

**CREATING CONTAGIOUS:  
HOW SOCIAL NETWORKS AND ITEM CHARACTERISTICS COMBINE TO SPUR  
ONGOING CONSUMPTION AND REINFORCE SOCIAL EPIDEMICS**

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***ABSTRACT***

Social epidemics occur when products or behaviors “take off” and achieve widespread prominence. But what leads certain things to become hugely popular and achieve continued consumption while others die out? This research examines how social networks and product characteristics combine to drive epidemic size and persistence. It goes beyond product adoption to consider how social interactions influence ongoing consumption. The authors build a psychologically realistic individual-level model of word-of-mouth (WOM), social contagion, and consumption. This model is then applied in a simulation study, and results (1) demonstrate how various factors interact to drive WOM and consumption and (2) shed light on the mechanisms through which social epidemics occur. Interest in most products naturally decays over time, so product characteristics (e.g., virality) and the network position of early consumers are critical for bolstering consumer enthusiasm. Importantly, however, they do so via different mechanisms, determining how often, and with what level of enthusiasm, products are talked about. The authors also suggest an agenda for future research on WOM, social contagion and social epidemics that aims to bridge macro-level, quantitative perspectives and micro-level, psychological perspectives on these complex social phenomena.

**KEYWORDS:** Word-Of-Mouth, Social Epidemics, Social Networks, Diffusion

Facebook.com caught on and spread like wildfire. Four years in, on April 8, 2009, the online social network reached 200 million users who log in almost daily to communicate with friends. While the user base has grown immensely, and is an integral part of many people's daily routine, other social networks have not done so well. Why has Facebook grown and persisted while other sites (e.g., Friendster) have not? Musician Britney Spears achieved similar ubiquity. Not only has she sold countless albums, but millions of fans loyally download and listen to her music and attend her concerts. Thousands of other musicians have not enjoyed similar levels of ongoing consumption. These are examples of social epidemics, or instances when cultural tastes and practices (e.g., products and behaviors) achieved widespread prominence.

What leads certain products to spread contagiously? While it is easy to see that particular products catch on and become successful, much less is known about why one item succeeds and another one fails. The marketing literature has a rich tradition of studying diffusion and new products (e.g., Bass 1969; Gatignon and Robertson 1985; Mahajan, Muller, and Bass 1990; Van den Bulte and Lilien 2001), as well as word-of-mouth (WOM) and viral marketing (e.g., Godes and Mayzlin 2004, 2008; Goldenberg, Libai, and Muller 2001; Goldenberg, Libai, Moldovan, and Muller 2007). The dominant focus, however, has been on aggregate outcomes (e.g., forecasting market size from early data), rather than individual consumer-level *transmission processes* behind such outcomes. What leads certain products to be discussed more frequently than others, and how might these patterns of conversations drive success or failure in the marketplace? Further, while diffusion literature has provided great insight into product adoption, less is known about social processes that drive *ongoing* consumption, a key driver of long-term product success. Why are some products tried once and discarded while others persist and continue to be consumed? While "better" products should, in general, be more likely to persist,

quality alone does not determine success (Salganik, Dodds, and Watts 2006), and social factors also play a role.

This paper suggests that word-of-mouth (WOM) reinforces enthusiasm for products, which in turn helps drive ongoing consumption. People must remain sufficiently enthusiastic about a product to continue consuming it, but because enthusiasm for many products declines over time (Wu and Huberman 2007), conversations with others can provide vital enthusiasm *reinforcement*. In particular, we examine how social networks and item (product) characteristics combine to influence enthusiasm-reinforcing WOM and thus drive epidemic size and persistence. While researchers have argued that network structure influences diffusion (e.g., Valente 2005; Watts 2002), or investigated why certain things are shared (Berger and Heath 2005; Heath, Bell, and Sternberg 2001), little work has integrated these perspectives to examine how they combine to influence *ongoing* product success. We not only (1) demonstrate how these potential drivers can *interact* to influence conversations and social epidemics, but also (2) use them to illuminate the WOM transmission processes that enables epidemics to occur and, critically, to persist (something rarely examined in prior work), and (3) build on this perspective to outline potential research directions that deepen understanding of social contagion and WOM by delving into the psychological processes behind them.

To study this area, we build an individual-level model of WOM, social contagion, and consumption. While our model is based on contagion models developed in applied physics and epidemiology, unlike many extant models, it is founded on a set of more psychologically realistic assumptions. We apply this model in a simulation study where we simulate hundreds of social epidemics, and focus on aspects that managers can manipulate. Managers cannot change the structure or composition of the underlying social network, but they can influence product

characteristics and who they target. Consequently, we vary certain product characteristics, and the network positions of early consumers or “initiators,” to illustrate how these factors, and their interactions, affect WOM and ongoing consumption. The results provide insight into what drives ongoing consumption, and have important managerial implications for product success.

The paper is organized as follows. First, we provide a brief overview of related research and describe some important unexamined issues that we attempt to address. Second, we present our individual-level model of social contagion and consumption. Third, we describe a simulation study based on this model. Finally, we summarize our findings and use them to provide a broader discussion, as well as a suggested agenda for future marketing research on WOM, social contagion, and social epidemics.

### ***LITERATURE ON SOCIAL CONTAGION AND SOCIAL EPIDEMICS***

Collective dynamics, including social epidemics, have been studied across numerous fields, including sociology, epidemiology, marketing, psychology, and economics. We briefly review related work before outlining some unexamined issues that we address in this paper.

#### ***Literature Focusing on Networks***

Network researchers in applied physics and sociology have examined how structural properties of networks influence epidemic size, or the number of people that adopt a product or catch a disease (Coleman, Katz, and Menzel 1957; Goldenberg et al. 2001; Klovdahl 1985; Watts 2002; Watts and Dodds 2007). Large epidemics or “cascades,” for example, are more likely to occur in more densely connected networks.

This literature, however, has less to say about which transmitted items will end up succeeding. A given network may have a high epidemic propensity (e.g., it is dense), but not all

products introduced in that network diffuse wildly. Some become contagious while others languish, so given they are diffusing across the same network, network structure alone is not enough to explain disparity in success. Similar issues relate to research suggesting that epidemics are driven by a critical mass of “easily influenced” people (Watts and Dodds 2007), as population influencability alone cannot explain why different items in the same network achieve differential success. By focusing on networks, this literature has tended to ignore how characteristics of items that spread along these paths influence transmission. While this literature has recognized that epidemic size is influenced by the “critical infectiousness” of a disease or item (Dodds and Watts 2004), there has been less attention to *why* certain things are more infectious than others.

### ***Literature Focusing on Item Characteristics***

Other researchers have taken a different perspective, focusing on how characteristics of products, viruses, and cultural items influence collective outcomes (e.g., Berger and Heath 2005; Hayes and Gubler 2006; Norenzayan, Atran, Faulkner, and Schaller 2006; Rogers 2003; Rubin 1995; Schaller, Conway, and Tanchuk 2002). The rich literature on diffusion of innovations, for example, has examined how item characteristics (e.g., complexity or relative advantage) influence what people adopt (see Rogers 2003 for a review). Though most work in this area has focused on adoption, or aggregate success, cultural items and products also differ in their infectiousness, or how likely they are to be talked about or shared. More surprising *New York Times* articles, for example, are more likely to make the most emailed list (Berger and Milkman 2009) and more disgusting urban legends are more likely to be shared (Heath, Bell, and Sternberg 2001). Thus, instead of considering how social structure affects diffusion, this

literature considers how characteristics of the items themselves affect their adoption, prominence, and transmission.

### ***IMPORTANT UNRESOLVED QUESTIONS***

While prior work has generated significant insights, a number of important questions remain unaddressed. (1) How do networks and item characteristics interact to drive epidemics? (2) Beyond first-time (or one-off) adoption, what drives ongoing consumption? (3) What aspects underlie a psychologically realistic model of the individual-level transmission process? We describe each before turning to our conceptualization, which addresses these questions in detail.

#### ***Lack of an Integrative Model***

First, little work has integrated social networks and item characteristics to provide a broader investigation of how they combine to drive aggregate outcomes (though see Frenzen and Nakamoto 1993). While network models are useful in identifying structural conditions under which epidemics are possible, little diffusion may actually occur if a particular item is unlikely to be transmitted. Similarly, certain items may be highly viral, but fail to spread widely if they start with people who are not well connected to the rest of the population. Truly understanding collective outcomes therefore requires comprehending how these different factors act in concert.

In an attempt to shed light on these complex phenomena, we examine how network and item characteristics *combine* to drive social epidemics. We focus on the interplay between (1) the nature of the product itself (i.e., properties of items that influence transmission and consumption) and (2) where the people who initiate the product's spread are positioned in the social network. Examining the confluence of these multiple factors not only provides deeper understanding of the drivers of social epidemics, but also how these underlying contagion processes operate.

### ***Ongoing Consumption vs. Adoption***

Second, the diffusion literature has largely ignored repeated consumption over time. Existing models typically focus on adoption (e.g., Bass 1969; Goldenberg et al. 2001; Watts and Dodds 2007). Coleman et al.'s (1957) classic *Medical Innovation* study, for example, examined whether social contagion influenced the first time doctors prescribed a new drug. But while adoption is useful, it only tells part of the story. Fifty people might adopt a new flavor of Pepsi, for example, but an adoption metric does not distinguish whether they continue to buy it, or try it only once. While musicians or websites want to encourage trial, success also depends on creating repeat users and long term fans. Focusing solely on adoption also ignores factors that drive product lifespan. Products can be disadopted, but by focusing almost solely on adoption, the diffusion literature has mostly ignored why products are abandoned (cf. Berger and Heath 2008), as well as drivers of enduring interest.

Consequently, we examine *ongoing* consumption. While people might be excited right after they adopt a product, this enthusiasm often decays over time. We investigate drivers of ongoing consumption while also demonstrating how this measure provides additional insight beyond adoption.<sup>1</sup>

### ***Assumptions and Psychological Realism***

Third, by focusing on aggregate outcomes, contagion models have tended to make unrealistic simplifying assumptions about the individual-level transmission processes behind these effects. The sociological literature has documented various individual-level motivations for social interactions, transmission, and influence (e.g., Burt 1992; Coleman 1988), but models built to investigate how social networks drive diffusion often ignore such richer individual-level

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<sup>1</sup> Ongoing consumption is similar to “re-infection” in epidemiological SIS models (Bartholomew 1976; Weiss and Dishon 1971) where a person who is not using a product at time  $t$  is “susceptible” and can be “infected” (i.e., use it) but is still “susceptible” after being “infected” the first time and therefore can be “re-infected” subsequently.

transmission processes (i.e., how people interact, pass on information, and react to this information). Conditional on meeting an infected person, for example, contagion models commonly assume that the probability someone is “infected” by others (i.e., the transmission probability) is random (e.g., Dodds and Watts 2004; Goldenberg et al. 2001). This is obviously unrealistic. Other approaches combine a deterministic component (e.g., a person’s propensity to transmit WOM) with a random component. This recognizes that some people are more talkative or influential than others, but it says little about why, and fails to illuminate plausible psychological drivers of transmission.

