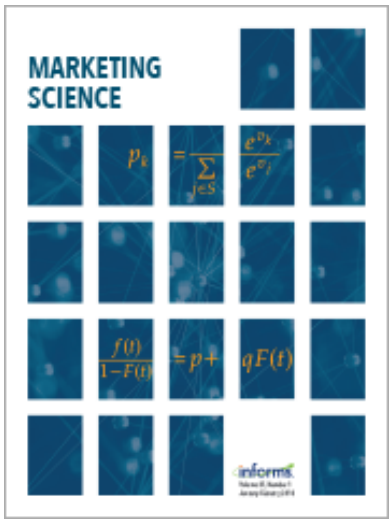

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New Product Diffusion with Influentials and Imitators

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We model the diffusion of innovations in markets with two segments: *influentials* who are more in touch with new developments and who affect another segment of *imitators* whose own adoptions do not affect the influentials. This two-segment structure with asymmetric influence is consistent with several theories in sociology and diffusion research, as well as many "viral" or "network" marketing strategies. We have four main results. (1) Diffusion in a mixture of influentials and imitators can exhibit a dip or "chasm" between the early and later parts of the diffusion curve. (2) The proportion of adoptions stemming from influentials need not decrease monotonically, but may first decrease and then increase. (3) Erroneously specifying a mixed-influence model to a mixture process where influentials act independently from each other can generate systematic changes in the parameter values reported in earlier research. (4) Empirical analysis of 33 different data series indicates that the two-segment model fits better than the standard mixed-influence, the Gamma/Shifted Gompertz, and the Weibull-Gamma models, especially in cases where a two-segment structure is likely to exist. Also, the two-segment model fits about as well as the Karmeshu-Goswami mixed-influence model, in which the coefficients of innovation and imitation vary across potential adopters in a continuous fashion.

Key words: asymmetric influence; diffusion of innovations; innovation; market segments; social contagion; social structure
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1. Introduction

Under pressure to increase their return on marketing investment through more astute targeting of resources, marketers are rediscovering the importance of social contagion. Recent "viral" and "network" marketing strategies often share two key assumptions: (1) some customers are more in touch with new developments than others, and (2) some (often, the same) customers' adoptions and opinions have a disproportionate influence on others' adoptions (e.g., Gladwell 2000, Moore 1995, Rosen 2000, Slywotzky and Shapiro 1993). Targeting those influential prospects who are more in touch with new developments and converting them into customers, the logic goes, allows marketers to benefit from a social multiplier effect on their marketing efforts. The two assumptions are quite reasonable, as they are consistent with several theories and a large body of empirical research (e.g., Katz and Lazarsfeld 1955, Rogers 2003, Weimann 1994), and the social multiplier logic cannot be faulted either (e.g., Case et al. 1993, Valente et al. 2003). However, marketing science provides little or no additional theoretical or descriptive insight into how new products diffuse in such markets. The reason is that the great majority of marketing diffusion models assume homogeneity rather than heterogeneity in the tendency to be in tune with new developments and the tendency to influence (or be influenced by) others. We address this gap between theory and emerging practice on the one hand, and marketing diffusion models on the other. Specifically, we model the aggregate-level diffusion path of a new product when the set of ultimate adopters is not homogeneous, but consists of two segments: *influentials* who are more in touch with new developments and who affect another segment of *imitators* whose own adoptions do not affect the influentials. We allow for the presence or absence of contagion among influentials and among imitators.

Many diffusion models incorporate the dual drivers of independent decision making affected by being in touch with new developments, and of imitation driven by others' prior adoptions, but they do so under the assumption that all potential adopters are ex ante equally affected by both factors. Taga and Isii (1959) in statistics; Mansfield (1961), Pyatt (1964), and Williams (1972) in economics; Coleman (1964) in sociology; and Bass (1969) and Massy et al. (1970) in marketing, all advanced a model specifying the rate at which actors who have not adopted yet do so at time t as $h(t) = p + qF(t)$, where $F(t)$ is the proportion of ultimate adopters that have already adopted, parameter q captures social contagion, and parameter p captures the time-invariant tendency to adopt

early affected by consumer characteristics, the innovation's appeal, and efforts of change agents.[1] Because the proportion that adopts at time t can be written as $f(t) = dF(t)/dt = h(t)[1 - F(t)]$, one obtains:

$$f(t) = dF(t)/dt = [p + qF(t)][1 - F(t)]. \quad (1)$$

The solution of this differential equation can be written as:

$$F(t) = [1 - e^{-g-(p+q)t}]/[1 + (q/p)e^{-g-(p+q)t}], \quad (2)$$

where g acts as a location parameter fixing the curve on the time axis (e.g., Mansfield 1961). When $t = 0$ corresponds to the actual launch time such that $F(0) = 0$, then $g = 0$ and Equation (2) reduces to the solution popular in marketing.

The rate is influenced by *both* the intrinsic tendency to adopt (p) and social contagion (q) at all times except at $t = 0$ when $qF(0) = 0$. To reflect this dual influence, Mahajan and Peterson (1985) refer to the model as the mixed-influence model. Because the rate contains no contagion pressure at $t = 0$, those adopting at that time are sometimes referred to as innovators and contrasted against all others adopting later, who are called imitators (e.g., Bass 1969). However, this terminology can only be used ex post, and the model does *not* represent a diffusion process in an ex ante mixture of two segments, the first adopting independently at rate p and the second adopting because of social contagion at rate $qF(t)$ (Bemmaor 1994, Jeuland 1981, Lekvall and Wahlbin 1973, Manfredi et al. 1998, Steffens and Murthy 1992, Tanny and Derzko 1988).

The objective of this study is to mathematically formalize prior theoretical arguments and research findings on social structure and diffusion, and to use this formalization to generate more refined theoretical insights on new product diffusion in a population of influentials and imitators. This is important because marketing practitioners increasingly deploy strategies assuming such a market structure, and because marketing researchers increasingly incorporate social structure into their diffusion investigations (e.g., Bronnenberg and Mela 2004, Frenzen and Nakamoto 1993, Garber et al. 2004, Godes and Mayzlin 2004, Putsis et al. 1997, Van den Bulte and Lilien 2001).

Our results offer formalized insights into current substantive and methodological research questions. First, diffusion in a mixture of influentials and imitators can exhibit a dip between the early and later parts of the diffusion curve. In contrast to what Moore (1991) claims, our model shows that it need not always be necessary for firms to change their product to gain traction among later adopters and the adoption curve to swing up again.[2] Like Steffens and Murthy (1992) and Karmeshu and Goswami (2001), but unlike Goldenberg et al. (2002), we obtain this result from a closed-form solution, and unlike those prior analyses, we show that a dip can occur even when influentials act independently from each other. Second, the proportion of adoptions stemming from influentials need not decrease monotonically, but may first decrease and then increase. The management implication is that while it may make sense to shift the focus of one's marketing efforts from influentials to imitators shortly after launch, as shown by Mahajan and Muller (1998) using a two-period model, one may want to revert one's focus back to influentials later in the process. Third, erroneously specifying a mixed-influence model to a two-segment process can generate the systematic changes in the parameter values over time reported in several studies (e.g., Van den Bulte and Lilien 1997, Venkatesan et al. 2004). This analytical result is a specific formalization of Van den Bulte and Lilien's (1997) more general but qualitative argument that unaccounted heterogeneity in p or q can generate changes in these parameters' estimates as one extends the data window. Our result also complements Bemmaor and Lee's (2002) simulation analysis because we consider heterogeneity in a process with genuine contagion rather than in a Gamma/Shifted Gompertz process without contagion.

We also perform an empirical analysis and assess the descriptive performance of the two-segment model compared to that of the mixed-influence model and of three diffusion models incorporating heterogeneity in the form of a continuous rather than a discrete mixture. Given the difficulty of unambiguously identifying causal processes from aggregate diffusion data (Bemmaor 1994, Hernes 1976, Lekvall and Wahlbin 1973, Lilien et al. 1981, Van den Bulte and Stremersch 2004), the objective of this empirical analysis is not to conclusively demonstrate the validity of any model. Rather, it is to assess whether the differences between the discrete mixture and other models are sufficiently important to lead to differences in descriptive performance when applied to data of interest to marketing researchers. The two-segment model fits better than the mixed-influence, Gamma/Shifted Gompertz (Bemmaor 1994), and Weibull-Gamma models (Hardie et al. 1998, Massy et al. 1970, Narayanan 1992), especially in cases where

[1] Following the convention in marketing, we refer to the rate at which nonadopters turn into adopters as the hazard rate and denote it as $h(t)$, even though the models we discuss are deterministic rather than probabilistic.

[2] Changing the product might consist of augmenting the core product with complementary services and products to provide a "whole product," or consist of offering simpler and more user-friendly versions of the core product.

a two-segment structure is likely (or even known) to exist, and fits about as well as a recently advanced mixed-influence model where p and q vary across potential adopters in a continuous fashion (Karmeshu and Goswami 2001).

We proceed by first outlining our model setting, and within that context, discuss five theories and frameworks that suggest the existence of ex ante influentials and imitators. Next, we develop a macro-level model of innovation diffusion in such a setting. Subsequently, we discuss how this model relates to the familiar mixed-influence model and to prior work on two-segment models. Finally, we report on the descriptive performance of the influential-imitator model compared to that of the mixed-influence and continuous-mixture models.

2. Theories Motivating a Two-Segment Structure of Influentials and Imitators

The situation we model is the following. The set of eventual adopters has a constant size M and consists of two a priori different types of actors, influentials and imitators. We use the subscripts 1 and 2 to denote each type, and the subscript m to denote the entire mixture population of adopters. We use θ to denote the proportion of Type 1 actors in the population of eventual adopters ($0 \le \theta \le 1$), and $F(t)$ to denote the cumulative penetration. Finally, w denotes the relative importance that imitators attach to influentials' versus other imitators' behavior ($0 \le w \le 1$). Each type's adoption behavior is then captured by the following hazard functions:

$$h_1(t) = p_1 + q_1 F_1(t); \tag{3}$$

$$h_2(t) = p_2 + q_2[wF_1(t) + (1-w)F_2(t)]. \tag{4}$$

Note the asymmetry in the influence process: Type 1 may influence Type 2, but the reverse is not true. Because, ex ante, anyone of Type 1 may influence anyone of Type 2, we label the former *influentials* and the latter *imitators*. When $p_2 = 0$, contagion from influentials to imitators ($wq_2 > 0$) is critical for the diffusion process among the latter to get started. Obviously, when $\theta = 1$ or $\theta = 0$, everyone falls into a single segment and the situation reduces to the mixed-influence model (MIM). When $0 < \theta < 1$ but $w = 0$, the model reduces to two disconnected MIMs and, with further restrictions, to a model with two disconnected logistic or exponential functions (e.g., Moe and Fader 2001, Perrin 1994). Also, when imitators put equal weight on all prior adoptions regardless of origin, then we have $h_2(t) = p_2 + q_2 F_m(t)$, which implies $w = \theta$ (see §3).

The distinction between influentials and imitators is based on what drives their adoption behavior, not on whether they adopt early or late. Hence, the distinction is different from that of innovators versus imitators in Bass (1969) and innovators versus early adopters versus early majority versus late majority versus laggards in Rogers (2003). Conceptually, causal drivers and time of adoption need not map one-to-one. Empirically, while those adopting early may act independently of others, and those adopting late may be subject to contagion, this is not always so: Many early adoptions may be driven by contagion, and the bulk of the late adoptions may stem from people not subject to social contagion (e.g., Becker 1970, Coleman et al. 1966).

Several theories and conceptual models suggest such a two-segment structure, although there is some disagreement on whether q_1 and p_2 may be larger than zero. We first describe sociological arguments focusing on social character, social status, and social norms. We then turn to the two-step flow hypothesis, which focuses on interest in new developments, and finally to the chasm idea, which focuses on enthusiasm for innovations versus risk aversion.

2.1. Social Character

In his classic treatise on the changing nature of modern society, Riesman (1950) distinguished three types of social character: autonomous, inner directed, and other directed. The first two have in common the presence of clear-cut internalized goals, but differ as to whether these are consciously chosen (autonomous) or inculcated during youth by elders (inner directed). Other-directed actors, in contrast, use their peers as their source of direction. The typology is in essence about conformity stemming from the need for approval and direction from others. Riesman worked on a broad social and cultural canvas and his typology is best used to refer to patterns of behavior found in a variety of specific contexts rather than to types of persons or personalities. However, his concepts have direct relevance for consumer behavior (e.g., Riesman 1950, Schor 1998). Some actors in some situations will exhibit autonomous or inner-directed adoption behavior independent from their peers (hence $q_1 = 0$), while others will exhibit other-directed behavior driven by social contagion from peers. Riesman did not narrowly specify who these peers are, and allowed them to be all of society (therefore $w = \theta$ being possible).

