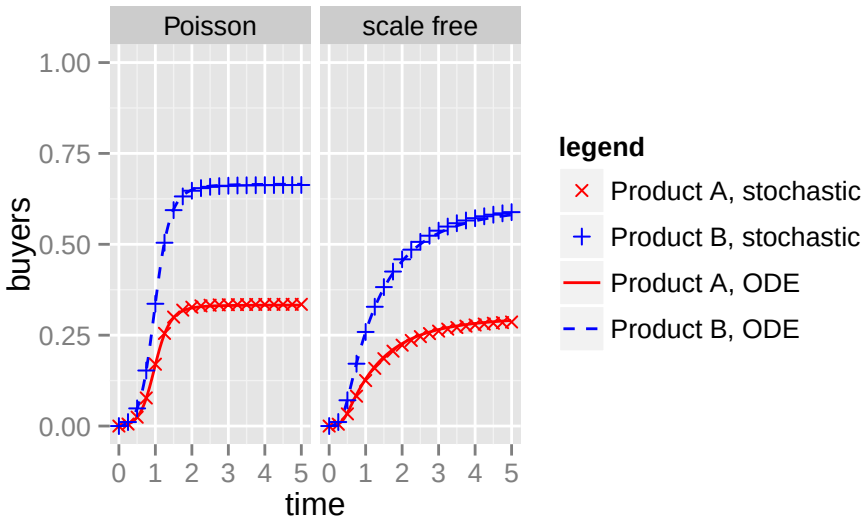

Figure 3: The comparison of the two-product model (11) with stochastic simulations on a Poisson and a scale-free network. The networks are the same as in Figure 2. The word-of-mouth transmission rates are $\beta_A = \beta_B = 1$, the advertisement rate $\alpha_A = 0.01$, $\alpha_B = 0.02$.

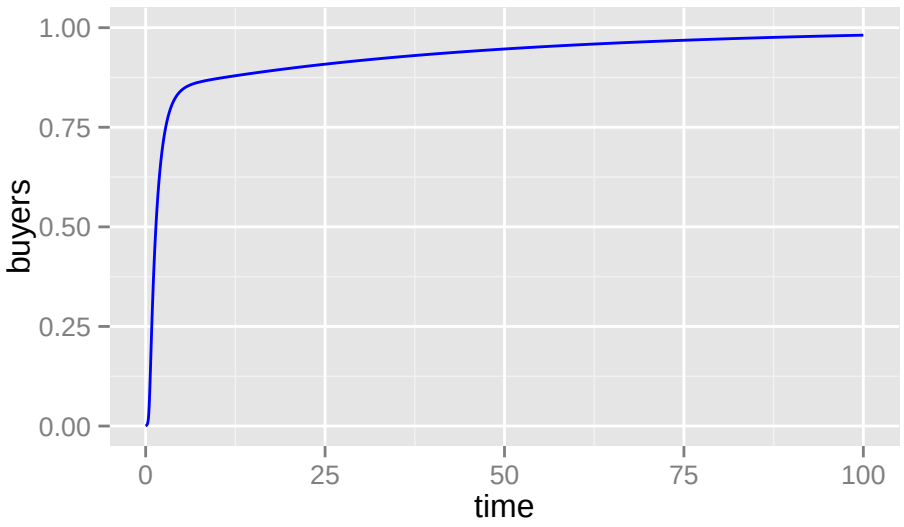

Figure 4: The fraction of buyers on a scale-free network converges to unity in two stages: it increases quickly and spreads through the large connected component, then approach unity exponentially through advertisement in disconnected components. The network and disease parameters are the same as in Figure 2.

This means that relative market share is proportional to relative advertising effort, regardless of the underlying network.

Proof. Since

$$\begin{aligned}(\alpha_B I_A - \alpha_A I_B)' &= \alpha_B I_A' - \alpha_A I_B' \\ &= \beta\Psi'(\theta_W)\theta_A\theta_B(\alpha_B\phi_A - \alpha_A\phi_B)\end{aligned} \tag{14}$$

we observe that the assertion will hold if we can prove that

$$\alpha_B\phi_A - \alpha_A\phi_B = 0 \tag{15}$$

is satisfied for all t. By assumption this holds for $t = 0$.

From Model (11),

$$(\alpha_B\phi_A - \alpha_A\phi_B)' = (\alpha_B\phi_A - \alpha_A\phi_B)\left[-\beta + \beta\frac{\Psi''(\theta_W)}{\Psi'(1)}\theta_A\theta_B\right].$$

This fact combined with the initial condition $\phi_A(0) = \phi_B(0) = 0$ implies that (15) holds. Finally, using $I_A(0) = I_B(0) = 0$, the assertion follows. □

Of course, the assumption that $\beta_A = \beta_B$ is overly simplistic. In practice, it is to be expected that these rates are not only different (consumers may on average like one product more than the other, and transmit with more enthusiasm), but they may also change with time. Our models readily adapt to this reality.

7 Concluding Remarks

We have generalized the classical Bass model for market penetration with one or two new products to the contexts of populations modelled as random networks of configuration type. The resulting systems of equations involve the generating functions of the edge distributions under consideration but are otherwise simple ODE systems, which allow efficient simulations of product adoption in random networks. Indeed, numerical simulations comparing the predictions of the new models with microscopic stochastic simulations show excellent agreement. In the homogeneous limit our models are consistent with the classical Bass model. We also saw that the models allow qualitative conclusions about market shares: Indeed, if the transmission rates for

word-of-mouth transmission inside the network are independent of the type of product and if the competing companies start at the same time and in a "clean" population, then the ultimate market shares are determined completely by the advertising efforts.