Realistic assumptions are important because aggregate outcomes taken from these models (e.g., epidemic size, market share, sales forecasts) depend on such individual-level assumptions (cf. the recent debate over Watts and Dodds 2007; see Shapira, Goldenberg, and Lowengart 2009), and becomes even more critical when such models are augmented with actual marketplace data as a forecasting method (e.g., Toubia, Goldenberg, and Garcia’s 2008 use of agent-based simulations to make better early-stage forecasts of new product diffusion). Thus, while existing models recognize that individual-level transmission processes drive macro-level phenomena, they have less to say about how, when, and why these aspects do so.

In particular, two aspects require attention: who people interact with (called “mixing” in epidemiology and physics) and the WOM transmission and impact processes (i.e., what people talk about and how they are affected by WOM). First, extant models often simplify the mixing process. Most models in epidemiology and physics (and the few related ones in marketing) assume people mix randomly (Dodds and Watts 2004; Keeling and Eames 2005) or that connections are organized in a lattice structure (mean-field mixing; cf. Goldenberg et al. 2004). These approaches are useful for modeling, but are unrealistic in the real *social* world. Extant

research has shown that people are disproportionately more likely to associate with similar others (Feld 1982), that real friendship networks rarely look like random networks or lattices (e.g., Watts and Strogatz 1998), and that the numbers of connections people have (“degree”) is typically heavily-skewed (Barabási and Albert 1999). Given that changes in network structures can produce different-sized epidemics or cascades (Watts 2002), it is particularly important that realistic network structures are used. Consequently, we take the more realistic (and common sense) approach of *social* network mixing (cf. Castellano, Fortunato, and Loreto 2007). This assumes that people are connected in a social network and have varying numbers of connections (i.e., friends). As in the real world, ties are opportunities for social interactions.

Second, extant models often make arbitrary assumptions regarding infection—or WOM transmission—probabilities. Conditional on contact being made, the probability of infection is often drawn from a diffuse probability distribution. Assuming that the probability ( $p_{ij}$ ) that current user  $j$  transmits WOM to person  $i$  is random (e.g.,  $p_{ij} \sim Uniform[0, 1]$ ), for example, is common in mainstream models (e.g., Dodds and Watts 2004; Goldenberg et al. 2001). This is unrealistic. People tend to talk about things they think their listeners would be interested in (Stephen and Lehmann 2009), and consume products that they hear people speak about more enthusiastically. Consequently, we use a more realistic model of how people interact, transmit WOM, and influence others’ consumption. Transmission probabilities in our model depend on characteristics of the talker, the receiver, and the item being discussed.

### ***THE CURRENT RESEARCH***

With these aspects in mind, we develop an individual-level model of social behavior that attempts to shed greater light on the processes driving social epidemics. We then use this

model in a simulation study to address our research questions. The process model is summarized in Figure 1. We now describe each component in detail.

[INSERT FIGURE 1 ABOUT HERE]

### ***Latent Enthusiasm for Products***

Consider a population of  $N$  people (indexed by  $i$ ) and a new product that is introduced at the beginning of period 1 ( $t = 1$ ). In each period person  $i$  can do two things: (1) transmit WOM about the product to others over the network (or not), and (2) consume the product (or not). What determines what they do?

Just as latent utility drives consumer choice in choice models, an individual-specific, product-specific and time-varying latent variable drives WOM transmission and product consumption. We call this *enthusiasm*, and it can be thought of as excitement about, and positive attitude toward, a given product. Just as viruses are contagious because they can spread between people, enthusiasm is contagious. Let  $Y_{it} \in [0,1]$  denote person  $i$ 's enthusiasm for a product at the beginning of period  $t$ .<sup>2</sup> Enthusiasm starts at 0, representing initial unawareness. People who are more enthusiastic about a product should be more likely to talk about it, speak positively when they talk, and consume it. Consequently, the higher  $Y_{it}$  the more likely person  $i$  is to transmit WOM about the product and consume it in period  $t$ .

### ***Enthusiasm Decay***

Enthusiasm for many products naturally decays over time. People have limited attention spans and an inherent desire for novelty (Hirschman 1980; Rogers and Shoemaker 1971), and what was exciting about a song or a website today becomes less exciting or novel over time (Wu

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<sup>2</sup> Time in our model is broken into discrete periods. In each period each person has one conversation with one of their social network neighbors (i.e., friends). Thus, time should be seen as tracking successive encounters as opposed to necessarily tracking days, weeks, months, or years.

and Huberman 2007). New information, or discussions with other enthusiastic individuals, can boost or reinforce enthusiasm (see below) but enthusiasm will otherwise decline.<sup>3</sup>

To capture decaying enthusiasm, let the beginning-of-period  $t$  enthusiasm be  $Y_{it} = (1 - \delta_i) \cdot Y_{i,t-1}^*$ , where  $Y_{i,t-1}^*$  is the enthusiasm at the *end* of the previous period.  $\delta_i \in [0,1]$  is the “decay rate” for the product for person  $i$ , and has two parts. First, enthusiasm for certain products declines more quickly, described by a product-specific decay rate ( $\delta$ ). Enthusiasm tends to decay more quickly for jokes than political candidates, for example, and certain brands (e.g., Porsche) may maintain enthusiasm more easily than others (e.g., Hyundai). More enjoyable or higher quality products, for example, should have smaller decay rates. Second, we include a person-specific component ( $\xi_i$ ) that allows for heterogeneity across people. Some people get bored more quickly than others, for example. Thus,  $\delta_i = \delta + \xi_i$ , where  $\xi_i$  is a random parameter from a probability distribution.<sup>4</sup>

### ***Competition for Conversation***

Products and cultural tastes compete with other interests for conversation. Conversations about a product can boost enthusiasm, but the number of available conversation topics is almost infinite. Thus even great products (that people are aware of) may be rarely talked about or used. To simplify this, without loss of generality, we assume that in each period a person can talk about one of two things: the focal product or an alternative outside good (a catch-all for anything else that could be talked about). As we discuss below, the propensity to talk about the focal product depends on individual enthusiasm for that product and is heterogeneous across people and time-varying. This means that, just like in regular life, products, ideas, practices, and

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<sup>3</sup> We discuss other potential patterns of enthusiasm change over time in the general discussion.

<sup>4</sup> In our simulation study we used a normal distribution with mean = 0 and standard deviation = .1, drawn independently for each person. We found this variance to be large enough to induce sufficient heterogeneity across peoples’ decay rates. We truncated any resultant decay rates below 0 to 0 and any above 1 to 1.

concepts compete for people (Mark 1998). The more people talk about one thing, the less time they have available to talk about others.

### *Social Contagion Process*

We specify a more realistic process for determining how enthusiasm toward a product spreads over social network ties. By spreading WOM about a product, people can boost others' enthusiasm and thus influence others' likelihoods of consumption. Network structure (which is exogenous and time-invariant) determines who talks to whom, such that only connected people can have conversations with—and potentially influence—one another. We now describe the components of our contagion process.

*Certain Individuals Start the Process.* Let there be a set of  $n \ll N$  people who are enthusiastic about the product at the beginning of period 1 (i.e., early consumers or “initiators”). These are the first people to find out, and be excited, about the product, either because they discovered it themselves or were “seeds” in a viral marketing campaign.  $Y_{i,t=1} = 0$  for non-initiators and  $Y_{i,t=1}^{initiator} \sim Uniform(\phi, 1)$  for initiators, where  $\phi \in [0, 1)$  is the product's “consumption threshold” (a product characteristic).<sup>5</sup>

*Selecting Initiators and Enthusiasm Reinforcement.* One of the key factors we examine is the selection of initiators; specifically, where they are positioned in the network. Contagion models usually pick people at random or use highly connected individuals (cf. Castellano et al. 2007; Dodds and Watts 2004; Shapira et al. 2009). The latter approach is prominent in marketing, where conventional wisdom suggests that well-connected people (i.e., high degree) should be targeted (e.g., Goldenberg et al. 2009).

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<sup>5</sup> This is the minimum enthusiasm required for person  $i$  to consume the product at the end of period  $t$ . Certain innovations have a lower threshold to adopt or consume (Rogers 2003). We are not theoretically interested in the effects of threshold (it should have an obvious effect on consumption, but is of little relevance to conversation), and our simulation findings below are robust to a full range of different consumption threshold levels.

Notwithstanding the current debate surrounding the role of degree or “social hubs” in diffusion of innovation (cf. Goldenberg et al. 2009; Shapira et al. 2009; Watts and Dodds 2007), does the same logic apply to ongoing consumption? When a conversation takes place we can think of enthusiasm flowing over a social tie from the transmitter to the recipient. Note that we do not require transmitters to currently consume a product in order to talk about it. For new products, enthusiasm will flow outwards from initiators to non-initiators, and then (hopefully) on to more non-initiators. But for products to *continue* to be consumed, enthusiasm must also be *reinforced* to combat its natural decay over time. Conversations can provide such reinforcement, but to sustain consumption, enthusiasm not only has to flow outwards from initiators, but also flow back to them to keep their enthusiasm high (which then allows for them to reinforce non-initiators’ enthusiasm, and so on).

How can enthusiasm be reinforced quickly and effectively? There are two possibilities: increasing transmission frequency (irrespective of how much enthusiasm is passed along each time), or increasing how much enthusiasm is passed along in each transmission (irrespective of how often this occurs). Frequency is similar to “blitz” advertising campaigns, where each ad exposure may not have much of an effect by itself, but add up after multiple exposures.

Increasing the amount of enthusiasm transmitted (e.g., how enthusiastically people talk) is made difficult by decay. Enthusiasm declines over time, so the longer the delay between transmissions, the less enthusiasm there will be left to transmit. Further, the greater network distance that enthusiasm has to travel between people, the more time that passes and hence the less enthusiasm that will likely remain. Consequently, the “paths” between pairs of non-directly connected people along which enthusiasm flows should be relatively “short” (in terms of the “degrees of separation” between them). Short paths mean that enthusiasm does not have to travel

too far (say from an initiator) to affect many people. Since it takes more time to travel larger distances in a network, shorter paths mean that enthusiasm spreads faster and therefore less is lost due to decay. In the specific case of enthusiasm flowing back-and-forth between initiators and non-initiators, shorter cycles (i.e., path lengths out from initiators and then back again) should also be better.

This suggests that where initiators are located in the network should impact ongoing consumption. Specifically, how “close” initiators are, on average, to everyone else is important, where closeness refers to the average “degrees of separation” a person is from others in the network.<sup>6</sup> Since one’s degree (number of friends) is not the same as how close they are to everyone else, connectedness may matter less than closeness. Someone on the outskirts of the network may have many friends, for example, but is far from everyone else. Thus, instead of degree centrality we focus on closeness centrality as a criterion for initiator selection.<sup>7</sup>

*Enthusiasm Transmission.* People do not talk to all their friends every period, and whether enthusiasm for a product spreads across a given tie in a given period is stochastic. For each person  $i$ , at the beginning of period  $t$ , one individual ( $j$ ) is randomly drawn from her set of friends (direct ties). Person  $j$  then becomes a *potential* transmitter of enthusiasm to person  $i$  in period  $t$  (i.e., they have a conversation about *something*). The influence that person  $j$  has on

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<sup>6</sup> Closeness centrality is inversely proportional to the average degrees of separation one is from others (cf. de Nooy, Mrvar, and Batagelj 2005). Though degree and closeness are often correlated in real social networks (making it hard to identify whether it is degree or closeness driving outcomes), it is possible to have low correlations between degree and closeness centralities between groups of initiators. I.e., the  $k_1$  people with the highest degree and the  $k_2$  people with the highest closeness do not excessively overlap (provided that  $k_1$  and  $k_2$  are small enough).