2.2. Status Competition and Maintenance

People buy and use products not only for functional purposes, but also to construct a social identity, and to confirm the existence and support the reproduction of social status differences (Bourdieu 1984). A long-held idea in diffusion theory is that people seek to emulate the consumption behavior of their superiors and aspiration groups (e.g., Simmel 1971), and also quickly pick up innovations adopted by others of similar status if they fear that such adoptions might undo the present status ordering (Burt 1987). In short, actors

tend to imitate the adoptions of those of higher and similar social status.

Assuming one can divide the population into a high-status and a low-status group, status considerations suggest that both groups may exhibit contagion. Higher-status actors may imitate each other out of fear of falling behind ($q_1 \geq 0$), and lower-status actors imitate to catch up. Whose adoptions the imitators act upon is not clear a priori. If they care only about adoptions by the high-status influentials, then $w \to 1$. However, most authors follow Simmel and posit a finer-grained hierarchy with multiple strata (approximated imperfectly by a dichotomy) and a cascading pattern where all prior adoptions contribute equally to social contagion ($w = \theta$). Finally, to the extent that status is maintained by adhering to social norms enforced among one's direct peers of similar position, imitators should care mostly about fellow imitators ($w \to 0$).

2.3. Middle-Status Conformity

Like theories of status competition and maintenance, middle-status conformity theory is about one's proper place in society. The main claim is that the relationship between status and conformity to norms—and hence susceptibility to social contagion—is an inverted U (e.g., Homans 1961, Philips and Zuckerman 2001). Because high-status actors feel confident in their social acceptance, they feel comfortable in deviating from conventional behavior and adopt appealing innovations independently from others. Low-status actors feel free to deviate from accepted practice and adopt innovations independently as well, because they feel that this cannot hurt their already low status. Middle-status actors, in contrast, feel insecure and strive to demonstrate their legitimacy by engaging in new practices only after they have been socially validated. Therefore, middle-status conformity theory is consistent with the presence of two kinds of actors, one adopting as a function of the innovation's appeal irrespective of others' actions ($q_1 = 0$), and one adopting as a function of the legitimation stemming from prior adoptions.

The theory does not specify whose adoptions are being imitated (w). Adoptions by high-status actors might legitimate the innovation in the eyes of the middle-status actors disproportionately, in which case the relation of w to θ is unclear because the latter captures both high and low status. Conversely, imitators may care only about social acceptability among their middle-status peers, and hence care only about the latter's adoptions ($w = 0$). Finally, applications of neo-institutional theory to innovation adoption tend to posit that the legitimacy of an innovation is affected by the overall penetration rate ($w = \theta$).

Note that higher status is often associated with higher economic resources, and hence a higher ability to adopt innovations. This leads to the interesting prediction that *only the adoptions at an intermediate stage of the overall diffusion process* (*made by middle-status actors*) *exhibit contagion* (e.g., Cancian 1979), because the earliest adoptions will come from high-status actors and the latest from low-status actors, none of which are subject to contagion.

2.4. Two-Step Flow

The two-step flow hypothesis, originally proposed to explain unexpectedly weak mass media effects in presidential elections, posits that "ideas often flow *from* radio and print *to* the opinion leaders and *from* them to the less active sections of the population" (Lazarsfeld et al. 1944, p. 151, emphasis in original). Therefore, in its original and starkest version, the two-step flow hypothesis posits two groups, one being affected only by mass media ($q_1 = 0$) and the other being affected only by social contagion ($p_2 = 0$). What distinguishes the two groups is the level of interest in the subject matter and alertness to new developments rather than exposure to mass communications (Lazarsfeld et al. 1944). Later studies in marketing have corroborated a strong relationship between opinion leadership and product interest and involvement (e.g., Coulter et al. 2002, Myers and Robertson 1972). Note that the two-step flow hypothesis does not require that an opinion leader in one sphere (politics, fashion, computer games, etc.) also be a leader in another sphere, and several studies indeed document only moderate to little overlap in leadership across product categories (e.g., Katz and Lazarsfeld 1955, Merton 1949, Myers and Robertson 1972, Silk 1966). Therefore, the relative size of the segments (θ) may vary across innovations. While early studies focused on information flows from opinion leaders to less active members of the population, subsequent research has documented extensive information exchange among opinion leaders (e.g., Coulter et al. 2002, Katz and Lazarsfeld 1955), consistent with $q_1 > 0$.

The two-step flow hypothesis emphasizes the flow of information. The contagion mechanism is one of information transfer increasing awareness of the product's existence and decreasing its perceived risk, not of normative legitimation or status competition. Of the five theories we consider, this is perhaps the most flexible. For low-risk innovations, for instance, the fraction of imitators in need of guidance can be quite small, and θ quite large. Who is being imitated is not clearly specified, and w may range from zero to one. The original two-step flow idea emphasizes that mass media influence on the less-active segment operates through opinion leaders who are the only ones to take an active interest in information available in the media. It does so without constraining the social influence exerted on the less-active segment to come only from opinion leaders, and allows for a cascading or rolling pat-

Table 1 Theoretical Frameworks Suggesting an Influential-Imitator Mixture

Framework	Influentials	Imitators	Reason to imitate	Who gets imitated[a]
Social character	Autonomous and inner-directed; $q_1 = 0$	Other-directed	Looking for approval and direction	—Not specified, possibly all adopters ($w = \theta$)
Status competition and maintenance	High status; $q_1 \geq 0$	Low status	Gaining or maintaining status	—All adopters ($w = \theta$) —Only influentials ($w = 1$) —Only imitators ($w = 0$)
Middle-status conformity	High and low status; $q_1 = 0$	Middle status	Conforming to social norms	—All adopters ($w = \theta$) —Only influentials with high status —Only imitators ($w = 0$)
Two-step flow	Active and involved (opinion leaders); $q_1 \geq 0$	Not active or involved	Transferring information	—All adopters ($w = \theta$) —Only influentials ($w = 1$) —Only imitators ($w = 0$)
Technology chasm	Technology enthusiasts and visionaries; $q_1 \geq 0$	Mainstream customers	Reducing risk	—All adopters ($w = \theta$) —Only imitators ($w = 0$)

[a]Parameter w denotes how much the social contagion affecting the imitators stems from the influentials (w) rather than fellow imitators ($1 - w$). Parameter θ is the fraction of ultimate adopters belonging to Segment 1 (influentials).

tern through the population where all prior adoptions contribute to social contagion (e.g., Katz 1957, Merton 1949). This suggests $w \approx \theta$. However, it is quite possible that opinion leaders are more influential, suggesting that—in the extreme case—they may be the only ones being imitated ($w = 1$). Conversely, it is also quite possible that imitators consider fellow imitators to be more representative, and hence valuable as information sources, suggesting low values of w.

2.5. High-Technology Adoption Chasm

In Moore's (1991) chasm framework for technology products, the so-called early market consists of "technology enthusiasts" and "visionaries" who are quick to appreciate the nature and benefits of the innovation, whereas the mainstream market consists of more risk-averse decision makers and firms who fear being stuck with a technology that is not user friendly, poorly supported, or at risk of losing a standards war. Whereas the mainstream market can be represented as responding only to the size of the installed base, i.e., prior adoptions (Mahajan and Muller 1998), Moore is unclear about the process among technology enthusiasts and visionaries. Whereas his textual discussions suggest that they act independently ($q_1 = 0$), his stylized graph of the bell-shaped adoption curve with a chasm is mathematically inconsistent with a constant-hazard process in the early stages of diffusion, and requires $q_1 > 0$. Note that for the chasm to be truly problematic, $p_2 \to 0$ is required.

Moore does not clearly specify whose adoptions are being imitated (w). On the one hand, one might argue that the legitimacy of a new technology is affected by the penetration rate in the overall population, i.e., the total installed base regardless of who adopted ($w = \theta$). On the other hand, Moore emphasizes that product and service offerings appealing to technology enthusiasts and visionaries need not appeal to the mainstream market, which implies that mainstream customers discount adoptions by technology enthusiasts and visionaries and care only about adoptions by other mainstream customers ($w = 0$).[3]

2.6. Conclusion

At least five different theoretical frameworks suggest modeling innovation diffusion using a two-segment structure consisting of influentials and imitators (Table 1). Two theories suggest that influentials adopt independently, implying $q_1 = 0$, but the other three suggest that influentials may exhibit contagion amongst themselves.[4] While one might intuitively expect $p_1 > p_2$ and none of the theories rules this out, this inequality is suggested only by adherents of the chasm framework. Also, several studies have documented that the majority of earliest adopters need not always be opinion leaders with disproportionate influence (Weimann 1994), implying $\theta p_1 < (1 - \theta)p_2$ and leaving $p_1 < p_2$ as a possibility. Similarly, while one might intuitively expect $q_1 < q_2$ and none of the theories rules this out, this inequality is required only

[3] Moore himself is far from clear on the issue when discussing the relationship between "visionaries" in the early market and "pragmatists," i.e., the early adopters among the members of the mainstream market. At one point, he admonishes the reader to "do whatever it takes to make [visionaries] satisfied customers so that they can serve as good references for the pragmatists," but on the very next page he writes that "pragmatists think visionaries are dangerous. As a result, visionaries, with their highly innovative . . . projects do not make good references for pragmatists" (Moore 1995, pp. 18–19).

[4] Independent decision making among influentials is also consistent with Midgley and Dowling (1978), who define innovativeness as "the degree to which an individual makes innovation decisions independently of the communicated experience of others" (p. 235). Therefore, our distinction between independent influentials (with $q_1 = 0$) and pure imitators with $p_2 = 0$ is the same as their dichotomy between "innate innovators" and "innate noninnovators."

by the two theories implying $q_1 = 0$ and $q_2 > 0$, and several studies have documented that opinion leaders with disproportionate influence may also greatly influence one another (e.g., Weimann 1994). All theories allow for the initial impetus among imitators to stem from influentials, and so allow for $p_2 = 0$.

The theories vary in their causal mechanisms and, consequently, in what kind of actors belongs to each segment and who the imitators imitate (w). The theories also suggest that the relative size of the segments (θ) can vary from innovation to innovation. It may be quite low for very nonmainstream products that only a very small pocket of "bleeding edge" customers find attractive, but that in spite of the latter's enthusiasm take a long time to diffuse, resulting in an adoption curve with a long left tail. Conversely, for products with low functional or financial risk and with little implications for social status, like marginally novel drugs or CDs and movies with already famous performers, most adopters may feel little need for information or legitimation from peers. This implies a high θ, a low q_1, and an exponential-like diffusion process (e.g., Moe and Fader 2001, Van den Bulte and Lilien 2001).

3. Two-Segment Mixture Models

We seek closed-form solutions in the time domain for an innovation's diffusion path when the set of eventual adopters, which has a constant size M, consists of two a priori different types of actors adopting according to Equations (3) and (4). The overall cumulative penetration is simply the average of both types' cumulative penetration weighted by their constant population weights (e.g., Cox 1959):

$$F_m(t) = \theta F_1(t) + (1-\theta)F_2(t). \tag{5}$$

Similarly, the fraction of the population adopting at time t is:

$$f_m(t) = \theta f_1(t) + (1-\theta)f_2(t). \tag{6}$$

In contrast, the population hazard function is not an average of the two hazards weighted by each segment's constant population weights, but is given by:

$$\begin{aligned} h_m(t) &= f_m(t)/[1-F_m(t)] \\ &= [\theta f_1(t) + (1-\theta)f_2(t)]/[1-F_m(t)] \\ &= \pi(t)h_1(t) + [1-\pi(t)]h_2(t), \end{aligned} \tag{7}$$

where $f_i(t) = h_i(t)[1-F_i(t)]$ and $\pi(t)$ is the proportion of actors not having adopted yet at time t that belong to Type 1:

$$\pi(t) = \theta\frac{1-F_1(t)}{1-F_m(t)}. \tag{8}$$

Finally, the proportion of adoptions made by actors of Type 1 taking place at time t is:

$$\phi(t) = \theta f_1(t)/f_m(t). \tag{9}$$

3.1. Asymmetric Influence Model (AIM) with $q_1 > 0$

Having defined the key functions, and having made the behavioral assumptions in the hazard functions (Equations (3) and (4)), we now develop the asymmetric influence mixture model (AIM). The process among the influentials is the well-known mixed-influence model. When $F_1(0) = 0$, the cumulative penetration function and instantaneous adoption function for influentials are:

$$F_1(t) = (1-e^{-(p_1+q_1)t})\Big/\left(1+\frac{q_1}{p_1}e^{-(p_1+q_1)t}\right); \tag{10}$$

$$f_1(t) = \left(p_1\left(1+\frac{q_1}{p_1}\right)^2 e^{-(p_1+q_1)t}\right)\Big/\left(1+\frac{q_1}{p_1}e^{-(p_1+q_1)t}\right)^2. \tag{11}$$