Because of conceptual similarity, the single-product model (10) can be extended to study the spread of water-borne diseases such as cholera [see, e.g., 16, 17, 19]. The two-product model (11) can be similarly extended to study the spread of two competing diseases with strong cross-immunity, such as competing strains of seasonal influenza [see, e.g., 4, 15].

There are many additional numerical and analytical experiments to be done with these models, and we intend to address these issues in future work. For example:

- What are the ultimate market shares if the transmission rates β_A and β_B are different, but the same advertising efforts are used? While this question is not easily answered by analytical tools, it can now be dealt with by numerical simulation.
- What are optimal advertising strategies for such a scenario, given that advertising is expensive?
- What advantage does "an early marketing start" produce for one of the companies?
- It would be very interesting to match our model with real data. This, of course, will require collaboration with the business community.
- Our model assumes a fixed random network. How should one incorporate dynamic changes in the network?
- Our model is a deterministic model. But the real marketing process is a random process. What would the distribution of product uptakes in such a setting?

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A The homogeneous mixing limit

In a homogeneously mixed population as assumed in the original Bass model, the two-product case is modeled by the two equations

$$I'_A = q_A I_A S + \alpha_A S \tag{16a}$$

$$I'_B = q_B I_B S + \alpha_B S, \tag{16b}$$

where we have employed notation consistent with the previous sections. Here we will show that these equations emerge naturally in a suitable limit if the model from Section 4 is considered on a complete graph with $N \gg 1$. In this situation every node has degree $N-1$, thus $S = \Psi(\theta_W)\theta_A\theta_B = \theta_W^{N-1}\theta_A\theta_B$ and

$$\begin{aligned}\phi'_A &= -\beta_A\phi_A + (N-2)\theta_W^{N-3}\beta_A\phi_A\theta_A\theta_B + \theta_W^{N-2}\alpha_A\theta_A\theta_B\,,\\ \phi'_B &= -\beta_B\phi_B + (N-2)\theta_W^{N-3}\beta_B\phi_B\theta_A\theta_B + \theta_W^{N-2}\alpha_B\theta_A\theta_B\,.\end{aligned}$$

Thus

$$\begin{aligned}S' =&(N-1)\theta_W^{N-2}(-\beta_A\phi_A - \beta_B\phi_B)\theta_A\theta_B - \alpha_A\theta_W^{N-1}\theta_A\theta_B -\\ &\alpha_B\theta_W^{N-1}\theta_A\theta_B\\ =&-q_A S\frac{\phi_A}{\theta_W} - q_B S\frac{\phi_B}{\theta_W} - \alpha_A S - \alpha_B S,\end{aligned}$$

where $q_A = \beta_A(N-1)$, $q_B = \beta_B(N-1)$.

Recall that β_A and β_B are transmission rates per edge. The limit of interest is $N \to \infty, \beta_A \to 0, \beta_B \to 0$ such that q_A and q_B are constant. We can therefore write $\beta_A = \frac{q_A}{N-1},\ \beta_B = \frac{q_B}{N-1}$, and therefore

$$\theta'_W = -\frac{q_A}{N-1}\phi_A - \frac{q_B}{N-1}\phi_B \geq -\frac{q_A+q_B}{N-1}.$$

An integration gives

$$1 \geq \theta_W(t) \geq 1 - \frac{1}{N-1}(q_A+q_B)t,$$

which shows that $\theta_W(t) \to 1$ for any t as $N \to \infty$.

Asymptotically, we should have $I_A \approx \frac{\phi_A}{\theta_W}$ and $I_B \approx \frac{\phi_B}{\theta_W}$. In fact, we find that

$$\begin{aligned}\left(\frac{\phi_A}{\theta_W}\right)' =& \frac{1}{\theta_W}\phi_A' - \frac{\phi_A}{\theta_W^2}\theta_W' \\ =& -\beta_A\frac{\phi_A}{\theta_W} + (N-2)\theta_W^{N-3}\beta_A\frac{\phi_A}{\theta_W}\theta_A\theta_B + \theta_W^{N-3}\alpha_A\theta_A\theta_B + \\ & \beta_A\frac{\phi_A^2}{\theta_W^2} + \frac{\phi_A}{\theta_W}\beta_B\frac{\phi_B}{\theta_W} \\ =& \left[\frac{(N-2)q_A}{N-1}\frac{\phi_A}{\theta_W} + \alpha_A\right]\frac{S}{\theta_W^2} + \frac{1}{N-1}\frac{\phi_A}{\theta_W}\left[-q_A + q_A\frac{\phi_A}{\theta_W} + q_B\frac{\phi_B}{\theta_W}\right].\end{aligned}$$

As $N \to \infty$ while q_A, α_A and q_B are kept constant, and by using that $\theta_W(t) \to 1$,

$$\left(\frac{\phi_A}{\theta_W}\right)' = q_A S\frac{\phi_A}{\theta_W} + \alpha_A S.$$

Similarly, we have

$$\left(\frac{\phi_B}{\theta_W}\right)' = \left[\frac{(N-2)q_B}{N-1}\frac{\phi_B}{\theta_W} + \alpha_B\right]\frac{S}{\theta_W^2} + \frac{1}{N-1}\frac{\phi_B}{\theta_W}\left[-q_B + q_A\frac{\phi_A}{\theta_W} + q_B\frac{\phi_B}{\theta_W}\right],$$

and as $N \to \infty$ while q_B, α_B and q_A are kept constant,

$$\left(\frac{\phi_B}{\theta_W}\right)' = q_B S\frac{\phi_B}{\theta_W} + \alpha_B S.$$

Clearly, these two limit equations are just Equations (16).

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