<sup>7</sup> In the network used in our simulation study there was a reasonable correlation between degree and closeness (.56). The rank-order (Spearman) correlation, however, was small (.03). We designated two groups of 20 people each as initiators. The groups of the top-20 degree and the top-20 closeness people only had seven overlapping people. This reduces the possibility that any effect of initiator position (based on closeness) is strongly confounded by initiators’ degrees. Underscoring the difference in these measures, our results differ substantially when high degree versus high closeness individuals are used (see below).

person  $i$  depends on (1) whether they talk about the product (as opposed to the outside good) and (2) how it is discussed (or the exact “dose” of enthusiasm the person  $i$  receives from person  $j$ ).

What people talk about depends on factors related to them, their relationship, and the items themselves (Frenzen and Nakamoto 1993; Stephen and Lehmann 2009). Let  $p_{ijt} \in [0,1]$  denote the probability that person  $j$  talks about the focal product to person  $i$  during period  $t$ . When deciding what to talk about, people usually consider both their own interests (i.e., their enthusiasm about the product,  $Y_{jt}$ ) and their conversation partner’s interests (i.e., the recipient’s enthusiasm about that product,  $Y_{it}$ ). When Mark decides what to tell Roger, for example, he not only considers what he is excited about but also what might interest Roger. The weight placed on these two aspects ( $\gamma_j$ ) varies across transmitters, with some transmitters preferring to talk about topics that they care about (i.e., “me-focus,” higher  $\gamma_j$ ), and others preferring to talk about topics that they think would interest their recipients (i.e., “them-focus”). This weight is a person-specific random variable drawn for each person from a *Uniform*(0,1) distribution.

What people talk about should also be influenced by characteristics of items. Irrespective of how enthusiastic transmitters and their receivers are, people may be more likely to talk about things that are particularly topical, surprising, or prominent. Even someone who tends to ignore politics or basketball may talk about politics near a presidential election, for example, or Michael Jordan because he is culturally prominent (Fast, Heath, and Wu 2009). Consequently, these aspects of products, which we call their *virality*,  $v \in [0,1]$ , also can influence  $p_{ijt}$ . The weight that a transmitter places on the combination of enthusiasm within the conversation dyad versus product virality when deciding what to talk about varies across people ( $\alpha_j \sim \text{Uniform}(0,1)$ ).

Combining these elements, the formal specification of the transmission probability for the product from person  $j$  to person  $i$  at time  $t$  is a linear combination of the within-person enthusiasm-based transmission probability and the product's virality:

$$p_{ijt} = \alpha_j \cdot p'_{ijt} + (1 - \alpha_j) \cdot v \quad (1)$$

Higher  $\alpha_j$  means more weight is put on enthusiasm over the product's virality by the transmitter. Further, based on the above discussion,  $p'_{ijt}$ , the within-person enthusiasm-based component of transmission probability in equation 1, is:

$$p'_{ijt} = \gamma_j \cdot Y_{jt} + (1 - \gamma_j) \cdot Y'_{it} \quad (2)$$

Given that people are never perfectly sure about others' interests,  $Y'_{it} = Y_{it} + \varsigma$ , where  $\varsigma$  is random noise<sup>8</sup> inducing uncertainty in person  $j$ 's perception of or belief about person  $i$ 's enthusiasm.

Whether person  $j$  talks about the product to person  $i$  at time  $t$  is a Bernoulli( $p_{ijt}$ ) random variable (where 1 is the product is the topic and 0 is the outside good is the topic). If Bernoulli( $p_{ijt}$ ) = 0 then no enthusiasm toward the focal product is transmitted (the "dose" is  $d_{ijt} = 0$ ). However, if Bernoulli( $p_{ijt}$ ) = 1 then the dose of enthusiasm is nonzero ( $d_{ijt} > 0$ ).

What determines the size of the dose ( $d_{ijt}$ )? Consistent with threshold models (e.g., Goldenberg et al. 2001; Watts and Dodds 2007), hearing about a particular product can boost one's enthusiasm and consumption likelihood. People do not always try a product the first time they hear about it, however, and often multiple doses of WOM are necessary to generate trial or maintain enthusiasm (Leskovec, Adamic, and Huberman 2007). Instead of the typical approach of drawing  $d_{ijt}$  from a probability distribution or setting a constant dose size, however, we adopt a more realistic perspective. Listening to someone talk about a particular product should affect a recipient's enthusiasm for that product, but exactly how it affects them should vary based on

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<sup>8</sup> In the simulation study this noise is drawn independently from a normal distribution with mean = 0 and standard deviation = .1. We truncated the resulting enthusiasm to lie between 0 and 1.

characteristics of the transmitter and the recipient. More enthusiastic transmitters probably speak more highly of the product, for example, and more recipients are probably more receptive. Consequently, we allow  $d_{ijt}$  to be a function of the transmitter's and the recipient's existing enthusiasm toward the product. Given that the product is talked about,  $d_{ijt}$  lies between the transmitter's and the recipient's enthusiasm levels; i.e.,  $d_{ijt} \sim \text{Uniform}(a, b)$ ,  $a = \min(Y_{it}, Y'_{jt})$  and  $b = \max(Y_{it}, Y'_{jt})$ . Person  $i$  then has an end-of-period enthusiasm toward the product of:

$$Y_{it}^* = \begin{cases} 0 & \text{if } Y_{it} + d_{ijt} \leq 0 \\ Y_{it} + d_{ijt} & \text{if } 0 < Y_{it} + d_{ijt} < 1 \\ 1 & \text{if } Y_{it} + d_{ijt} \geq 1 \end{cases} \quad (3)$$

This becomes person  $i$ 's enthusiasm at the beginning of period  $t + 1$  (before applying decay).<sup>9</sup>

### ***Determining Product Consumption***

After conversations have taken place, and doses of enthusiasm have been transmitted, each person ( $i$ ) either does or does not consume the product at the end of each period ( $t$ ). This is determined by comparing  $Y_{it}^*$  to  $\phi$ , the consumption threshold. If  $Y_{it}^* > \phi$  person  $i$  consumes the product at the end of period  $t$ , otherwise they do not. For a person to continue to consume the product in successive periods, their enthusiasm must stay above the threshold *despite decay*. Thus, enthusiasm reinforcement, as discussed previously, is critical.

## ***SIMULATION STUDY***

We use a simulation study to demonstrate how product characteristics (decay, virality) and initiator network position can combine and interact to reinforce product enthusiasm and drive ongoing consumption. Though real contagion data would be ideal, it is not really possible

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<sup>9</sup> The truncation on post-dose enthusiasm keeps enthusiasm on the  $[0,1]$  interval. This recognizes that there are likely limits to a person's enthusiasm (or lack thereof) for any given product.

given the complexity required. We would need (1) individual-level data on (2) ongoing consumption *and* (3) WOM transmission, for (4) multiple products (5) over time in (6) a social network. Importantly, since we are explicitly interested in how network and product characteristics interact, (7) initiators' network positions and (8) products' characteristics would have to vary *across* products. Consequently, we turn to stochastic “agent-based” simulation methods. These are useful for studying interplays between individual-level (micro) factors and aggregate-level (macro) outcomes in complex systems, and have been used in marketing to study diffusion (e.g., Goldenberg et al. 2001, 2007; Libai, Muller, and Peres 2005).

We use this method to “experimentally” examine how parameters in our model interact to drive social epidemics. We manipulate characteristics of the product (decay [ $\delta$ ] and virality [ $\nu$ ]), as well as the position of the initiators within the social network (high vs. low closeness; position is exogenous to the model). These factors are at least somewhat controllable by managers through the marketing mix; that is, managers can influence product characteristics (e.g., design, advertising, positioning) and can select initiators (segmentation, targeting). We are interested in how these factors *combine* to affect consumption.<sup>10</sup> We vary them across an extensive multidimensional space in a full factorial experiment and simulate consumption and conversation data from our individual-level model. We then use statistical analysis across hundreds of scenarios to estimate the effects of the parameters and, critically, their interactions.

### ***Simulation Setup***

We use a fixed population size ( $N = 1,000$ ) of individuals. In each run of the simulation (one experimental cell in the full factorial; see below), one new product is introduced into the population at period  $t = 1$  by seeding a set of  $n = 20$  initiators (2% of the population). Each run is

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<sup>10</sup> As noted earlier, consumption threshold ( $\square$ ) is not of theoretical interest and used as a control factor. The  $\alpha$  and  $\gamma$  parameters (see equations 1 and 2) were not manipulated since they are outside managerial control. We discuss how these variables may be useful in future research later.

for 200 periods.<sup>11</sup> Per the individual-level model, heterogeneity is built-in through the several random parameters.<sup>12</sup> People are connected in a non-evolving, exogenously determined, single-component social network (i.e., the network is static, it is constant across cells, and all pairs of people can reach each other in a finite number of “degrees of separation”).<sup>13</sup> We use a social network structure generated using a stochastic social network graph algorithm suggested by Newman and Park (2003) and described in the appendix.<sup>14</sup>

### ***Treatments***

Table 1 lists the manipulated parameters and the levels that were used. The 20 people with highest (lowest) centrality were chosen as initiators in the “close” (“peripheral”) condition. The product-specific and the initiator position characteristics form a set of four factors that we manipulated in a 9 (decay)  $\times$  11 (virality)  $\times$  2 (initiator position)  $\times$  4 (consumption threshold) full factorial design.<sup>15</sup> This gives 792 cells.

[INSERT TABLE 1 ABOUT HERE]

### ***Data and Statistical Analysis***

For each simulation run, we tracked the complete time series of our main dependent variables:  $Talk_t$ , and  $Consumption_t$ .  $Talk_t$  tracks the product’s conversation share as the proportion of the population who transmits enthusiasm toward the product in period  $t$  (i.e., an instantaneous measure of the amount of “buzz”).  $Consumption_t$  tracks the proportion of the

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<sup>11</sup> Preliminary trials indicated that 200 periods were sufficient time for “equilibrium” steady-states of consumption and conversation shares. Results are robust to longer or slightly shorter amounts of time.

<sup>12</sup> The random shocks on decay and perceived recipient enthusiasm were drawn from independent normal distributions with mean = 0, standard deviation = .1. Sufficient heterogeneity on the [0,1] interval was induced.

<sup>13</sup> We use a single connected component and people cannot always reach everyone in a finite number of steps in real world networks, these connected parts can be seen as components or the larger whole.

<sup>14</sup> Since our aim is to run a realistic simulation of social interactions, it makes sense to use a mixing rule (network structure) that corresponds to how people are actually connected.