The diffusion path among imitators, in contrast, does not follow any standard diffusion model, because it is driven by the prior adoptions of both influentials and other imitators. As shown in Appendix A1, when $F_2(0) = 0$, the cumulative penetration function for imitators in the AIM is:

$$\begin{aligned} F_2(t) = 1 &+ (-p_2q_1 + q_2(p_1w - q_1(1-w))) \\ &\cdot\left(q_1q_2(1-w)H_1 + e^{(p_2+q_2)t}\left(\frac{p_1+q_1e^{-(p_1+q_1)t}}{p_1+q_1}\right)^{wq_2/q_1}\right. \\ &\left.\cdot(p_2q_1 + q_2(q_1(1-w)(1-H_2) - p_1w))\right)^{-1}, \end{aligned} \tag{12}$$

where

$$H_1 = {}_2F_1\left(1, \frac{wq_2}{q_1}, 1+\frac{wq_2}{q_1}-\frac{p_2+q_2}{p_1+q_1}, \frac{p_1}{p_1+q_1e^{-(p_1+q_1)t}}\right),$$

$$H_2 = {}_2F_1\left(1, \frac{wq_2}{q_1}, 1+\frac{wq_2}{q_1}-\frac{p_2+q_2}{p_1+q_1}, \frac{p_1}{p_1+q_1}\right),$$

and ${}_2F_1(1, b; c; k)$ is the Gaussian hypergeometric function:

$${}_2F_1(1, b; c; k) = \sum_{n=0}^{\infty}\frac{\Gamma(b+n)\Gamma(c)}{\Gamma(b)\Gamma(c+n)}k^n. \tag{13}$$

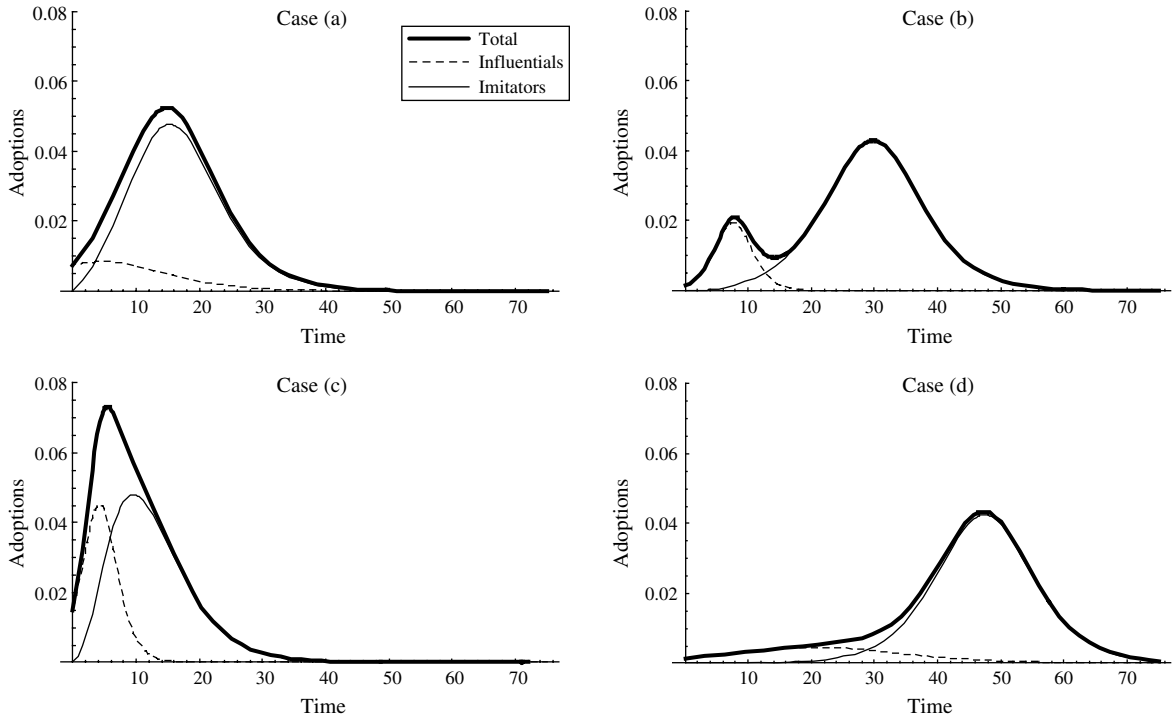
This hypergeometric series is convergent for arbitrary b, c if $|k| < 1$; and for $k = \pm 1$ if $c > 1 + b$. This implies that the closed-form solution in Equation (12) is well defined as long as $q_1 > 0$.[5] Once $F_1(t)$ and $F_2(t)$ are known, one can obtain the instantaneous adoption function $f_2(t)$ by substituting Equations (10) and (12) into:

$$f_2(t) = [p_2 + q_2[wF_1(t) + (1-w)F_2(t)]][1-F_2(t)]. \tag{14}$$

With solutions for $F_1(t)$, $f_1(t)$, $F_2(t)$, and $f_2(t)$ available, one can enter those into Equations (5) through (9) to

[5] While Gaussian hypergeometric functions ${}_2F_1(1, b; c; k)$ can be simplified to incomplete beta functions, we do not perform this simplification because it requires the overly restrictive condition that $p_1w > q_1(1-w+p_2/q_2)$.

Figure 1 Adoption Functions for Four AIM Diffusion Processes



obtain closed-form solutions for the population-level functions.[6]

In Figure 1, we plot the function $f_m(t)$ and its two components $\theta f_1(t)$ and $(1-\theta)f_2(t)$ for four sets of parameter values chosen to illustrate various types of diffusion behavior possible in this model when $p_2 = 0$ and interconnection between segments is crucial:

Case (a): $p_1 = 0.05$; $q_1 = 0.1$; $q_2 = 0.2$; $\theta = 0.15$; $w = 0.20$;

Case (b): $p_1 = 0.01$; $q_1 = 0.5$; $q_2 = 0.2$; $\theta = 0.15$; $w = 0.01$;

Case (c): $p_1 = 0.05$; $q_1 = 0.5$; $q_2 = 0.2$; $\theta = 0.30$; $w = 0.30$;

Case (d): $p_1 = 0.01$; $q_1 = 0.1$; $q_2 = 0.2$; $\theta = 0.15$; $w = 0.001$.

Diffusion process (a) exhibits a bell-shaped adoption curve $f_m(t)$ that is unimodal and close to symmetric around its peak. This is the pattern commonly associated with the mixed-influence model. Diffusion process (b) is bimodal and exhibits a marked dip because adoptions by influentials are already well past their peak by the time the imitators start adopting in numbers (the delay being caused by the low w value). This is the much-debated "chasm" pattern. Diffusion processes (c) and (d), finally, are again unimodal but exhibit a clear skew to the right or left, which the mixed-influence cannot account for very well (e.g., Bemmaor and Lee 2002).[7] Note that in all four cases, $f_1(t)$ reaches zero before $f_2(t)$ does, so the commonly expected association between being an imitator and being a late adopter holds. Also note that, as one would intuit, low values of w cause the diffusion among imitators to be delayed and $f_2(t)$ to shift to the right. We now turn to the case where $q_1 = 0$, and study it in more detail using the functions $h_m(t)$, $\pi(t)$, and $\phi(t)$.

3.2. Asymmetric Influence Model (AIM) with $q_1 = 0$ and Pure-Type Mixture Model (PTM)

When influentials adopt independently and $q_1 = 0$, the process among the independents is the well-known constant-hazard exponential process. When $F_1(0) = 0$,

[6] Even though our closed-form solution for $F_2(t)$ in the AIM looks quite different from the solution presented by Steffens and Murthy (1992), theirs is actually nested in ours. After imposing the constraints $p_2 = 0$ and $w = \theta$, reparameterizing the Steffens-Murthy solution in terms of m, θ, p_1, q_1, and q_2, correcting for an (most likely typographic) error in their solution, and performing additional derivations, one can show that our closed-form solution for $F_2(t)$ in the AIM, and hence $F_m(t)$, is identical to theirs. One difference, though, is that their solution requires $q_1 > q_2\theta$ (or $q_1 > q_2 w$) for a series expansion term in their solution to converge, whereas the solution in Equation (12) only requires $q_1 > 0$.

[7] All four patterns for the total number of adoptions shown in Figure 1 have been documented in prior research. Pattern (a) is probably the most commonly reported in the marketing literature. Steffens and Murthy (1992) and Karmeshu and Goswami (2001) report data series exhibiting the bimodal pattern (b). Dixon (1980) reports the presence of long right tails, i.e., pattern (c), in many of the data he analyzed. Van den Bulte and Lilien (1997) report several data series exhibiting long left tails, i.e., pattern (d).

we have:

$$F_1(t) = 1 - e^{-p1t}; \tag{15}$$

$$f_1(t) = p_1 e^{-p1t}. \tag{16}$$

As shown in Appendix A2, when $q_1 = 0$ and $F_1(0) = F_2(0) = 0$, the cumulative penetration function for imitators in the AIM is:

$$F_2(t) = 1 + \exp\left(-p_2 t - q_2 t - \frac{q_2}{p_1} w e^{-p_1 t}\right) \cdot \left(\frac{q_2}{p_1}(1-w)\left(\frac{q_2}{p_1} w\right)^{-(p_2+q_2)/p_1} \cdot \left(\Gamma\left(\frac{p_2+q_2}{p_1}, \frac{q_2}{p_1} w e^{-p_1 t}\right) - \Gamma\left(\frac{p_2+q_2}{p_1}, \frac{q_2}{p_1} w\right)\right) - \exp\left(-\frac{q_2}{p_1} w\right)\right)^{-1}, \tag{17}$$

where $\Gamma(\eta, k)$ is the "upper" incomplete gamma function:

$$\Gamma(\eta, k) = \int_k^\infty v^{\eta-1} e^{-v}\, dv.$$

The instantaneous adoption function $f_2(t)$ is obtained by substituting Equations (15) and (17) into (14). With solutions for $F_1(t)$, $f_1(t)$, $F_2(t)$, and $f_2(t)$ available, one can enter those into Equations (5) through (9) to obtain closed-form solutions for the population-level functions.

A case of special interest is that of a pure-type mixture (PTM) of *pure independents* with $q_1 = 0$ and *pure imitators* with $p_2 = 0$. In Figure 2, we plot the functions $f_m(t)$, $h_m(t)$, $\pi(t)$, and $\phi(t)$ for three sets of parameter values chosen to illustrate various types of diffusion behavior possible in this model:[8]

Case (a): $p_1 = 0.15$, $q_2 = 0.50$, $\theta = 0.25$, $w = 0.25$;
Case (b): $p_1 = 0.25$, $q_2 = 0.40$, $\theta = 0.15$, $w = 0.01$;
Case (c): $p_1 = 0.15$, $q_2 = 0.65$, $\theta = 0.60$, $w = 0.05$.

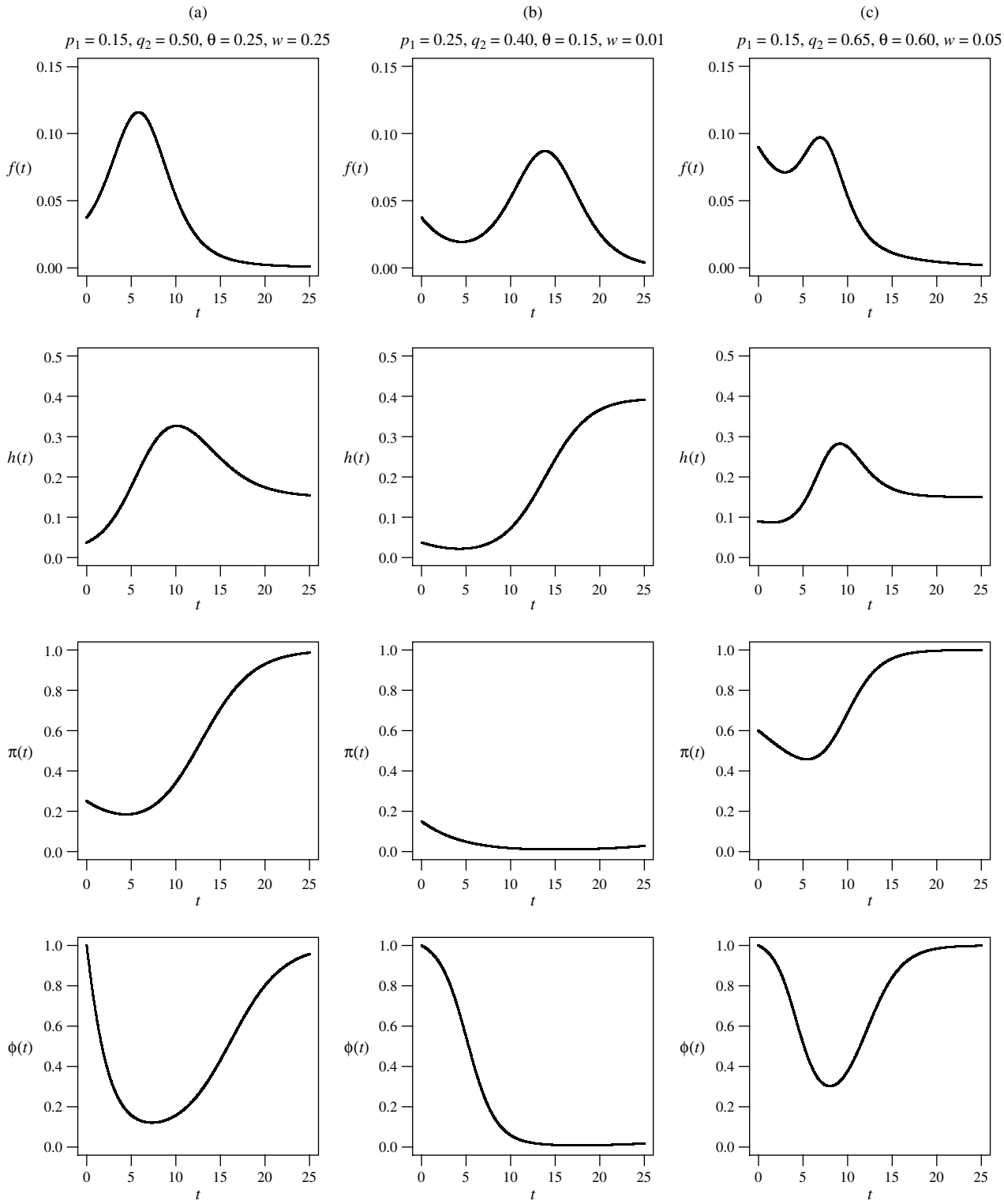
Diffusion process (a) exhibits the common unimodal, symmetric-around-the-peak adoption curve $f_m(t)$ well captured by the mixed-influence model. More interesting is that the hazard function is not monotonic as in the mixed-influence model. Rather, it is roughly bell shaped and seems to converge to a value in-between the minimum and the maximum. Here is why. The very earliest adopters consist of independents, and the population hazard equals $\theta p_1 = 0.0375$ at first. As more and more imitators adopt with hazard $q_2 F_m(t)$, the population hazard increases. Once $q_2 F_m(t) > p_1$, which can happen quickly when q_2 is markedly larger than p_1, the set of imitators not having adopted yet will start depleting faster than the set of independents not having adopted yet. As a result, the laggards still remaining to adopt consist increasingly of independents—as indicated by the function $\pi(t)$ reaching a minimum around $t = 5$ and then increasing to 1—and the population hazard converges back to an asymptote of $p_1 = 0.15$. This pattern of relative speed of depletion also explains the nonmonotonic pattern in $\phi(t)$, the proportion of adoptions taking place at time t stemming from independents. Note that in this diffusion process, independents make up the bulk not only of the early adopters, but also of the very late adopters. Importantly, the point at which $\phi(t)$ starts increasing and independents start gaining rather than losing importance ($t = 7.3$) occurs when the process is still far from complete and the remaining market potential is still quite sizable (37% because $F_m(t) = 0.63$ at $t = 7.3$).

Diffusion process (b) differs in several respects from process (a). First, the adoption curve $f(t)$ does not have a smooth bell shape, but exhibits a clear dip early on. This is easily explained. The independents adopt rapidly because $p_1 = 0.25$ is rather high. However, imitators' reaction to those independent adoptions is very muted because they imitate mostly fellow imitators ($w = 0.01$). As a result, the adoptions by independents show an exponential decline that is not immediately compensated by the imitators' slowly developing adoptions, resulting in an early dip in the population curve. Note that independents account for the bulk of the adoptions only early in the diffusion process, as $\phi(t)$ declines steeply to close to zero. Therefore, while the adoption curve does not fit the standard model, we do have the commonly expected association between being an imitator and being a late adopter.

Diffusion process (c) looks mostly like an exponential-like process commonly observed for fast-moving consumer goods, CDs and films, but with a marked boost after the early periods. What is happening is that most adopters are independents ($\theta = 60\%$), so the majority of adoptions follow an exponential decline. However, there is also a sizable segment of imitators that are very sensitive to social contagion ($q_2 = 0.65$), but mostly from fellow imitators rather than independents ($w = 0.05$). As a result, the imitators are slow to adopt at first, but once the snowball starts rolling, tend to adopt in a very short time. This is reflected in the shape of $\phi(t)$: The proportion of adoptions accounted for by independents tends to be close to 100%, except for a relatively narrow time window during which it first declines and then increases

[8] Of the three shapes of adoption curve in Figure 2, pattern (a) is probably the most commonly reported in the diffusion literature. The other two shapes have not been documented as extensively, but do occur in previously analyzed data. For instance, the sales curve of several music CDs studied by Moe and Fader (2001) exhibit pattern (b) or (c), and the classic *Medical Innovation* data analyzed by Coleman et al. (1966) also exhibit pattern (c).

Figure 2 Plots of Functions Characterizing Three PTM Diffusion Processes



again. The contrast between process (a) and (c) is informative: They have similar p_1 and q_2 values, and the composition of both adopters $\phi(t)$ and remaining nonadopters $\pi(t)$ tend to evolve similarly, as do their respective population hazard functions $h(t)$. However, because of the different segment sizes θ and contagion weights w in the two processes, the resulting adoption curves are quite different.

3.3. Some Special Cases of Theoretical Interest

Our review of prior theories and frameworks indicates that three cases of the social influence struc-

ture captured by w are of special theoretical interest. The first is where imitators imitate only influentials ($w = 1$), such that $h_2(t) = p_2 + q_2F_1(t)$.[9] The second is where imitators imitate only other imitators ($w = 0$), such that $h_2(t) = p_2 + q_2F_2(t)$. The third is where imitators mix randomly with both independents and imitators, such that $w = \theta$ and $h_2(t) = p_2 + q_2F_m(t)$. In the first and third case, $F_2(t)$ and $f_2(t)$ are easily derived by imposing $w = 1$ and $w = \theta$, respectively, in Equations (12), (14), and (17). The second case poses an issue when $p_2 = 0$ and the process among imitators is only a function of prior adoptions by other imitators: The process is then simply the well-known logistic process, which does not allow for $F_2(0) = 0$.[10]

A fourth case of special interest is less obvious: When all independents adopt instantaneously with $p_1 \to \infty$ and pure imitators ($p_2 = 0$) have a very specific influence weight $w = \theta(1 + q_2 - \theta)/q_2 > \theta$, then the PTM reduces to the MIM (see Technical Appendix A).[11]

4. Relation to Prior Diffusion Models

4.1. Mixed-Influence Model vs. Pure-Type Mixture Model

As the closed-form solutions and the plots in Figure 2 indicate, the mixed-influence model (MIM) does not capture diffusion processes in a discrete mixture of pure independents and pure imitators (PTM). Two exceptions to this are the case where $p_1 = 0$ or $\theta = 0$ and both models collapse to the logistic model, and the case where $q_2 = 0$ or $\theta = 1$ and both models collapse to the exponential model. A third, less obvious, exception is when $p_1 \to \infty$ and $w = \theta(1 + q_2 - \theta)/q_2 > \theta$, and the PTM also reduces to the MIM.

Our analysis allows one to assess the widely accepted notion (e.g., Mahajan et al. 1993) that rewriting the standard differential equation for the mixed influence model (Equation (1)) into

$$f(t) = p[1 - F(t)] + qF(t)[1 - F(t)] \tag{18}$$

allows one to interpret the term $p[1 - F(t)]$ as the number of adoptions made by people adopting with hazard p; and the term $qF(t)[1 - F(t)]$ as the number of adoptions made by people adopting with hazard $qF(t)$. While the manipulation of the equation is evidently correct, the interpretation is not. The main reason is that, in each term, the fraction of actors not having adopted yet, $1 - F(t)$, refers to the total population, rather than to the fractions in each of the segments, $1 - F_1(t)$ and $1 - F_2(t)$. In addition, the sizes of each segment are ignored. The correct expression for a mixture is:

$$\begin{aligned} f_m(t) &= \theta f_1(t) + (1 - \theta) f_2(t) \\ &= \theta h_1(t)[1 - F_1(t)] + (1 - \theta) h_2(t)[1 - F_2(t)] \\ &= \theta p_1[1 - F_1(t)] + (1 - \theta) q_2 \\ &\quad \cdot [wF_1(t) + (1 - w)F_2(t)][1 - F_2(t)]. \end{aligned} \tag{19}$$

When imitators randomly mix with independents and imitators and are equally affected by both, then $w = \theta$ and the equation simplifies to:

$$f_m(t) = \theta p_1[1 - F_1(t)] + (1 - \theta) q_2 F_m(t)[1 - F_2(t)]. \tag{20}$$

Even if $p = \theta p_1$, $q = (1 - \theta)q_2$, and one omits the m-subscript from the population-level $f_m(t)$ and $F_m(t)$, the mixture Equation (20) is different from the mixed-influence Equation (18).

Within a homogeneous population with mixed influence, one can only interpret the relative size of the two terms $p[1 - F(t)]$ and $qF(t)[1 - F(t)]$ as reflecting the relative influence of time-invariant elements (p) versus social contagion ($qF(t)$) on the adoptions at time t, keeping in mind that each and every adoption is influenced by both p and $qF(t)$ for any $t > 0$. For instance, the ratio $p/(p + qF(t))$ can be used as a measure of the relative strength of time-invariant elements at time t (Lekvall and Wahlbin 1973), as can the decomposition presented by Daley (1967) and Mahajan et al. (1990), but neither can be interpreted as the fraction of all adoptions at time t stemming from pure-type actors adopting a priori with hazard p.

Another common belief about the mixed-influence model that is inconsistent with its mathematical structure is that "the importance of innovators will be greater at first but will diminish monotonically with time," where innovators are defined as those who "are not influenced in the timing of their initial purchase by the number of people who have already bought the product" (Bass 1969, p. 217). In a homogeneous population where everyone behaves according to the hazard rate $p + qF(t)$, the only actors with hazard p are those adopting at $t = 0$ when $F(0) = 0$. Anyone adopting afterwards is influenced by prior adoptions. Hence, in the mixed-influence model, the proportion of adoptions occurring at time t that are unaffected by social contagion follows a step function with value 1 at $t = 0$ and value 0 for any $t > 0$. Conversely, in a mixture with $p_1 \ll \infty$, the proportion of independents adopting with a constant hazard, i.e., function $\phi(t)$, need not diminish monotonically over time, as shown in Figure 2.

[9] This model, with the additional constraints $q_1 = p_2 = 0$, was also developed independently from us by Beck (2007).

[10] Note that when $p_2 = w = 0$ or $p_2 = \theta = 0$, the process among imitators cannot get started within the model. As is well known, the closed-form solution for the logistic requires that $F_2(0) > 0$. Hence, while the cases with $p_2 = w = 0$ or $p_2 = \theta = 0$ are conceptually nested within the AIM, their closed-form solutions are not because they make different assumptions about the initial conditions.

[11] All technical appendices are available online at the *Marketing Science* website.

4.2. Consequence of Imposing a Mixed-Influence Structure on a Pure-Type Mixture Process

From comparing Equations (18) and (20), one may get the impression that a diffusion process in a discrete mixture with $h_1(t) = p_1$ and $h_2(t) = q_2F_m(t)$ could be approximated quite well by a mixed-influence model with $h(t) = p + qF(t)$, even if they are not identical. However, the adoption functions $f_m(t)$ and hazard functions $h_m(t)$ suggest some potentially important deviations. More insight comes from rewriting the expression for $f_m(t)$ in Equation (20) into a form similar to that for $f(t)$ in the mixed-influenced model (following Manfredi et al. 1998):

$$\begin{aligned} f_m(t) &= \theta p_1[1-F_1(t)] + (1-\theta)q_2F_m(t)[1-F_2(t)] \\ &= \left[\theta p_1\frac{1-F_1(t)}{1-F_m(t)} + (1-\theta)q_2F_m(t)\frac{1-F_2(t)}{1-F_m(t)}\right][1-F_m(t)] \\ &= [p(t) + q(t)F_m(t)][1-F_m(t)] \end{aligned} \tag{21}$$

where

$$p(t) = \theta\frac{1-F_1(t)}{1-F_m(t)}p_1 = \pi(t)p_1 \tag{22}$$

$$q(t) = (1-\theta)\frac{1-F_2(t)}{1-F_m(t)}q_2 = [1-\pi(t)]q_2. \tag{23}$$

Deleting the m subscript from Equation (21) to reflect one's ignoring that the population consists of a mixture results in:

$$f(t) = [p(t) + q(t)F(t)][1-F(t)]. \tag{24}$$

Therefore, one is able to rewrite the pure-type mixture model with $w = \theta$ into an expression akin to the mixed-influence model, but with both hazard-rate parameters varying systematically over time. More specifically, $p(t)$ changes in exactly the same way as $\pi(t)$, the proportion of actors not having adopted yet by time t that belong to the segment of independents. At $t = 0$, $\pi(t) = \theta$ and $p(t) = \theta p_1$. Because at the very beginning adoption tends to be more prevalent among independents than among imitators, the number of independents who have not adopted yet gets depleted faster than the number of imitators who have not. Consequently, $\pi(t)$ and $p(t)$ decline at first. However, when $q_2 \gg p_1$, the relative speed of adoption between the two segments quickly reverses and the set of actors who have not adopted yet tends to become increasingly dominated by independents. As a result, $\pi(t)$ and $p(t)$ increase over most of the time window. The reverse pattern takes place for $q(t) = [1-\pi(t)]q_2$. It starts at $(1-\theta)q_2$, increases for a very short period, but starts decreasing very soon. Note, when $\theta \approx 0$ or $\theta \approx 1$, then $\pi(t)$ will not vary much, and neither will $p(t)$ or $q(t)$.