<sup>15</sup> Note that the levels for decay listed in Table 1 do not cover the full [0,1] parameter space for  $\delta$ . This was because above the maximum level of .2 conversations and consumption of the products were effectively “killed” by the decay. Thus, levels above .2 for decay did not generate any interesting or meaningful results since decay then dominated the process.

population who consumes the product in period  $t$  (i.e., epidemic size). We also tracked two other variables to examine how these factors impact enthusiasm reinforcement:  $Dose_t$  (the mean dose size transmitted in period  $t$ ) and  $Transition_{01}$  (the proportion of people who had talked about the outside good in period  $t - 1$  and who switched to talking about the product in period  $t$ ). Based on our previous discussion, we expect initiator position but not virality to affect  $Dose_t$ , and virality but not initiator position to affect  $Transition_{01}$  (since irrespective of enthusiasm higher virality can get people talking about the product).

Our simulation generated a panel dataset with 792 cross-sectional “products,” each with 200 time-ordered observations. A dynamic random effects regression with cell random effects was estimated (using maximum likelihood; Greene 2003). We regressed the dependent variables on first-order autoregressive lags of themselves, the consumption threshold (as a control variable), and main effects, two-way interactions, and three-way interactions for decay, virality, and initiator position (additional lags were checked but did not significantly improve fit; a more complex error structure, e.g., moving-average, was also checked but not needed). Since *Consumption* and *Talk* are proportions, we used a generalized linear model to properly model these outcomes as being conditionally beta-distributed (conditional on the regressors).<sup>16</sup>

## **RESULTS**

### ***Manipulation Checks***

Parameter estimates and standard errors for the regressions of  $Talk_t$  and  $Consumption_t$  on the experimental factors and control covariates are reported in Table 2. Results show that the

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<sup>16</sup> The proper distributional assumption—in this case beta, for proportions—reduces the chances that the parameter estimates are biased. See Smithson and Verkuilen (2006) for an overview of beta regression. The mean (location parameter) of the beta distribution is a function of a set of regressors, linked to this distribution parameter with a link function that transforms the linear function of regressors to  $[0, 1]$ . Following previous applications of beta regression, a logit link was used.

decay and virality parameters operate as intended. While decay hurts conversation share and consumption ( $ps < .001$ ; Figure 2), virality has a positive effect on both outcomes ( $ps < .05$ ; Figure 3). There were no main effects of initiator position ( $ps > .40$ ), but this was also expected, as position is exogenous to the contagion process (whereas decay and virality are parameters within the process model itself). Position does, however, play a role through interactions.

In addition, as expected, consumption threshold has a significant negative effect on consumption but not conversation share ( $p < .001$ ). Also, the first-order autoregressive lags of the dependent variables are positive and significant ( $ps < .001$ ). There is also a significant positive effect of conversation share on consumption ( $p < .001$ ), indicating the importance of conversations in driving consumption under our framework. These models fit the data very well (pseudo- $R^2s > .95$ ).

[INSERT TABLE 2, FIGURE 2 AND FIGURE 3 ABOUT HERE]

### ***Interaction Effects on Conversation Share and Consumption: Mitigating Decay***

We next examine what might mitigate the negative effects of decay. Because decay drives down enthusiasm, it reduces the likelihood that the product is talked about, as well as the dose size, and as a result, kills social epidemics. What helps mitigate this negative effect of decay on conversations and epidemic size?

***Virality × Decay Interaction.*** First, we find a significant decay × virality interaction ( $ps < .001$ ; see Figure 4), indicating that decay has a weaker effect at higher levels of virality. This shows that virality not only works directly in opposition to decay to keep enthusiasm high (indicated by main effects of opposing signs), but also works indirectly, moderating the effect of decay on conversation and consumption. When virality is high, outcomes are less sensitive to

decay. Increasing virality by one increment (e.g., going from  $v = .1$  to  $.2$ ) reduces the strength of decay's effect on both conversation share and consumption by about 11%.

[INSERT FIGURE 4 ABOUT HERE]

Virality mitigates decay by increasing the transmission probability. This can be seen most clearly by looking at how decay, virality and position affect  $Transition_{01}$ . There is a positive effect of virality on transitions ( $p < .001$ ), as well as a decay  $\times$  virality interaction ( $p < .001$ ). Higher virality leads to a shift towards talking more about the product (and away from the outside good). Because transmissions are more likely and frequent, enthusiasm stays higher. Average dose sizes should also be higher (even though the primary role of virality is to increase the frequency, not amount, of enthusiasm reinforcement). A regression of  $Dose$  on the same regressors illustrates this: although there is no main effect of virality on average dose size ( $p = .27$ ), there is a significant interaction between decay and virality ( $p < .001$ ) such that higher virality diminishes the strength of the negative decay effect on dose size. Thus, more viral products are also less sensitive to decay reducing their dose sizes.

***Initiator Position  $\times$  Decay Interaction.*** Second, we find a significant decay  $\times$  initiator position interaction ( $p < .001$ ; see Figure 5), such that the negative effect of decay on consumption is weaker under close initiators. The effect of decay on consumption is reduced by 25% when close, rather than peripheral, initiators are used. Similar to what we found for virality, having close initiators makes the system less sensitive to decay's undesirable effect. However, whereas virality can also directly combat decay (i.e., through main effects of opposite signs), initiator position's contribution lies solely in moderating the decay effect.

[INSERT FIGURE 5 ABOUT HERE]

Close initiators mitigate decay by boosting average dose size. While there is no effect of position on conversation share or on people switching to talk about the product ( $Transition_{01}$ ,  $p_s > .18$ ), position directly affects enthusiasm by leading people to transmit larger doses ( $Dose$ ,  $p < .05$ ). In other words, the average dose size that gets transmitted is larger when close initiators are used. As discussed earlier, for close initiators the path lengths between them and non-initiators are, on average, relatively short (and therefore the cycles from initiators, to non-initiators, and back again are also relatively short). Consistent with our prior suggestion, under close initiators dose sizes stay higher despite decay because enthusiasm has to travel relatively shorter distances.

Importantly, this effect is related to distance and *not* degree. We ran the same model including a high degree initiator condition. Results indicate that high-degree initiators have a similar effect on dose size as peripheral initiators (contrast  $p > .05$ ), but a significantly weaker effect than close initiators (contrast  $p < .02$ ). Similar to peripheral initiators, high degree initiators do little to weaken the effect of decay on consumption. This is consistent with our argument about why closeness, rather than degree, is important for ongoing consumption. Since ongoing consumption requires enthusiasm reinforcement, high-degree initiators (whose closeness is not also very high) are not well-positioned to keep cycles of enthusiasm flowing over short distances, back-and-forth between them and as many non-initiators as possible.

Taken together, these interactions demonstrate that both product characteristics (virality) and initiator position in the social network (close initiators) can mitigate decay. While they do not cancel-out decay in our parameter space, they reduce its deleterious effect on consumption. The effects also highlight that different routes to enthusiasm reinforcement—either by changing the frequency of doses irrespective of size, or by affecting dose size—can be effective.

***Interchangeable or Super-Additive Effects?*** We find a significant three-way interaction between decay, virality, and initiator position ( $p < .001$ ). The effects indicate that when close initiators are used, the decay  $\times$  virality interaction is smaller. Similarly when virality is higher, the decay  $\times$  position interaction is smaller (such that at higher levels of virality close and peripheral initiators have similar effects). This indicates a reasonable degree of interchangeability—and *not* super-additivity—between having high-virality products versus relying on close initiators. Either virality or close initiators can be useful in helping to combat decay, but there does not appear to be any additional benefit to having *both* present. From a practical standpoint this is encouraging. Assuming that creating highly viral products and identifying and influencing close initiators are both costly marketing activities, managers need not invest in *both* to effectively combat decay and to enhance enthusiasm reinforcement.

### ***GENERAL DISCUSSION AND FUTURE RESEARCH***

This research has focused on understanding social epidemics of ongoing consumption. We base our contagion model on more psychologically realistic individual-level social behavior (WOM, social contagion, and product consumption), and use this model to examine drivers of epidemic size and persistence. Overall, we show how network and product characteristics can combine to encourage WOM, which in turn, reinforces product enthusiasm and bolsters ongoing consumption. Critically, these factors moderate each others' effects. Accordingly, it is important to consider the interplay of different potential epidemic drivers, rather than focusing on each in isolation. Under realistic conditions where products compete as potential conversation topics, and enthusiasm naturally decays over time, increasing a product's virality *or* relying on initiators who are close to many others can reinforce enthusiasm and help prevent abandonment. Virality

and initiator position work to mitigate the negative effects of decay and make the overall system less sensitive to natural declines in enthusiasm.

While these aspects both reinforce enthusiasm, they do so in different ways. Virality reinforces enthusiasm by increasing the likelihood that people will talk about the item. More viral things are talked about more, but each separate conversation's impact is not necessarily large. Hence, virality is more about increasing transmission likelihood (and helping to keep it high) than changing dose size. On the other hand, using close initiators reinforces enthusiasm by helping WOM spread over larger network distances more quickly, which maintains higher doses of enthusiasm. Importantly, it is closeness (based on distances) and not degree (based on direct connections) that matters; in our analysis high degree initiators were no better than peripheral initiators when it came to affecting dose sizes and the enthusiasm reinforcement process.

### ***Contributions, Limitations, and Suggestions for Future Research***

*A Rich and Psychologically Realistic Transmission Process.* This work has tried to provide a richer model of transmission behavior. While analytical and simulation-based work is praised for its simplicity, it is also often criticized for lacking realism, particularly regarding modeling assumptions. Such models allow researchers to examine how individual-level factors drive outcomes, but they are only as useful as their assumptions are realistic. We tried to base this work on more realistic assumptions that integrate findings from multiple perspectives.

Our model builds on extant models in a number of ways: (1) transmission probabilities vary based on aspects of the conversation dyad and the item being discussed, (2) dose size varies based on aspects of the conversation dyad, and (3) in the absence of conversation, enthusiasm decays over time. Taken together, these aspects generate a model of individual behavior that

more closely resembles how people *actually* think and behave when spreading WOM and consuming products.

Much more remains to be done, however, to empirically examine the transmission process. What do people like to talk about and how is this influenced by characteristics of their audience? What characteristics of things make them more likely to be shared and how does WOM and cultural transmission drive collective outcomes? While we have suggested a realistic transmission process based on existing work in this area, relatively little empirical work has actually examined such questions. A burgeoning body of research has convincingly shown that social networks, WOM, and social contagion influence consumer behavior and sales (e.g., Godes and Mayzlin 2008; Goldenberg et al. 2001, 2004; Iyengar, Van den Bulte, and Valente 2009; Stephen and Toubia 2009). Researchers must now shoulder the difficult task of moving beyond these first generation questions to investigate the psychological and sociological aspects underlying the transmission process.