In short, specifying a mixed-influence model with $h(t) = p + qF(t)$ when the true data-generating process is that of a discrete mixture with $h_1(t) = p_1$ and $h_2(t) = q_2F_m(t)$ where $q_2 \gg p_1$ will yield increasing values of p and decreasing values of q (except for the first very few periods). This is consistent with the pattern in mixed-influence model estimates described in prior research. Although Van den Bulte and Lilien (1997) focused their analysis on ill-conditioning in the absence of model misspecification, they recognized that unobserved heterogeneity in p and q forms an alternative explanation for the systematic changes they observed in empirical applications. Our results formalize their argument for the case of two segments where one segment has $p = 0$ and the other has $q = 0$.

4.3. Relation to Other Two-Segment Models

Figure 3 shows how our models relate to a few other models, including two earlier two-segment models. Tanny and Derzko (1988) used a discrete mixture with $h_1(t) = p_1$ and $h_2(t) = p_2 + q_2F_m(t)$. Steffens and Murthy (1992) used a discrete mixture with $h_1(t) = p_1 + q_1F_1(t)$ and $h_2(t) = q_2F_m(t)$. Therefore, as shown in Figure 3, both these models conceptually nest both the mixed-influence model and PTM3 with $w = \theta$. The diagram also shows that, like the mixed-influence model, the pure-type mixture models have both the exponential and logistic models nested in them, with the exception that PTM1 with $w = 1$ does not nest the logistic because if $h_2(t) = q_2F_1(t)$ and either $\theta = 0$ or $p_1 = 0$, then $h_2(t)$ is undefined. Note that only the PTMs feature two "pure types," i.e., independents and imitators without any mixed influence.

As shown in Technical Appendix B, the solution for $F_2(t)$ in PTM3 is consistent with Jeuland's (1981, p. 14) earlier work. The differences are that he did not specify $h_1(t)$, but kept it general, and that his partial solution still contained unknown integrals. In contrast, we specify the process among independents and solve the equations using incomplete gamma functions, making parameter estimation and empirical analysis possible.

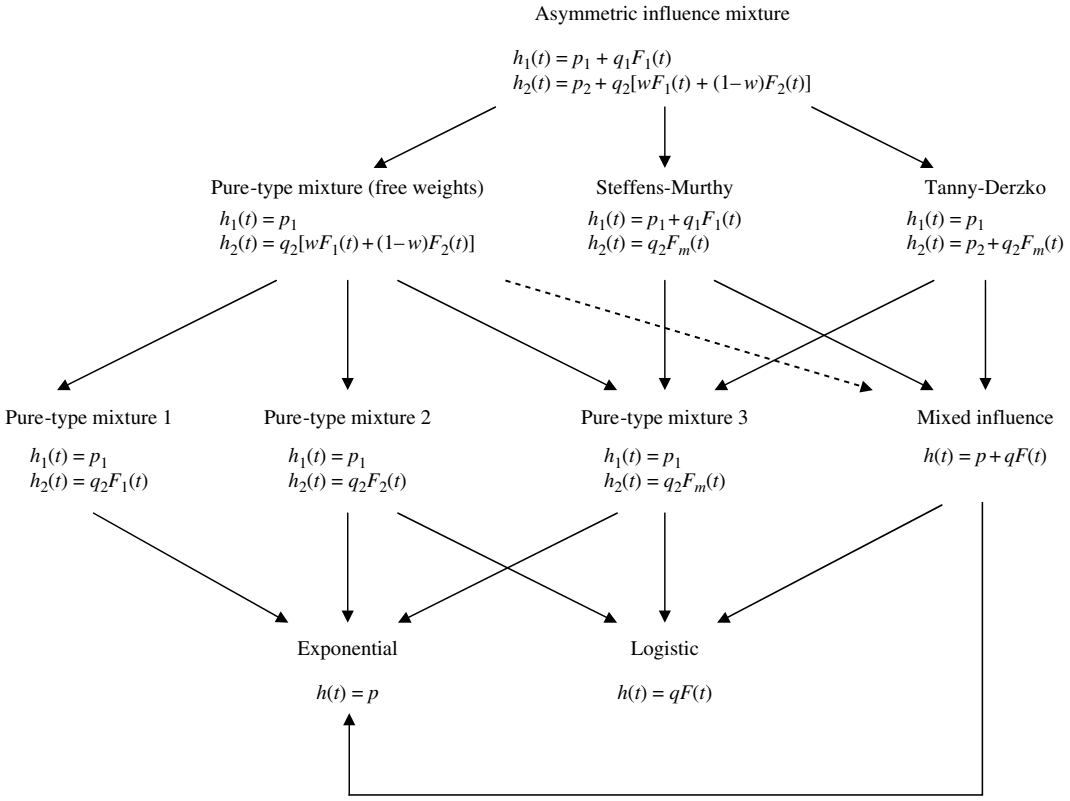
5. Empirical Analysis

To what extent does the two-segment asymmetric influence model, consistent with several theoretical frameworks, agree with empirical diffusion patterns? And how well does it do compared to the mixed-influence model and other, more flexible, models? We provide insights on those issues through an empirical analysis of 33 data series.

5.1. Data

One must use an informative variety of data sets if one is to draw sound conclusions on model performance. We therefore analyze four sets of data. The first consists of a single series on the diffusion of

Figure 3 Relations Among the AIM and PTM Models, the Steffens-Murthy and Tanny-Derzko Models, and the Mixed-Influence, Exponential, and Logistic Models[a]



[a]A model receiving an arrow is conceptually nested in the model where the arrow originates. For instance, the general PTM with $w = 1$ generates PTM1 and the PTM1 with $q_2 = 0$ generates the exponential. The link between PTM and MIM is indicated by a broken line as it holds only as $p_1 \to \infty$.

the broad-spectrum antibiotic tetracycline among 125 Midwestern physicians over a period of 17 months in the mid-1950s. This series comes from the classic *Medical Innovation* study (Coleman et al. 1966). It warrants special attention because it is commonly accepted as an instance of diffusion in a mixture of independents and imitators (e.g., Jeuland 1981, Lekvall and Wahlbin 1973, Rogers 2003).

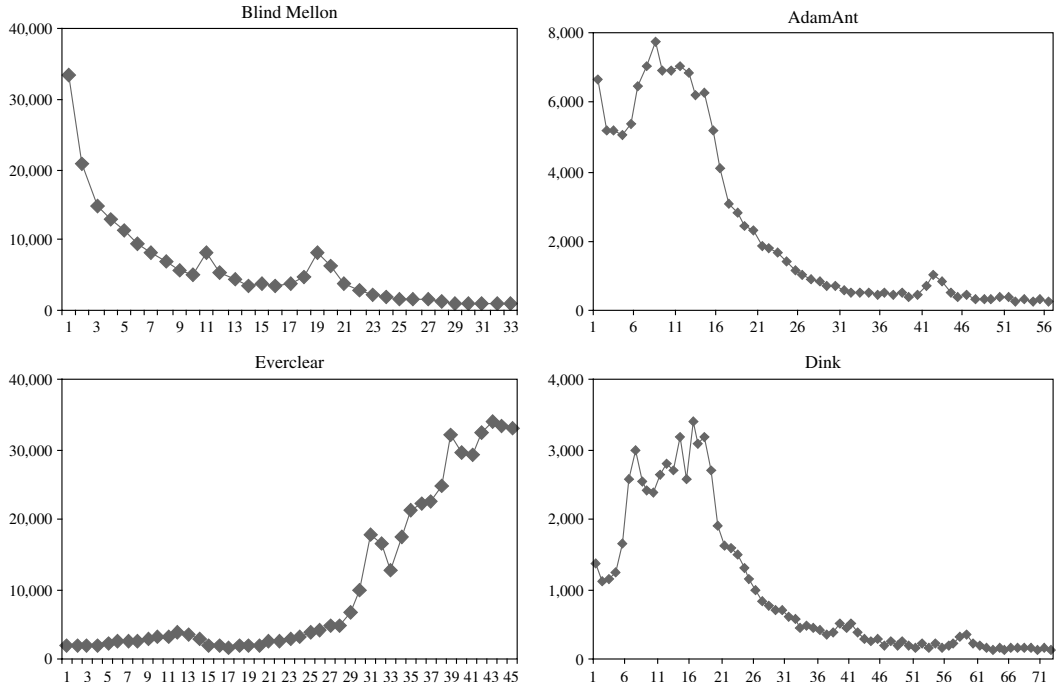
The second set of data series consists of 19 music CDs, a category where a two-segment structure is also likely to exist a priori. Some customers are dedicated fans buying products by their favorite performers almost unconditionally, while others end up buying the CD only after it has become popular and a must-buy (Farrell 1998, Yamada and Kato 2002). Therefore, $q_1 = 0$ and $p_2 = 0$ are quite possible. We use the weekly U.S. sales data analyzed previously by Moe and Fader (2001).[12] Because people are very unlikely to buy two identical CDs for themselves or to replace an older copy, the sales data are unlikely to be contaminated by multiple or repeat purchases and can be treated as new product adoptions. Figure 4 shows the data of four CDs, each illustrating one typical path: a rather smooth decline for Blind Mellon, an early dip followed by a recycle for AdamAnt, a slowly developing "sleeper" pattern for Everclear, and a bell shape for Dink.

The third set of data consists of five series of high-technology products, for which a two-segment structure with $q_1 > 0$ is quite possible (e.g., Moore 1991). The first three series consists of adoptions of CT scanners, ultrasound, and mammography equipment among hospitals of all sizes (Van den Bulte and Lilien 1997). The fourth series consists of the penetration between 1979 and 1993 of CT scanners among hospitals with 50 to 99 beds. Controlling for size may be important, as larger hospitals have larger budgets and more highly skilled staff, and these differences may mask genuine contagion processes (e.g., Davies 1979). The fifth series consists of the penetration of personal computers among U.S. households. The series covers the years 1981–1996, but to avoid left-censoring artifacts we impose 1975 as the actual launch year. The first three series are roughly bell shaped, the latter two series show two "bells" separated by a dip or "chasm."

The final set is a miscellaneous mix of eight data series analyzed previously by Van den Bulte and Lilien (1997) and Bemmaor and Lee (2002) (these studies

[12] The full set consists of 20 data series, but we deleted one that still had not reached the time of peak sales.

Figure 4 Weekly Sales (Adoption) Data for Four CDs



also included the tetracycline and three of the high-tech series). There is no compelling a priori reason to expect a mixture of independents and imitators to be able to better account for those diffusion data than traditional models, and several innovations need not have diffused through contagion at all (Griliches 1962, Van den Bulte and Stremersch 2004). The adoption curves all have a very pronounced bell shape, with several showing skew that the MIM cannot account for (Bemmaor and Lee 2002).

5.2. Parameter Estimates

One of our closed-form solutions involves Gaussian hypergeometric functions, the estimation of which is very troublesome.[13] Fortunately, one can estimate the AIM through direct integration, that is, by computing nonlinear least-squares estimates at the same time as one numerically solves the following differential equation:[14]

$$\begin{aligned} &dX(t)/dt \\ &= M[\theta f_1(t) + (1-\theta) f_2(t)] + \varepsilon(t) \\ &= M\Bigg[\theta f_1(t) + (1-\theta) \\ &\quad \cdot \Bigg[p_2 + q_2\left\{wF_1(t) + (1-w)\frac{X(t)/M - \theta F_1(t)}{1-\theta}\right\}\Bigg] \\ &\quad \cdot \left\{1 - \frac{X(t)/M - \theta F_1(t)}{1-\theta}\right\}\Bigg] + \varepsilon(t) \end{aligned} \tag{25}$$

where $X(t)$ is the cumulative number of adopters observed at time t, $f_1(t)$ and $F_1(t)$ are the closed-form solutions to the adoption and penetration functions of the MIM, and $f_2(t)$ is expressed as in Equation (14), but with $(X(t)/M - \theta F_1(t))/(1-\theta)$ replacing $F_2(t)$. The latter is based on $X(t) = MF_m(t)$ (absent error) and $F_m(t) = \theta F_1(t) + (1-\theta)F_2(t)$. We allow the error term $\varepsilon(t) \sim \mathrm{N}(0, \sigma^2)$ to exhibit serial correlation up to order 2 when the time series contains more than 20 observations or the Durbin-Watson statistic falls outside the 1.5–2.5 range. We impose the contraint that hazard parameters p_1, q_1, p_2, and q_2 be nonnegative (≥ 0) and that $0 \leq \theta \leq 1$. Because hazard rates can be larger than one in continuous time, we do not impose p_1, q_1, p_2, and $q_2 \leq 1$. As to w, we impose $0.01\% \leq w \leq 1$, choosing a very small but positive lower bound so the model itself ensures the "seeding" of the contagion process among imitators even when $p_2 = 0$. Because estimation through direct integration fits the cumulative adoptions $X(t)$ rather than the periodic adoptions $X(t) - X(t-1)$, the R^2 values are often extremely high and noninformative (the lowest we obtained was 0.992, and several were higher than 0.9995). Therefore, we report the mean absolute percentage error (MAPE) instead, as well as an alternative R^2 metric

[13] Nonlinear regression using the "difference in-closed-form-cdfs" approach (Srinivasan and Mason 1986) in R and Mathematica either did not converge at standard convergence criteria or enabled us to obtain point estimates, but not standard errors. We experienced these problems even with simulated data, which rules out model misspecification as an explanation for these difficulties. Maximum-likelihood estimation is known to be troublesome as well, even when the parameters of interest enter the function linearly rather than nonlinearly as in the AIM (e.g., Fader et al. 2005).