Two areas that would be particularly worthwhile to develop are (1) conversation dynamics (i.e., the back-and-forth nature of conversation) and (2) bidirectional (or multidirectional) influence. To focus on mechanisms of enthusiasm reinforcement, we selected dyads in which one person was the transmitter, but in reality, both participants usually contribute information and influence to an ongoing conversation. Someone may start talking about a product, but listeners usually chime in, and competitors often get brought up as well (e.g., talking about a popular movie may remind people of other movies currently in theaters). Consequently, rather than being a directed shot of influence, both the original talker and the listener may be influenced in different ways by whatever information is brought up. Conversation events are not independent and more should be done to study both conversation dynamics (i.e., how topics

unfold) as well as the outcomes for enthusiasm, consumption, and social epidemics. This area is rich with possibilities for both empirical work and simulation modeling.

It would also be interesting to think more about the effect of conversation valence on dose size. We focused on situations where talking always led to positive doses of enthusiasm, but product discussions can also take a negative tone. While many perspectives have discussed negative word-of-mouth (e.g., Goldenberg et al. 2007), however, it is not obvious how valence should affect enthusiasm. While one could argue that positive WOM increases enthusiasm and negative WOM decreases it, even negative WOM may have positive effects by increasing product awareness and accessibility (Moore and Hutchinson 1983).

*Adoption versus Ongoing Consumption.* At a conceptual level, this work extends prior work on contagion to examine ongoing consumption. This distinction can also be seen empirically. We ran the same analyses using  $Adopt_t$  as the dependent variable, which tracks the proportion of people who have tried (consumed) the product at least once by the end of period  $t$ . While some effects were similar (i.e., decay still has a negative effect that can be mitigated by virality), there was no main effect of virality ( $p = .12$ ). Initiator position had a weak main effect (in favor of close initiators;  $p < .05$ ), but did not help mitigate decay (i.e., no interaction with decay;  $p = .13$ ). Thus reinforcement through larger dose sizes does not seem to be as important for adoption. This makes sense, as subsequent dose sizes matter little when only considering first-time adoption. These empirical distinctions further illustrate that past findings regarding drivers of new product adoption does not necessarily apply to ongoing consumption.<sup>17</sup>

Thinking about ongoing consumption also highlights what keeps people enthusiastic, and has important theoretical and practical implications. At the conceptual level, adoption only tells

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<sup>17</sup> Methodologically, analyzing only first-time adoption is essentially a type of data truncation problem. If such data are being used for statistical analysis this can lead to biased estimates, as we basically see here (e.g., effects being non-significant when they are in fact significant).

part of the story. Considering ongoing usage provides deeper insight into product lifespans and what leads products to be abandoned and eventually die out (cf. Berger and Heath 2008; Berger and Le Mens 2009). On a practical level, there are important implications for customer satisfaction, loyalty, and retention. While BMW wants consumers to adopt the brand (i.e., buy a BMW), they also want to keep them enthusiastic so that they will tell their friends about BMW and purchase a BMW next time they buy a car. The more enthusiastic people are when they first consume a product (when adoption is measured), the more likely they should be to tell others about the product, and to spread larger doses of enthusiasm when they talk. Hence, it seems useful to think beyond adoption to ongoing consumption.

More broadly, these distinctions suggest that diffusion research would benefit from considering a wider array of domains. Existing theorizing has been heavily based on the types of products the literature has tended to investigate. Adopting things that are expensive (e.g., hybrid corn) or risky (e.g. a new drug) requires being relatively certain of the benefits, and thus seeking information from peers to mitigate risk is seen as an important factor influencing whether social contagion will operate (Iyengar et al. 2009). For a host of other products, however, the question is less about risk mitigation and more about whether people are (and remain) enthusiastic. There is little cost involved in trying a website or listening to a new band, but given the multitude of options available, consumers must remember someone told them about a particular option and be enthusiastic enough to give it a try. Further, while the continued usage of hybrid corn or a new drug will depend mostly on product efficacy, in many other instances, continued product enthusiasm depends less on the product itself and more on conversations with others. Part of the utility of listening to a particular musician is talking about them with your friends, and in these instances, continued consumption will depend on the product continuing to be discussed. This is

particularly likely in cases where utility is driven by social factors rather than just functional aspects (e.g. using a website or being a fan). Consequently, factors that drive what people talk about, and enthusiasm with which people talk, likely drive ongoing consumption and the size and persistence of social epidemics.

*Decay and Enthusiasm Dynamics.* A major component of our conceptualization is that, in the absence of reinforcement through conversations, peoples' enthusiasm toward products naturally changes over time. We focused on situations where enthusiasm naturally decays, and found that this can have large effects, reducing overall consumption and leading otherwise good products to die out.

Incorporating enthusiasm shifts has important implications for estimating social contagion. Though social contagion is often discussed, few studies have actually been able to empirically demonstrate its occurrence (above and beyond the effects of marketing effort and other factors, Van den Bulte and Lillien 2001; see also Iyengar et al. 2009 and Stephen and Galak 2009). Though this is partially due to broader problems with identifying peer effects (e.g., Manski 1993), contagion may also end up seeming small (or nonexistent) due to a failure to incorporate enthusiasm decay. Our ancillary results indicate that decay can have strong negative effects on product adoption. This suggests that models which fail to include a parameter that picks up natural changes in enthusiasm might underestimate contagion effects.

Future research might also examine other potential enthusiasm trajectories. First, it would be useful to empirically examine exactly how enthusiasm changes over time, and what factors moderate this process. While enthusiasm often declines for many products (think of a child and their new toy), in other cases, enthusiasm may actually increase over time. Skiing, for example, is difficult at first, but people may enjoy it more the second or third time as they learn the

relevant skills. Similarly, changes may not always be monotonic and enthusiasm could initially appreciate and then later depreciate. Certain changes might be more typical for certain product categories. Second, it would be useful to incorporate different trajectories into contagion models to examine their broader implications for social epidemics. We found that decreasing enthusiasm had an important effect in killing off social epidemics, but different parameter values should lead to different outcomes. Thus future work might examine the parameter space more fully.

*Degree vs. Closeness.* These findings also speak to the growing debate about the importance of high-degree individuals in social epidemics (Goldenberg et al. 2009; Shapira et al. 2009; Watts and Dodds 2007). Our ancillary results indicate that only high-closeness, and not high-degree, initiators mitigate decay. Because enthusiasm naturally decays, it needs to be reinforced for consumption to persist. Social interactions can provide this needed boost, but the people spreading the word need to have many others in close reach. The key is not the number of friends someone has (i.e., lots of direct connections, high degree), but how many others that can be *quickly* reached through indirect connections (e.g., friends of friends). As one would expect, the higher a product's decay rate, the more important this distinction becomes. Hence, high-degree initiators' effects—which we found to be not significantly different from peripheral initiators'—are probably constrained to their “local” neighborhoods, whereas close initiators seem to be able to affect non-initiators located beyond their immediate vicinity.

While this finding suggests that closeness is a better characteristic to use to identify and target desirable early consumers, degree may still serve as a reasonable alternative. In real social networks, degree and closeness are typically positively correlated. So, when targeting high-degree people, there is a good chance that some of them will also have high closeness. Further, given that it is easier to ask people how many friends they have than determine network positions

(see Iyengar et al. 2009 for the utility of such self-report metrics), reported degree may be a reasonable alternative to use. Our finding, however, suggests that the observed efficacy of high-degree initiators in real epidemics might not be because they have many direct friends, but rather because they also happen to also have high closeness.

*Understanding What Makes Items Viral.* As noted briefly above, future research might also delve more deeply into why certain things are more viral than others. Our results indicated that virality plays an important role in both directly boosting transmission probability and moderating the negative effects of decay on consumption. But what makes certain products, content, and cultural tastes and practices are more viral? Just as certain fundamental templates may underlie quality ads (Goldenberg, Mazursky, and Solomon 1999), certain characteristics may make things more likely to be shared. We suggested that topicality, prominence, and surprisingness may all be linked to virality, but much more can be done. A great deal of work has discussed how item characteristics influence adoption by, for example, reducing the threshold for trial (see Rogers 2003 for a review), but there has been much less attention to what characteristics make things more likely to be transmitted or shared between individuals. In addition to being academically interesting, this is also obviously managerially relevant, as designing items that are more viral should increase revenue.

In particular, it is interesting to consider how consumer motives, and shared psychology, drive virality. Similar to notions of selection in the biological realm (Dawkins 1976), researchers have argued that which products, ideas, and behaviors succeed will depend on their fit with human memory, emotion, and motivation (Berger and Heath 2005; Heath et al 2001). This suggests that understanding motivations for WOM and cultural transmission should provide insight into aggregate success. One could argue that consumers share to entertain, express

identity, gain status, inform others, and/or deepen social bonds, and digging more deeply into these motives at the consumer level can help illuminate why certain item characteristics are linked to sharing.

It would also be interesting to examine drivers of social epidemics that are more passive. Diffusion can be active or passive. Active diffusion, such as WOM, involves people actively sharing information, recommendations, and advice, and sometimes involves the goal of influencing another's behavior. Passive diffusion, in contrast, describes case where a person does not actively try to change another's behavior, but the mere fact that their behavior is visible to another may lead to contagion. Merely seeing someone drive a particular car may increase purchase likelihood (Zajonc 1968), and research on behavioral mimicry finds that people mimic the actions of others even without awareness (Bargh and Chartrand 1999). While virality should play less of a role in such instances, other product characteristics, such as observability (Rogers 2003), should make passive diffusion more likely. Consequently there may also be interesting interactions between these aspects and network structure.

*Overall vs. Realized Network Structure.* Although we focused on how product characteristics can influence epidemics through WOM, they may also impact the realized network structure. The overall network may have a particular structure, but given that people may talk about certain things with some of their ties and not others, the actual usable or *activated* structure may vary across items or product categories, even if the overall structure is the same. Asking everyone who they are friends with, for example, will draw a particular network structure, but the structure would look different if people were asked to list only friends that they talk about soccer with, or friends with whom they discuss movies. Consequently, the item being transmitted may activate ties in an important way. It would be interesting to examine how

product characteristics influence the ties over which things are shared, and how this influences epidemic size and persistence. Certain items may be more likely to diffuse broadly, for example, because they can be shared across ties that bridge different social groups.

### ***Conclusion***

In summary, social epidemics are complex phenomena. Individuals decide what to talk about based on aspects on human psychology and item characteristics, and these discussions are situated in a broader social network structure. Beginning to understand these multiply-determined outcomes requires not only digging deeply within disciplinary traditions, but also linking more effectively across them. Insights on memory, emotion, social influence, identity, social networks, and more must be brought together more cohesively before integrative progress is made. Only then will researchers and practitioners truly understand what drives social epidemics and creates contagious.