[14] This can be done quite conveniently, e.g., using the model procedure in SAS or the odesolve package in *R*.

Table 2 AIM Results for All Data[#]

	N	p_1	q_1	p_2	q_2	θ	w	AR1	AR2	DW	MAPE	R_p^2
Tetracycline	18	0.102[c]	0[*]	0[*]	0.998[c]	0.81[c/c]	0.01%[*]			1.82	2.2%	0.799
AdamAnt	57	0.061[c]	0[*]	0[*]	0.369[c]	0.63[c/c]	0.10[/c]	−0.14	0.07	0.65	0.6	0.986
Beastie Boys	97	1.256[c]	0[*]	0[*]	0.041[c]	0.28[c/c]	1[*]	0.20[c]	0.06[c]	0.67	0.2	0.991
Blind Mellon	34	0.210[c]	3.291	0[*]	0.073[c]	0.24[c/c]	1[*]	0.40	0.16	1.44	0.5	0.964
Bob Seger	24	0.084[c]	0[*]	0[*]	1.357[c]	0.81[c/c]	0.01%[*]	−0.02	0.08	1.67	1.3	0.814
Bonnie Raitt 1	107	0.291[c]	0[*]	0[*]	0.040[c]	0.41[c/c]	1[*]	0.07[a]	−0.09[b]	1.31	0.2	0.984
Bonnie Raitt 2	22	0.096[c]	0[*]	0[*]	1.538[c]	0.74[c/c]	0.01%[*]	0.02	−0.03	1.45	1.9	0.823
Charles & Eddie	32	0.024[b]	0.541[c]	0.050[c]	0.007	0.26[a/c]	0.01%	−0.82	−0.70	1.75	0.7	0.971
Cocteau Twins	127	0.000	14.848	0[*]	0.051[c]	0.10[c/c]	1[*]	0.86[c]	0.25[b]	1.88	0.2	0.950
Dink	73	0.019[c]	0.162[c]	0[*]	0.011	0.67[a/]	1[*]	0.36[c]	0.37[c]	1.67	1.3	0.938
Everclear	46	0.024	0.273[a]	0[*]	0.188[c]	0.05[c/c]	0.01%[*]	0.37[a]	0.22	1.88	3.2	0.969
Heart	124	0.000	1.909[c]	0.074[c]	0[*]	0.08[c/c]	0.01%	−0.18[c]	0.04	0.33	0.3	0.993
John Hiatt	24	0.274	3.282[a]	0[*]	0.192[b]	0.16[a/c]	0.29[/c]	0.37	§	2.04	1.1	0.683
Luscious Jackson	85	0.065	4.153	0[*]	0.028[c]	0.10[c/c]	1[*]	0.41[c]	0.20	1.43	0.4	0.883
Radiohead	73	0.041[c]	0.141[c]	0.001	0.102[c]	0.16[/c]	0.01%	0.43[c]	0.10	1.44	1.5	0.867
Richard Marx	113	0.122[c]	0.074	0.023[a]	0.023	0.43[c/c]	0.01%	0.21[c]	−0.08	0.92	0.3	0.982
Robbie Robertson	79	0.075[c]	0.054	0[*]	0.010	0.58[c/a]	1[*]	0.22[b]	−0.04	1.32	0.6	0.888
Smoking Popes	40	0.089[c]	0.143[c]	0[*]	0.142[c]	0.75[c/c]	0.01%[*]	−0.22[b]	0.01	0.96	0.7	0.966
Supergrass	38	0.157	2.715	0[*]	0.058[a]	0.09[b/c]	0.66[a/]	0.71[c]	0.41[a]	1.43	0.6	0.876
Tom Cochrane	22	0.108[c]	0[*]	0[*]	1.741	0.97[c/]	0.72	−0.01	0.13	0.72	1.8	0.915
Home PC	17	0.000	0.407[c]	0[*]	2.567[c]	0.65[c/c]	0.65[**]			2.20	11.9	0.333
Mammography	15	0.000	1.350[c]	0.015[b]	0.602[c]	0.38[b/b]	0.38[**]			2.89[@]	5.9	0.976
Scanners (all)	18	0.003[c]	0.634[c]	0[*]	0.476	0.63[c/c]	0.01[/c]			2.05	19.7	0.927
Scanners (50–99)	15	0.002	1.031[a]	0.000	0.821[c]	0.60[c/c]	0.01%[*]			1.79	15.0	0.831
Ultrasound	15	0.022	0.309[c]	0[*]	1.113[b]	0.58[a]	0.00			2.49	7.7	0.937
Hybrid corn 1943	16	0.000	0.868[c]	0.192	2.866	0.85[c/c]	0.01%[*]	0.88[a]	0.27	2.39	13.1	0.974
Hybrid corn 1948	15	0.037	0.482	0[*]	0.861	0.20	0.01%[*]			2.47	12.6	0.744
Accel. program	13	0.001	0.786[c]	0[*]	2.394[c]	0.85[c]	0.01%[*]			2.44	26.9	0.842
Foreign language	13	0.656	0[*]	0[*]	0.716[c]	0.06[/c]	0.00[a]			2.81[@]	3.1	0.919
Comp. schooling	15	0.006	0.746[b]	0[*]	0.694	0.69	0.01[/c]			1.82	17.6	0.627
Color TV	17	0.000[a]	0.361[c]	0[*]	1.272[c]	0.78[c/c]	0.01%[*]			1.48[@]	4.0	0.391
Clothes dryers	17	0.000	0.508[c]	0[*]	5.593[b]	0.61[c/c]	1[*]			2.04	3.5	0.819
Air conditioners	17	0.000	1.044[a]	0.000	0.511[c]	0.28[/c]	0.01%[*]			2.37	9.5	0.706

[#] N = number of observations (incl. $X(0) = 0$); AR1, AR2 = first-order and second-order serial correlation, DW = Durbin-Watson statistic, $R_p^2 = r^2$ of actual adoptions with difference in predicted cumulative adoptions.

[*] Boundary constraint; [**] constrained to equal θ to aid convergence; § including AR2 results in convergence problems; [@] adding AR1 and AR2 does not improve DW.

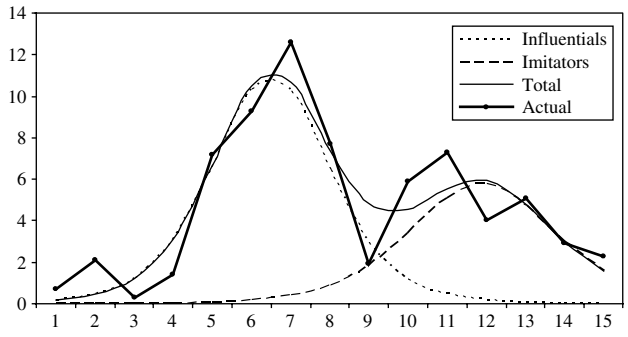
[a] $p \leq 0.05$, [b] $p \leq 0.01$, [c] $p \leq 0.001$; for θ and w, the entry left of the slash (/) refers to the significance of the test against zero and those to the right refer to the test against one.

defined as the squared Pearson correlation between the actual periodic adoptions and the difference in predicted cumulative adoptions (R_p^2).

Table 2 reports the results of estimating the AIM to all 33 data series.[15] Values for p_1 tend be smaller than 0.3. There are two exceptions to this: Foreign Language, where θ is so low that $f_m(0) = \theta p_1$ equals only 0.04, and the Beastie Boys CD that exhibited an extreme "blockbuster" pattern, i.e., extremely quickly declining sales. Values for q_1 show much more variance. This is especially so for CDs. For about half of them, q_1 equals zero, indicating the absence of word of mouth among influentials. In six cases, q_1 is larger than one, suggesting very strong word of mouth among influentials. However, these large estimates are very imprecise, and only two are significant at 95% confidence. Values for p_2 are most often zero, and only four of the 33 estimates are significantly different from zero. Values for q_2 also show considerable variance, with several high values recorded for the set of miscellaneous innovations. The latter may result from the strong left skew in the adoption time series (Bemmaor and Lee 2002). Finally, θ is often significantly different from both zero and one, indicating that the AIM does not reduce to the mixed-influence or logistic models. Also, only weakly correlated with w ($r = -0.16$). That θ is often larger than 2.5% or 16%, traditional values used to separate innovators from imitators based on time of adoption, is an indication—in addition to the $\phi(t)$ function—that the dichotomy based on drivers of adoption underlying the model is conceptually different from that based on time of adoption. The MAPE and R_p^2 values indicate that the model tracks the data well. While the MAPE is higher than 10% for some of

[15] We do not report the ceiling parameter values M due to space constraints in the table.

Figure 5 **Actual and Predicted Adoptions of CT Scanners in Small Hospitals (50–99 Beds)**



the shorter data series, like the 15% value for scanners in small hospitals with 50–99 beds, such high MAPE values can be misleading because they tend to result from a few deviations early in the process when the base for calculating the percentage error is small. Figure 5 shows that the model can indeed track bimodal patterns rather well even with a high MAPE.

Because combining nonlinear least-squares estimation with direct integration may be new to marketing (diffusion) researchers, we briefly report, for the case of tetracycline, estimates obtained through direct integration (DI) with those obtained through the popular Srinivasan-Mason (SM) procedure fitting the difference in closed-form cdfs to the difference in cumulative adoptions. The results in Table 3 clearly show that both procedures produce very similar estimates for the PTM and the MIM. Direct integration has somewhat higher serial correlation because it fits the cumulative adoptions $X(t)$ rather than the periodic adoptions $X(t) - X(t-1)$. The difference in the dependent variable also explains why direct integration produces much lower MAPE values even when the mean squared error (MSE) values are very similar. That the DI method leads to lower R_p^2 values than the SM method is not surprising, because the latter method finds those estimates that minimize the sum of squared errors (SSE), and hence maximizes the correlation between predicted and observed *periodic* adoptions. The parameter estimates of the AIM and PTM, with the zero value of q_1 meaning that Segment 1 consists of independents, and the high value of θ meaning that contagion affected only a minority, are consistent with previous analyses using individual-level data on adoption times and actual network structure (Coleman et al. 1966, Van den Bulte and Lilien 2003), as is the decomposition of total adoptions in Figure 6. The graph indicates that by Month 11, when 25% of all physicians still had to adopt, all imitators had already adopted and the "laggards" consisted only of independents. This is consistent with the original finding by Coleman et al. (1966) using individual-level data that the laggards tended to be very poorly integrated in the social network, and hence unaffected by social influence. Finally, the mixture models generate an estimate of M close to the entire sample of physicians ($N = 125$), whereas the mixed-influence estimates are very close to the number of adopters having adopted at the end of the observation period ($X(t_{17}) = 109$). This is consistent with our analytical result that imposing a mixed-influence model on a mixture process can generate the kinds of estimation artifacts documented by Van den Bulte and Lilien (1997).