## **REFERENCES**

- Barabási, Albert-Laszlo and Reka Albert (1999), "Emergence of Scaling in Random Networks," *Science*, 286, 509-512.
- Bargh, John A. and Tanya L. Chartrand (1999), "The Chameleon Effect : The Perception-Behavior Link and Social Interaction," *Journal of Personality and Social Psychology*, 76 (6), 893-910.
- Bartholomew, D.J. (1976), "Continuous Time Diffusion Models with Random Duration of Interest," *Journal of Mathematical Sociology*, 4 (2), 187-199.
- Bass, Frank M. (1969), "A New Product Growth Model for Consumer Durables," *Management Science*, 15 (5), 215-227.
- Berger, Jonah and Chip Heath (2005), "Idea Habitats: How the Prevalence of Environmental Cues Influences the Success of Ideas," *Cognitive Science*, 29 (2), 195-221.
- and ——— (2008), "Who Drives Divergence? Identity-Signaling, Outgroup Dissimilarity, and the Abandonment of Cultural Tastes," *Journal of Personality and Social Psychology*, 95 (3), 593-607.
- and Katherine Milkman (2009), "What Spreads? The Virality of News," working paper, University of Pennsylvania.
- and Gael Le Mens (2009), "Adoption Velocity and the Abandonment of Cultural Tastes," *Proceedings of the National Academy of Sciences*, forthcoming.
- Burt, Ronald S. (1992), *Structural Holes: The Social Structure of Competition*, Cambridge, MA: Harvard University Press.
- Castellano, Claudio, Santo Fortunato, and Vittorio Loreto (2007), "Statistical Physics of Social Dynamics," arXiv preprint , 0710.3256v.
- Coleman, James (1988), "Social Capital in the Creation of Human Capital," *American Journal of Sociology*, Supplement 94, S95-S120.
- , Elihu Katz, and Herbert Menzel (1957), "The Diffusion of an Innovation Among Physicians," *Sociometry*, 20 (4), 253-270.
- Dawkins, Richard (1979), *The Selfish Gene*, Oxford University Press.
- Dodds, Peter S. and Duncan J. Watts (2004), "Universal Behavior in a Generalized Model of Contagion," *Physical Review Letters*, 92 (21), 218701.
- Fast, Nathanael J., Chip Heath and George Wu (2009), "Common Ground and Cultural Prominence: How Conversation Strengthens Culture," forthcoming, *Psychological Science*.
- Feld, Scott L. (1982), "Social Structural Determinants of Similarity Among Associates," *American Sociological Review*, 47 (6), 797-801.
- Frenzen, Jonathan and Kent Nakamoto (1993), "Structure, Cooperation and the Flow of Market Information," *Journal of Consumer Research*, 20 (December), 360-375.
- Godes, David and Dina Mayzlin (2004), "Using Online Conversations to Study Word-of-Mouth Communication," *Marketing Science*, 23 (4), 545-560.

- and ——— (2008), “Firm-Created Word-of-Mouth Communication: A Field-Based Quasi-Experiment,” forthcoming, *Marketing Science*.
- Goldenberg, Jacob, Barak Libai, Sarit Moldovan and Eitan Muller (2007), “The NPV of Bad News,” *International Journal of Research in Marketing*, 24 (3), 186-200.
- , ———, and Eitan Muller (2001), “Talk of the Network: A Complex Systems Look at the Underlying Process of Word-of-Mouth,” *Marketing Letters*, 12 (3), 211-223.
- , ———, and ——— (2004), “Complex, Yet Simple: Cellular Automata as an Enabling Technology in Marketing Strategy Research.” In Christine Moorman and Donald R. Lehmann (eds), *Assessing Marketing Strategy Performance*, Cambridge, MA: Marketing Science Institute.
- , Sangman Han, Donald R. Lehmann, and Jae Weon Hong (2009), “The Role of Hubs in the Adoption Process,” *Journal of Marketing*, 73 (2), 1-13.
- , David Mazursky, and Sorin Solomon (1999), “Toward Identifying the Inventive Templates of New Products: A Channeled Ideation Approach,” *Journal of Marketing*, 36 (2), 200-210.
- Gatignon, Hubert and Thomas S. Robertson (1985), “A Propositional Inventory for New Diffusion Research,” *Journal of Consumer Research*, 11 (4), 849-867.
- Greene, William H. (2003), *Econometric Analysis*, 5<sup>th</sup> ed., New York: Prentice Hall.
- Hayes, Edward B. and Duane J. Gubler (2006), “West Nile Virus: Epidemiology and Clinical Features of an Emerging Epidemic in the United States,” *Annual Review of Medicine*, 57, 181-184.
- Heath, Chip, Chris Bell and Emily Sternberg (2001), “Emotional Selection in Memes: The Case of Urban Legends,” *Journal of Personality and Social Psychology*, 81 (6), 1028-1041.
- Hirschman, Elizabeth C. (1980), “Innovativeness, Novelty Seeking, and Consumer Creativity,” *Journal of Consumer Research*, 7 (3), 283-295.
- Iyengar, Raghuram, Thomas Valente, and Christophe Van den Bulte (2008), “Opinion Leadership and Social Contagion in New Product Diffusion,” working paper, University of Pennsylvania.
- Keeling, M.J. and K.T. Eames (2005), “Networks and Epidemic Models,” *Journal of the Royal Society, Interface*, 2 (4), 295-307.
- Klovdahl, A.S. (1985), “Social Networks and the Spread of Infectious Diseases: The AIDS Example,” *Social Science and Medicine*, 21 (11), 1203-1216.
- Leskovec, Jure, Adamic, Lada A., Huberman, Bernardo, A. (2007), “The Dynamics of Viral Marketing,” *ACM TWeb* 1(1), 2007.
- Libai, Barak, Eitan Muller, and Renana Peres (2005), “The Role of Seeding in Multi-Market Entry,” *International Journal of Research in Marketing*, 22 (4), 373-393.
- Mahajan, Vijay, Eitan Muller, and Frank M. Bass (1990), “New Product Diffusion Models in Marketing: A Review and Directions for Research,” *Journal of Marketing*, 54 (1), 1-26.

- Manski, Charles F. (1993), "Identification of Endogenous Social Effects: The Reflection Problem," *Review of Economic Studies*, 60 (3), 531-542.
- Mark, Noah (1998), "Birds of a Feather Sing Together," *Social Forces*, 77 (2), 453-485.
- Moore, Danny L. and J. Wesley Hutchinson (1983), "The Effects of Ad Affect on Advertising Effectiveness," R. P. Bagozzi and A. M. Tybout (eds.), *Advances in Consumer Research*, 10, 526-531.
- Newman, Mark E.J. and Juyong Park (2003), "Why Social Networks are Different from Other Types of Networks," *Physical Review E*, 68, 036122.
- de Nooy, Wouter, Andrej Mrvar, Vladimir Batagelj (2005), *Exploratory Social Network Analysis with Pajek*, Cambridge, UK: Cambridge University Press.
- Norenzayana, Ara, Scott Atranb, Jason Faulknera and Mark Schallera (2006), "Memory and Mystery: The Cultural Selection of Minimally Counterintuitive Narratives," *Cognitive Science*, 30, 531-553.
- Rogers, Everett M. (2003), *Diffusion of Innovations*, New York, NY: Free Press.
- and F. Floyd Shoemaker (1971), *Communication of Innovations*, New York, NY: Free Press.
- Rubin, David (1995). *Memory in Oral Traditions: The Cognitive Psychology of Epic, Ballads, and Counting-Out Rhymes*. Oxford, England: Oxford University Press.
- Salganik, Matthew J., Peter S. Dodds, and Duncan J. Watts (2006), "Experiment Study of Inequality and Unpredictability in an Artificial Cultural Market," *Science*, 311 (5762), 854-856.
- Schaller, Mark, Lucian Gideon Conway III and Tracy L. Tanchuk (2002), "Selective Pressures on the Once and Future Contents of Ethnic Stereotypes: Effects of the Communicability of Traits," *Journal of Personality and Social Psychology*, 82 (6), 861-877.
- Shapira, Danny, Jacob Goldenberg and Oded Lowengart (2009), "The Effect of Social Hubs on the Diffusion of an Innovation," working paper, Hebrew University of Jerusalem.
- Smithson, Michael and Jay Verkuilen (2006), "A Better Lemon Squeezer? Maximum-Likelihood Regression With Beta-Distributed Dependent Variables," *Psychological Methods*, 11 (1), 54-71.
- Stephen, Andrew T. and Donald R. Lehmann (2009), "Why Do People Transmit Word-of-Mouth? The Effects of Recipient and Relationship Characteristics on Transmission Behaviors," working paper, Columbia University.
- and Jeff Galak (2009), "The Returns to Media Publicity," working paper, Columbia University.
- and Olivier Toubia (2009), "Deriving Value from Social Commerce Networks," *Journal of Marketing Research*, forthcoming.
- Toubia, Olivier, Jacob Goldenberg and Rosanna Garcia (2008), "A New Approach to Modeling the Adoption of New Products: Aggregated Diffusion Models," working paper, Columbia University.

- Valente, Thomas W. (2005), "Network Models and Methods for Studying the Diffusion of Innovations." In Peter J. Carrington, John Scott and Stanley Wasserman (eds), *Models and Methods in Social Network Analysis*, Cambridge, UK: Cambridge University Press.
- Van den Bulte, Christophe and Gary L. Lilien (2001), "Medical Innovation Revisited: Social Contagion versus Marketing Effort," *American Journal of Sociology*, 106 (5), 1409-1435.
- Watts, Duncan J. (2002), "A Simple Model of Global Cascades on Random Networks," *Proceedings of the National Academy of Sciences of the United States of America*, 99 (9), 5766-5771.
- and Peter Sheridan Dodds (2007), "Influentials, Networks, and Public Opinion Formation," *Journal of Consumer Research*, 34 (4), 441-458.
- and Steven H. Strogatz (1998), "Collective Dynamics of Small-World Networks," *Nature*, 393 (6684), 440-42.
- Weiss, George H. and Menachem Dishon (1971), "On the Asymptotic Behavior of the Stochastic and Deterministic Models of an Epidemic," *Mathematical Biosciences*, 11 (3-4), 261-265.
- Wu, Fang and Bernardo A. Huberman (2007), "Novelty and Collective Attention," *Proceedings of the National Academy of Sciences of the United States of America*, 104 (45), 17599-17601.
- Zajonc, Robert B. (1968), "Attitudinal Effects of Mere Exposure," *Journal of Personality and Social Psychology*, 9 (2), 1-27.

**TABLE 1**  
**PARAMETERS AND EXPERIMENTAL FACTORS**

<b>Parameter</b>	<b>Levels Used in Experiment</b>	<b>Definition</b>
<b>Product Characteristics</b>		
Decay [0,1]	0; .025; .05; .075; .1; .125; .15; .175; .2	The per-period rate at which a person's enthusiasm for a product decays between periods. Related to how interesting and top-of-mind a product is: more interesting and top-of-mind products have smaller decay.
Virality [0,1]	0; .1; .2; .3; .4; .5; .6; .7; .8; .9; 1	The probability that a person talks to a neighbor about a product irrespective of their and their neighbor's enthusiasm for a product. Related to how "viral," "buzz-worthy," and topical a product is: more viral or more topical product have a higher virality and are more likely to be talked about irrespective of individuals' enthusiasm.
Consumption threshold [0,1]	.2; .4; .6; .8	The minimum level of enthusiasm a person must have to consume the product that period. Products with a higher threshold require more enthusiasm to consume.
<b>Network Characteristics</b>		
Initiator position (closeness centrality)	Close (high closeness); Peripheral (low closeness)	Closeness centrality is a network centrality measure that is related to the length of the paths that connect a person to other people in the social network, and is a function of the average path length between a person and all other reachable people in the network. Close (high closeness) people have a relatively short average path length between them and others. Peripheral (low closeness) people have a relatively long average path length between them and others.

**TABLE 2**  
**PARAMETER ESTIMATES**

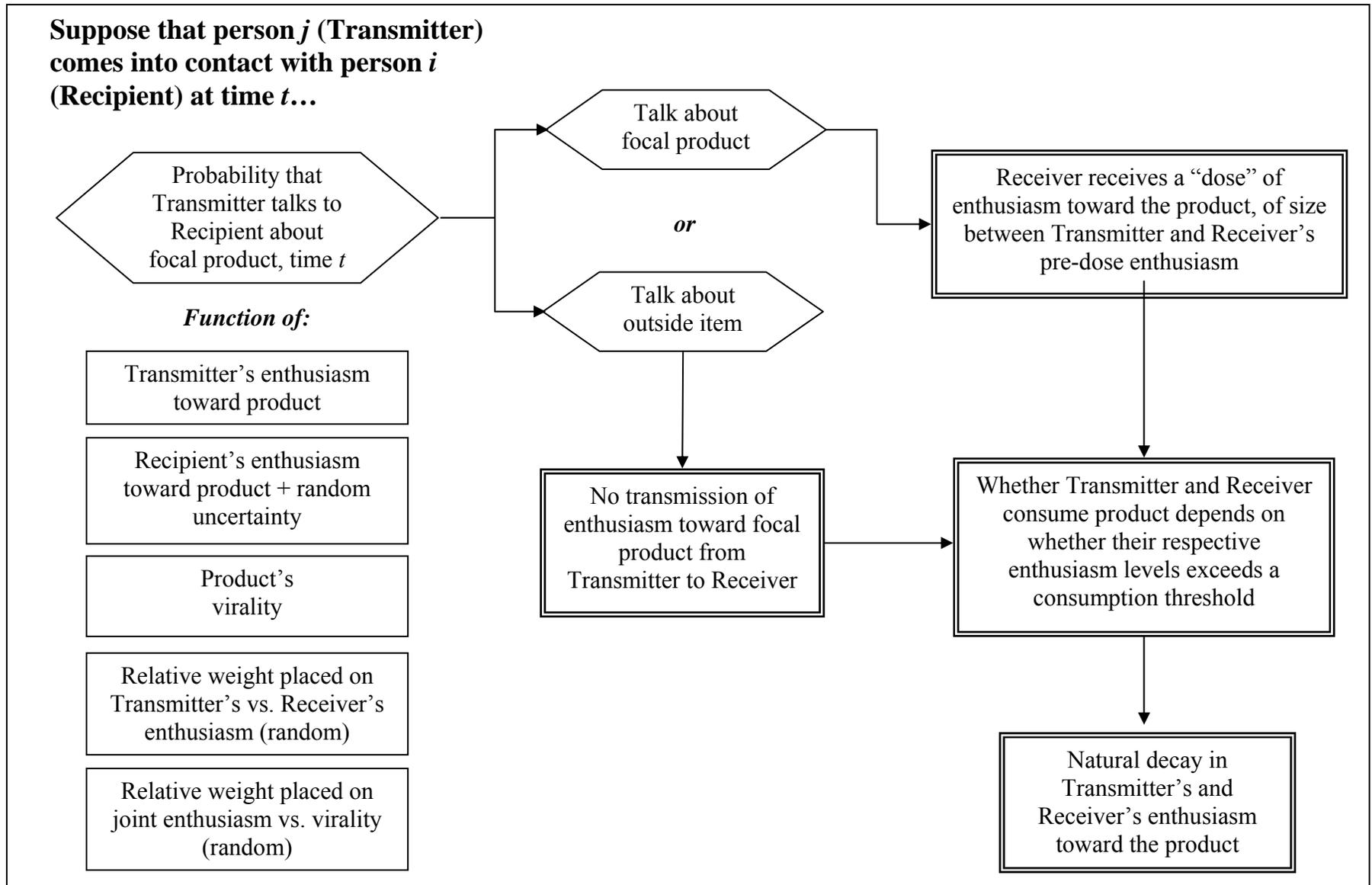
	Parameter Estimates (Standard Errors)			
	<i>Consumption<sub>t</sub></i>		<i>Talk<sub>t</sub></i>	
Intercept	-1.85	(.138)*	-2.45	(.131)*
<i>Consumption<sub>t-1</sub></i>	2.62	(.031)*	n/a	
<i>Talk<sub>t-1</sub></i>	10.26	(.072)*	4.74	(.003)*
Consumption threshold	-1.88	(.115)*	.04	(.109) <sup>ns</sup>
Decay	-25.24	(1.108)*	-9.42	(1.000)*
Virality	-4.72	(.214)*	.59	(.201)*
Initiator position (base = peripheral)	-.17	(.177) <sup>ns</sup>	.14	(.168) <sup>ns</sup>
Decay × Virality	30.40	(1.832)*	10.55	(1.689)*
Decay × Position	7.23	(1.544)*	.67	(1.414) <sup>ns</sup>
Virality × Position	.20	(.298) <sup>ns</sup>	-.17	(.284) <sup>ns</sup>
Decay × Virality × Position	-8.66	(2.568)*	-1.08	(2.389) <sup>ns</sup>

\*  $p < .01$ , <sup>ns</sup> not significant. Model fit:  $-2 \text{ Log Likelihood} = 481,197$ ,  $\chi^2 = 315,216$ .

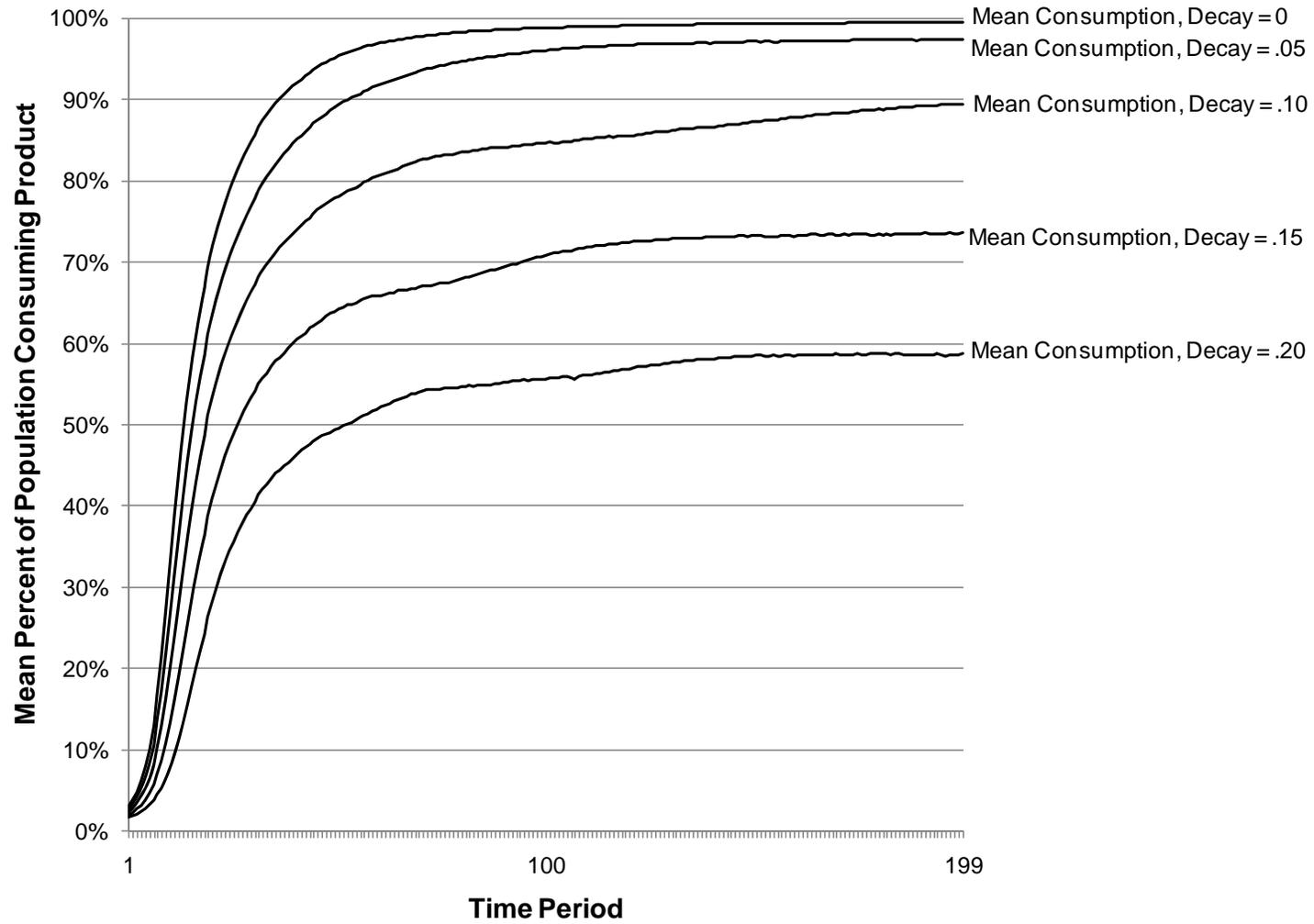
Estimates of variances of random effects: *Consumption* .51 (.027), *Talk* .47 (.024).

Estimates of beta distribution scale parameters: *Consumption* 62.03 (.225), *Talk* 242.65 (.870).