5.3. Descriptive Performance Compared to Benchmark Models

To assess the descriptive performance of the two-segment model, we compare it against that of the mixed-influence (MIM), Gamma/Shifted Gompertz (G/SG), Weibull-Gamma (WG), and Karmeshu-Goswami (KG) models. Because all of these benchmark models have a closed-form solution, we estimate them using the standard Srinivasan-Mason (1986) approach. To avoid having comparisons across model specifications be affected by differences in estimation method and dependent variable, we do not estimate the full AIM using the DI approach. Instead, we estimate two restricted versions, one with $w = 0$ and the other with

Table 3 **AIM, PTM, and MIM Results for *Medical Innovation* Tetracycline Data, Using Estimation by Direct Integration (DI) and by the Srinivasan-Mason Procedure (SM)[#]**

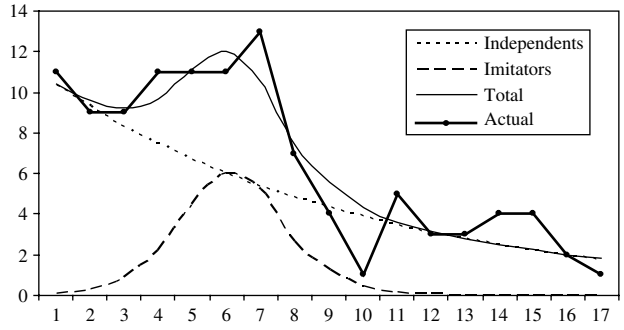
	M	p_1	q_1	p_2	q_2	θ	w	AR1	AR2	DW	MSE	MAPE	R_p^2
AIM-DI	127.0[b]	0.102[b]	0*	0*	0.998[b]	0.81[b/b]	0.01%*	—	—	1.82	2.10	2.2%	0.799
PTM-DI	127.0[b]	0.102[b]	—	—	0.998[b]	0.81[b/b]	0.01%*	—	—	1.82	2.10	2.2	0.799
PTM-SM	131.2[b]	0.097[b]	—	—	1.059[b]	0.81[b/b]	0.03[b/b]	—	—	1.69	2.02	38.8	0.908
MIM-DI	111.6[b]	0.097[a]	0.155	—	—	—	—	0.10	0.14	1.47	4.15	2.6	0.717
MIM-SM	111.3[b]	0.085[a]	0.188	—	—	—	—	0.32		1.82	4.35	43.1	0.784

[#]AR1, AR2 = first-order and second-order serial correlation; DW = Durbin-Watson statistic; for estimation on cumulative data using direct integration (DI), $R_p^2 = r^2$ of actual adoptions with difference in predicted cumulative adoptions; for estimation on periodic data using SM-method, $R_p^2 = r^2$ of actual and predicted adoptions.

*Boundary constraint.

[a]$p \leq 0.05$; [b]$p \leq 0.001$; for θ and w, the entry left of the slash (/) refers to the significance of the test against zero and those to the right refer to the test against one.

Figure 6 **Actual and Predicted Number of Adopters in *Medical Innovation* (Predictions from SM Estimates of the PTM Without Serial Correlation)**



$q_1 = 0$, that lead to closed-form solutions for $F_m(t)$ that do not involve Gaussian hypergeometric functions and that can hence be estimated using the SM approach.

We assess model performance under three error structures: (1) i.i.d. additive error, (2) additive error with AR1 serial correlation, and (3) lognormal multiplicative error.[16] Estimating the models without serial correlation provides a more informative assessment of descriptive performance because incorporating serial correlation into a model might alleviate a poor fit of its mean function to the data (Franses 2002). Still, the question remains to what extent serial correlation alone helps close the gap between two models.

We use four measures of descriptive performance: mean absolute deviation (MAD), mean absolute percentage error (MAPE), mean square error (MSE), and the Bayesian Information Criterion (BIC). Note that only the latter two penalize models with a larger number of free parameters.[17] To save space and aid interpretation, we report only the *ratio* of the baseline models' MSE and MAD to that of the two-segment model. This relative measure controls for differences across data series in their total variance, with one being the neutral value and higher values indicating superior fit of the two-segment model. To save space, we report only the *difference* in BIC and MAPE, with zero being the neutral value and higher values indicating superior fit of the two-segment model.

[16] For the model with lognormal multiplicative error, we estimate its log-transform, i.e., $\ln\{X(t) - X(t-1)\} = \ln M + \ln\{F(t) - F(t-1)\} + \varepsilon(t)$, where $F(t)$ is the closed-form solution of the cdf under the model, and $\varepsilon(t)$ is i.i.d. normal.

[17] $\text{MSE} = \text{SSE}/(n-k)$, where n is the number of observations and k the number of free parameters. $\text{BIC} = -2\text{LL}^c + k\ln(n)$, where LL^c is the concentrated log-likelihood function. Under the assumption of normally distributed errors, the latter is computed from the nonlinear regression solution as $\text{LL}^c = 1/2n\{\ln(n) - 1 - \ln(\text{SSE})\}$ (e.g., Davidson and MacKinnon 1993, Seber and Wild 1989). The use of the concentrated rather than true log-likelihood is immaterial for our purpose. For instance, for nested models, the likelihood ratio test statistic constructed using the concentrated log-likelihood remains χ^2 distributed (Seber and Wild 1989).

Table 4 reports the performance indicators averaged for each of the four sets of data as well as for all 33 data series. Technical Appendix C reports results for the individual series. The first panel pertains to models with additive i.i.d. errors. Let us start by focusing on the BIC, where a three-point difference is large enough to be evidence of superior fit and a 10-point difference provides strong to very strong evidence of superior fit (Raftery 1995). The two-segment model fits markedly better than the MIM, G/SG, and WG models for tetracycline, music CDs, and high-tech products, but *not* for the miscellaneous products where the presumption of a discrete mixture is not strong a priori. The two-segment model fits about equally as well as the continuous-mixture KG model, except for tetracycline, where it beats it by a sizable margin. The same pattern exists for the other three performance measures: The two-segment model fits markedly better than MIM, G/SG, and WG for data where a two-segment structure is a priori likely, but not elsewhere; and the two-segment model fits about equally as well as the Karmeshu-Goswami model in all data sets.

Turning our attention to the second panel in Table 4, we see that allowing for serial correlation in the more poorly specified models tends to somewhat narrow the gap with the two-segment model. However, the performance gap for products where a two-segment structure is a priori likely does not vanish. For high-technology products, adding serial correlation even increases the gap in BIC and MSE vis-à-vis MIM, G/SG, and WG. The results in the third panel of Table 4 indicate that using a multiplicative rather than additive error structure does not affect the main conclusion from the first two panels very much: The two-segment model fits about as well as the continuous-mixture KG model, and markedly better than the MIM, G/SG, and WG models for new products for which a two-segment structure is a priori likely.

6. Conclusion

We have analyzed the diffusion of innovations in markets with two segments: *influentials* who are more in touch with new developments and who affect another segment of *imitators* whose own adoptions do not affect the influentials. Such a structure with asymmetric influence is consistent with several theories in sociology and diffusion research, including the classic two-step flow hypothesis and Moore's (1991) more recent technology adoption framework. Our model allows diffusion researchers to operationalize these theories without recourse to micro-level diffusion data and to estimate parameters from real data. There are four main results.

Table 4 Descriptive Performance of the Two-Segment Model Compared to Mixed-Influence, Gamma/Shifted Gompertz, Weibull-Gamma, and Karmeshu-Goswami Models for Different Error Structures[#]

	BIC difference				MAPE difference				MSE ratio				MAD ratio			
	MIM	G/SG	WG	KG	MIM	G/SG	WG	KG	MIM	G/SG	WG	KG	MIM	G/SG	WG	KG
1. Additive error without serial correlation (AR0)																
Tetracycline	10.44	13.27	14.45	8.99	7.96	8.10	7.88	−0.11	2.21	2.38	2.55	1.46	1.71	1.72	1.76	1.24
Music CDs	58.83	44.09	34.98	1.17	18.73	17.92	8.82	−1.58	2.66	2.06	2.21	0.92	1.69	1.54	1.47	0.98
High-tech	9.84	3.82	4.47	2.05	51.19	6.75	7.16	8.21	2.41	1.57	1.74	1.10	1.92	1.35	1.47	1.05
Miscellaneous	0.73	−2.16	0.54	−0.55	5.56	1.13	3.83	−2.76	1.21	0.93	1.11	0.90	1.24	1.05	1.11	0.88
All	35.14	25.27	19.51	1.12	20.17	11.67	7.12	−0.34	2.14	1.62	1.76	0.96	1.60	1.37	1.37	0.97
2. Additive error with serial correlation (AR1)																
Tetracycline	8.92	8.19	10.06	8.81	4.44	−11.11	6.94	1.14	2.00	1.75	2.13	1.47	1.73	1.44	1.82	1.17
Music CDs	34.19	22.59	28.24	7.65	4.87	2.79	9.30	2.10	2.12	1.65	1.93	1.11	1.47	1.17	1.54	1.11
High-tech	12.67	5.15	7.83	0.22	35.39	6.80	6.82	7.22	2.97	1.70	2.19	0.98	2.12	1.46	1.67	0.91
Miscellaneous	2.62	−1.62	2.79	−1.59	4.12	5.16	1.33	−5.02	1.30	0.91	1.25	0.79	1.23	0.99	1.13	0.80
All	22.45	12.75	16.25	4.21	8.60	3.63	6.39	1.09	1.95	1.42	1.75	1.01	1.48	1.17	1.43	0.99
3. Multiplicative error (log-log model) without serial correlation (AR0)																
Tetracycline	−1.24	−1.37	1.09	−1.68	3.21	3.02	2.90	2.08	1.11	1.10	1.16	0.78	1.24	1.22	1.25	1.02
Music CDs	51.73	35.95	15.32	4.04	1.58	1.20	0.63	−0.09	2.34	1.97	1.54	0.97	1.70	1.54	1.30	0.95
High-tech	9.10	−7.29	3.51	−7.93	8.54	4.38	14.49	−5.56	2.25	0.76	1.65	0.55	1.81	1.06	1.83	0.74
Miscellaneous	4.46	−0.45	7.82	1.68	14.04	−4.75	15.11	0.97	1.60	1.04	1.93	1.03	1.37	1.01	1.48	0.96
All	32.32	19.77	11.26	1.78	5.74	0.14	6.54	−0.44	2.06	1.45	1.64	0.92	1.60	1.31	1.40	0.93

[#]To save space and aid interpretation, we report only the relative fit performance by comparing the fit of the two-segment discrete mixture model against that of the alternative models. For BIC and MAPE, we report the alternative models' value minus that of the two-segment model. For MSE and MAD, we report the alternative models' value divided by that of the two-segment model. Therefore, for the BIC and MAPE differences, the neutral value is zero; for the MSE and MAD ratios, it is one. For all metrics, higher values indicate superior fit of the two-segment model. For the BIC and MAPE differences, the average values reported are arithmetic means. For the MSE and MAD ratios, they are geometric means because this is a better measure of the central tendency of a ratio than the arithmetic mean.

(1) Diffusion in a mixture of influentials and imitators can exhibit the traditional symmetric-around-the-peak bell shape, asymmetric bell shapes, as well as a dip or "chasm" between the early and later parts of the diffusion curve. In contrast to Moore's (1991) contention, the model suggests that it need not always be necessary to change the product to gain traction among later adopters and for the adoption curve to swing up again. Tetracycline is an example.

(2) The proportion of adoptions stemming from independents need not decrease monotonically; it can also first decline and then rise again to unity. This result disproves a common contention among diffusion researchers based on an erroneous mixture interpretation of the mixed-influence model (e.g., Bass 1969, Mahajan et al. 1993, Rogers 2003).

(3) Specifying a mixed-influence model to a mixture process with pure independents and pure imitators can generate systematic changes in the parameter values. As several authors have noted, diffusion within a pure-type mixture of independents and imitators with hazards p and $qF(t)$, respectively, is distinct from diffusion in a homogeneous population with mixed-influence, where everyone adopts with hazard $p + qF(t)$. The closed-form solutions we present not only prove this mathematically, but also show that imposing a mixed-influence specification on a pure-type mixture process can generate the systematic changes in the parameter values reported by Van den Bulte and Lilien (1997), Bemmaor and Lee (2002), and Van den Bulte and Stremersch (2004), unless θ is close to either zero or one, or unless $p_1 \to \infty$ and pure imitators ($p_2 = 0$) have a very specific influence weight $w = \theta(1 + q_2 - \theta)/q_2 > \theta$.

(4) Empirical analysis of four sets of data comprising a total of 33 different data series (the classic *Medical Innovation* data, 19 music CDs, five high-tech products, and eight miscellaneous innovations) indicates that the two-segment model fits markedly better than the MIM, the G/SG, and the WG models, at least for innovations for which a two-segment structure is likely to exist. Hence, the model does better when it is theoretically expected to, and does not when it is not theoretically expected to. The two-segment model fits about equally well as the mixed-influence model proposed by Karmeshu and Goswami (2001), where p and q vary in a continuous fashion. Overall, the findings on descriptive performance are robust to changes in the error structure and indicate that the discrete-mixture model is sufficiently different and the data sufficiently informative for the model to fit real data better than other models.

The model we presented provides sharper insight into how social structure can affect macro-level diffusion patterns, and should prove useful in five areas of application where influentials and imitators are a

priori likely to exist. The first two are high-technology and health care products, including pharmaceuticals. In these two areas, innovations are often perceived to be complex or risky, and mainstream imitators refuse to be on the "bleeding edge," unlike opinion leaders and lead users. The third area is that of entertainment and mass culture products like gaming software, music, books, and movies, where the distinction between aficionados and the casual mainstream audience can loom large.[18] Teen marketing is the fourth area where the distinction between influentials and imitators may be critical in the new product diffusion process. For several years, P&G has been operating Tremor as a mechanism to connect with highly involved and influential teens, encourage adoption among them, and through them reach out to the larger teen population. Categories in which Tremor and similar services have been used include not only fashion-oriented apparel and entertainment, but also more mundane fast-moving consumer goods like beauty aids and food. The fifth area of particular potential consists of situations where a segment of enthusiasts has pent-up demand. For instance, when Internet access providers started operating in France in 1996, a rather large number of people adopted their services. New adoptions dipped in 1997, only to increase again from 1998 onwards. The deviation from the standard bell shape was not the low number in 1997, but the high initial number in 1996, when many university users who had been accessing the Internet exclusively through the university RENATER network were finally able to start using the Internet at home as well (Fornerino 2003). In cases where enthusiasts can place advance orders that the marketing analyst can observe (e.g., Moe and Fader 2002), it may be useful to explicitly allow for a difference between the start time of the diffusion process of the two segments.