**FIGURE 1**  
**OVERVIEW OF INDIVIDUAL-LEVEL PROCESS MODEL**



**FIGURE 2**  
**CONSUMPTION UNDER DIFFERENT LEVELS OF DECAY**



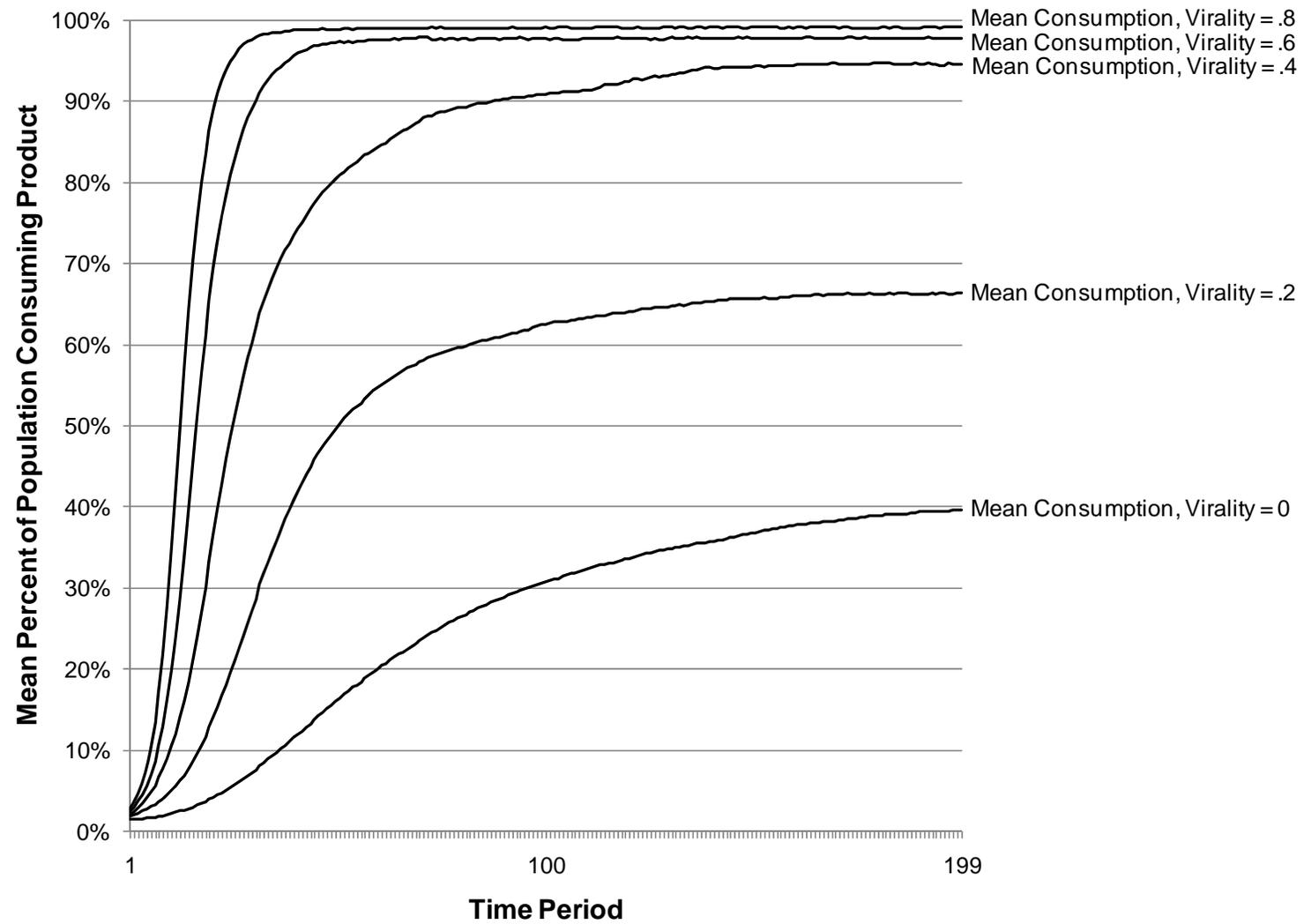
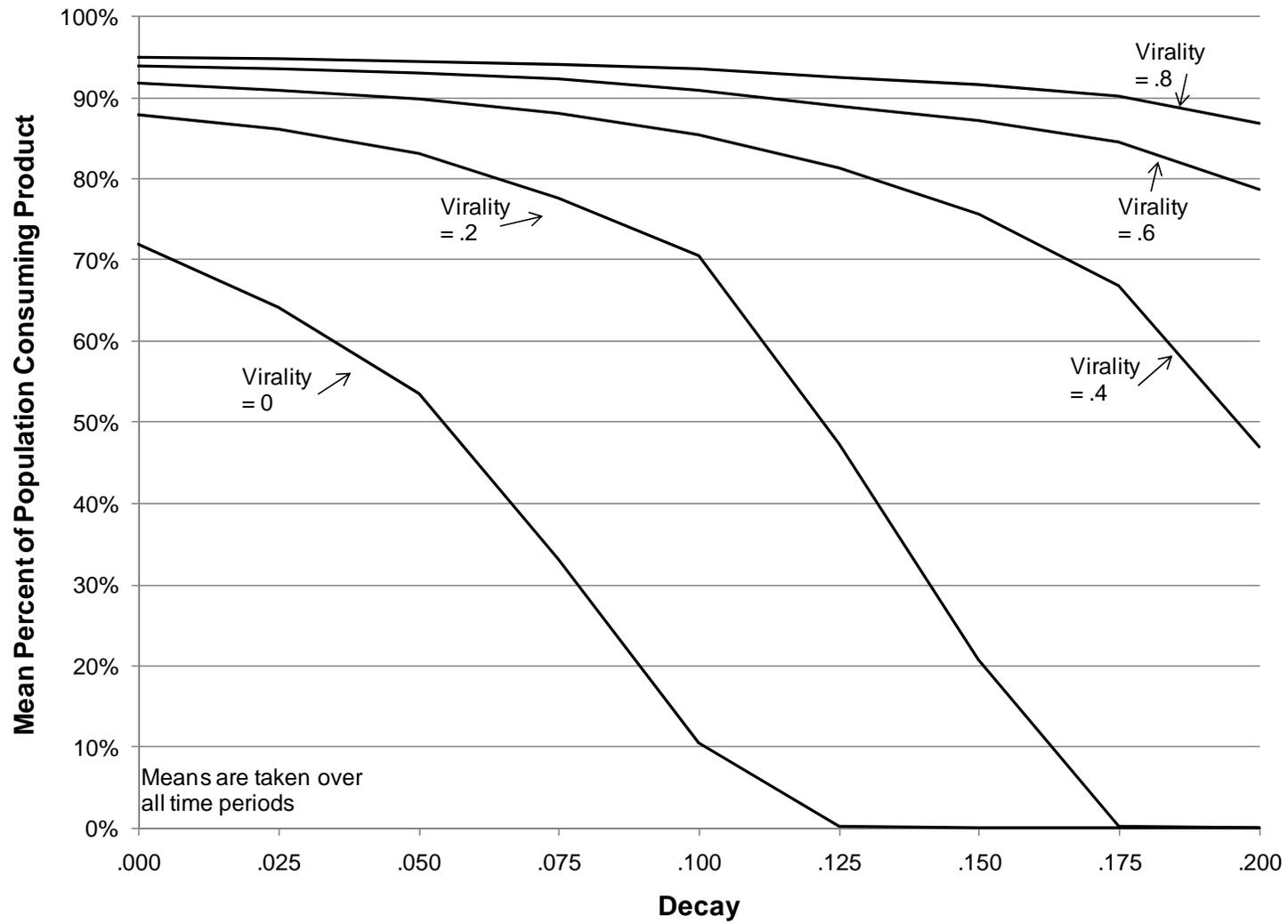
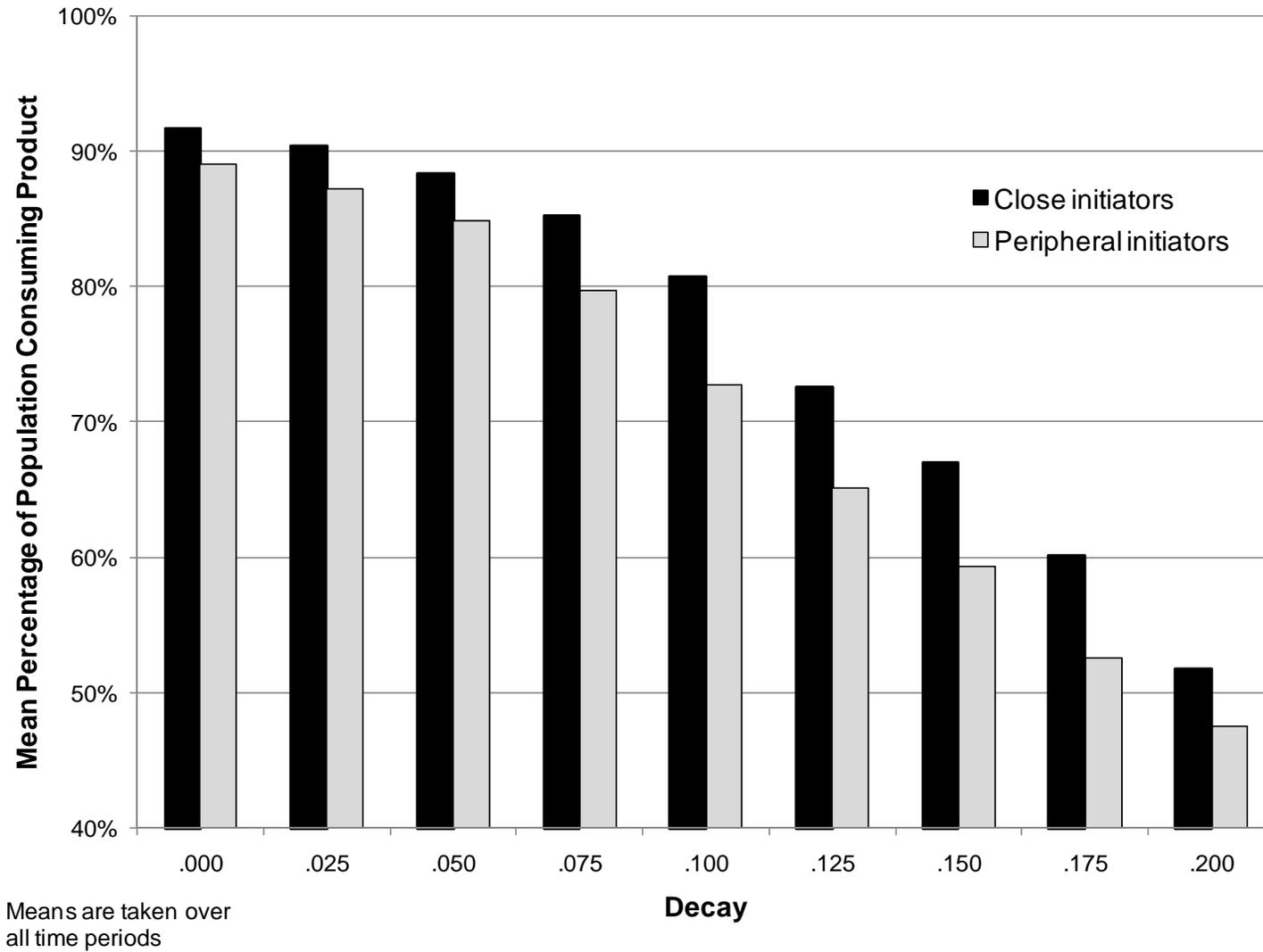
**FIGURE 3****CONSUMPTION UNDER DIFFERENT LEVELS OF VIRALITY**

FIGURE 4

DECAY  $\times$  VIRALITY INTERACTION FOR CONSUMPTION

**FIGURE 5*****DECAY × INITIATOR POSITION INTERACTION FOR CONSUMPTION***

## *APPENDIX*

### *SOCIAL NETWORK GENERATION ALGORITHM*

The following network generation algorithm is based on a procedure described in Newman and Park (2003). We generated a realistic social network structure where people belong to “communities” and can only be connected to a person if they are in at least one of the same communities. Since social networks are different from many other types of networks, random graphs are not ideal.

Our population has  $N = 1,000$  people. The underlying community affiliation structure has  $N_C = 25$  communities that people can belong to, with each having a capacity of  $M_c$  people (for  $c = 1, \dots, N_C$ ), with  $M_c \sim \text{Poisson}(N/N_C)$ . Person  $i$  is a member of  $h_i$  communities, where  $h_i \sim \text{Poisson}(2)$ . Specifically, if two people are members of the same community then they have a 2% probability of being connected in the social network (this probability of 2% ensures that the generated network is sparse and has the defining properties of real social networks: a mean geodesic distance of approximately 4, a mean clustering coefficient of approximately .03, a skewed and approximately power-law degree distribution).