[18] Explicitly allowing for influentials and imitators may be especially useful for products carried by characters, writers, actors, or directors who already have a small following among aficionados, but have not yet broken through to the mainstream. In such cases, one would expect the former to adopt according to an independent process and the latter to adopt only through contagion, if at all. This might result in a temporary dip. Movies starring Christina Ricci and movies directed by Ang Lee exhibit this pattern. Early in her career, Ricci played in several independent movies that won critical acclaim and earned her the label of "Indie Queen." These early movies exhibited the bell curve typical of very successful "sleepers" (The Ice Storm-1997, The Opposite of Sex-1998, Buffalo 66-1998). Then followed a small movie exhibiting a dip (Desert Blue-1998), while her recent movies are more standard Hollywood fare exhibiting the standard monotonic, exponential decline (e.g., The Man Who Cried-2001). The same pattern is observed for movies directed by Ang Lee: bell-shaped for The Ice Storm-1997, a temporary dip for Ride with the Devil-1999, and monotonic decline for his more recent Hollywood production, The Hulk-2003.

6.1. Implications for Practice

The first two of our results have clear managerial implications. Because dips in the adoption curve can stem from the mere presence of influentials and imitators, it need not *always* be necessary for firms to change their product to gain traction among later adopters and for the adoption curve to swing up again. In contrast to what Moore (1991) claims, launching a new version to appeal to prospects who have not yet adopted need not always be necessary, let alone optimal, to get out of the dip. Of course, when the dip results not from a social chasm between segments (very low w) but from a difference in what constitutes an acceptable product offering, changing the product will be necessary to gain traction in the second segment.

We have also shown that the proportion of adoptions stemming from influentials need not decrease monotonically; it can also first decline and then rise again. Hence, while it may make sense for firms to shift the focus of their marketing efforts from independents to imitators shortly after launch as shown by Mahajan and Muller (1998) using a two-period model, they may want to start increasing their resource allocation to independent decision makers again later in the process. Managers who confuse the distinction between influentials and imitators with that between early and late adopters, and ignore our results and others' empirical evidence that the bulk of the late adoptions may stem from people not subject to social contagion (e.g., Becker 1970, Coleman et al. 1966), may end up wasting money by poor targeting.

Both of these prescriptive implications assume the existence of influentials and imitators. Of course, thoughtful managers will want to check these assumptions against data from their own markets to assess to what extent they should trust these implications. Standard aggregate-level data and models can be quite misleading for identifying a causal mechanism affecting new product diffusion (e.g., Bemmaor 1994, Van den Bulte and Stremersch 2004). Managers and market researchers must realize that disaggregate data are necessary to gain a better understanding of whether and how social contagion drives the diffusion of their products (e.g., Burt 1987, Van den Bulte and Lilien 2001).

Our work also has important implications for how managers should develop more effective network marketing efforts. Several firms in the pharmaceutical industry, longtime leaders in applying marketing analytics, are now conducting research in which they ask physicians to name the opinion leaders in their social network. Typically, firms use this information to guide their sales reps to the more central physicians. In terms of our model, they are allocating their resources to make $F_1(t)$ grow faster, in the hope that this will get $F_2(t)$ growing faster as well through the

social multiplier effect captured by wq_2. This makes sense, but should be complemented with efforts to increase the multiplier, especially the weight factor w. Rather than focusing only on identifying and converting influentials, firms should also identify ways to increase their impact (e.g., Valente et al. 2003).

The limited value of aggregate-level data to detect contagion effects does not mean that nothing can be learned from them. Following the lead of studies like that of Hahn et al. (1994), firms could analyze the sales evolution of multiple products and look for systematic differences in parameters like θ, w, and q_2 that can be related to product or market characteristics. This, in turn, may help firms develop a better understanding of why product sales evolve the way they do, and might even result in better forecasting models. Such analysis should be useful in all five areas of application identified earlier. From a data availability point of view, it should be particularly appealing to firms in the book, music, and film industries who launch many products each year, and to consulting and research firms with many clients in pharmaceuticals or in high-tech industries.

6.2. Additional Implications for Education and Research

We have shown that some ideas in mathematical diffusion modeling that have become part of the standard marketing curriculum through influential papers and books (Bass 1969, Rogers 2003) are wrong and have misleading marketing implications. We hope our work will help redress this situation in both education and research training.

Several of the implications for practice we presented above have clear research opportunities attached to them. Another important extension of our work would be to incorporate control variables, including marketing efforts. This may not only be useful for empirical research (e.g., to what extent are dips simply caused by exogenous demand shocks?), but may also enable one to more rigorously study the decision to target independents versus imitators. Even a simplified three-period model might be helpful in studying under what conditions it is profit maximizing to change one's targeting from independents to imitators and, possibly, to independents again (Esteban-Bravo and Lehmann 2006). Like the model we presented, this extension would allow one to better understand current arguments and findings, to formalize richer theoretical arguments, and perhaps even to operationalize them into estimable models that help bridge the gap between theory and data.

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Appendix

A.1. Solution for $F_2(t)$ in AIM with $q_1 > 0$

To simplify notation, we omit the time argument from functions and write F_1 instead of $F_1(t)$, etc. We know that

$$F_1 = (1 - e^{-(p_1+q_1)t}) \Big/ \left(1 + \frac{q_1}{p_1} e^{-(p_1+q_1)t}\right).$$

Because $h_2 = p_2 + q_2(wF_1 + (1-w)F_2)$ we write:

$$\begin{aligned}\frac{dF_2}{dt} &= \left(p_2 + q_2 w \frac{1 - e^{-(p_1+q_1)t}}{1 + q_1 e^{-(p_1+q_1)t}/p_1}\right) \\ &\quad + \left(q_2(1-w) - p_2 - q_2 w \frac{1 - e^{-(p_1+q_1)t}}{1 + q_1 e^{-(p_1+q_1)t}/p_1}\right) F_2 \\ &\quad - q_2(1-w)F_2^2. \qquad \text{(A.1.1)}\end{aligned}$$

This is a Ricatti equation of the general form $dy/dx = P(x) + Q(x)y + R(x)y^2$. Setting $y = F_2$ and $x = t$, we get

$$P(x) = p_2 + q_2 w \frac{1 - e^{-(p_1+q_1)t}}{1 + q_1 e^{-(p_1+q_1)t}/p_1};$$

$$Q(x) = q_2(1-w) - p_2 - q_2 w \frac{1 - e^{-(p_1+q_1)t}}{1 + q_1 e^{-(p_1+q_1)t}/p_1};$$

and $R(x) = -q_2(1-w)$. $F_2 = 1$ is a potential solution for this Ricatti equation. We use the transformation

$$z = \frac{1}{F_2 - 1} \quad \Rightarrow \quad F_2 = \frac{z+1}{z}.$$

For F_2 continuous in $[0, 1]$, z is continuous in $(-\infty, -1]$. Note,

$$\frac{dF_2}{dt} = -\frac{1}{z^2}\frac{dz}{dt}.$$

The equation now becomes:

$$\begin{aligned}\frac{dz}{dt} &= q_2(1-w) \\ &\quad + \left(q_2(1-w) + p_2 + q_2 w \frac{1 - e^{-(p_1+q_1)t}}{1 + q_1 e^{-(p_1+q_1)t}/p_1}\right) z. \qquad \text{(A.1.2)}\end{aligned}$$

This is of the form $d\mu/dx + P_1(x)\mu = Q_1(x)$ with $\mu = z$; $x = t$;

$$P_1(x) = -\left(q_2(1-w) + p_2 + q_2 w \frac{1 - e^{-(p_1+q_1)t}}{1 + q_1 e^{-(p_1+q_1)t}/p_1}\right);$$

and $Q_1(x) = q_2(1-w)$. The general solution for such an equation is

$$\mu = \frac{\int u(x) Q_1(x)\, dx + c}{u(x)},$$

where $u(x) = \exp(\int P_1(x)\, dx)$ is the integrating factor. Because

$$\int P_1(x)\, dx = -p_2 t - q_2 t - \frac{q_2 w}{q_1} \ln(p_1 + q_1 e^{-(p_1+q_1)t}),$$

we get

$$u(x) = \exp\left(-p_2 t - q_2 t - \frac{q_2 w}{q_1} \ln(p_1 + q_1 e^{-(p_1+q_1)t})\right).$$

Hence,

$$\int u(x) Q_1(x)\, dx = \frac{e^{-(p_2+q_2)t} q_1 q_2 (1-w)(p_1 + q_1 e^{-(p_1+q_1)t})^{-wq_2/q_1} H_1}{q_2(p_1 w - q_1(1-w)) - p_2 q_1},$$

where

$$H_1 = {}_2F_1\left(1, \frac{wq_2}{q_1}, 1 + \frac{wq_2}{q_1} - \frac{p_2+q_2}{p_1+q_1}, \frac{p_1}{p_1 + q_1 e^{-(p_1+q_1)t}}\right)$$

and ${}_2F_1(1, b, c, k)$ is the Gaussian hypergeometric function, the series representation of which is

$$\sum_{n=0}^{\infty} \frac{\Gamma(b+n)\Gamma(c)}{\Gamma(b)\Gamma(c+n)} k^n.$$

This series is convergent for arbitrary b, c when $|k| < 1$; and when $k = \pm 1$ if $c > 1 + b$. This implies that the series is convergent as long as $q_1 > 0$.

Substituting back, we get

$$z = \frac{\dfrac{e^{-(p_2+q_2)t} q_1 q_2 (1-w)(p_1 + q_1 e^{-(p_1+q_1)t})^{-wq_2/q_1} H_1}{q_2(p_1 w - q_1(1-w)) - p_2 q_1} + c}{\exp\left(-p_2 t - q_2 t - \dfrac{q_2 w}{q_1} \ln(p_1 + q_1 e^{-(p_1+q_1)t})\right)}.$$

Transforming z back to F_2, we obtain

$$F_2 = 1 + \frac{\exp\left(-p_2 t - q_2 t - \dfrac{q_2 w}{q_1} \ln(p_1 + q_1 e^{-(p_1+q_1)t})\right)}{\dfrac{e^{-(p_2+q_2)t} q_1 q_2 (1-w)(p_1 + q_1 e^{-(p_1+q_1)t})^{-wq_2/q_1} H_1}{q_2(p_1 w - q_1(1-w)) - p_2 q_1} + c}. \tag{A.1.3}$$

Because $F_2(0) = 0$,

$$c = \frac{(p_1+q_1)^{-wq_2/q_1}(p_2 q_1 + q_2(q_1(1-w)(1-H_2) - p_1 w))}{q_2(p_1 w - q_1(1-w)) - p_2 q_1},$$

where

$$H_2 = {}_2F_1\left(1, \frac{wq_2}{q_1}, 1 + \frac{wq_2}{q_1} - \frac{p_2+q_2}{p_1+q_1}, \frac{p_1}{p_1+q_1}\right).$$

Simplifying, we obtain as a closed-form expression:

$$F_2(t) = 1 + (-p_2 q_1 + q_2(p_1 w - q_1(1-w))) \cdot \left(q_1 q_2 (1-w) H_1 + e^{(p_2+q_2)t} \left(\frac{p_1 + q_1 e^{-(p_1+q_1)t}}{p_1+q_1}\right)^{wq_2/q_1} \cdot (p_2 q_1 + q_2(q_1(1-w)(1-H_2) - p_1 w))\right)^{-1}. \tag{A.1.4}$$

As $w \to 0$, this expression for $F_2(t)$ reduces to the closed-form solution for the MIM.

A.2. Solution for $F_2(t)$ in AIM with $q_1 = 0$

We know that $F_1 = 1 - e^{-p_1 t}$, and hence $f_2 = dF_2/dt = (p_2 + q_2(wF_1 + (1-w)F_2))(1 - F_2)$ equals:

$$\frac{dF_2}{dt} = p_2 + q_2 w(1 - e^{-p_1 t}) + (q_2(1 - 2w + we^{-p_1 t}) - p_2)F_2 - q_2(1-w)F_2^2. \tag{A.2.1}$$

The above equation is a Ricatti equation of the general form $dy/dx = P(x) + Q(x)y + R(x)y^2$. Setting $y = F_2$ and $x = t$, we have $P(x) = p_2 + q_2 w(1 - e^{-p_1 t})$; $Q(x) = q_2(1 - 2w + we^{-p_1 t}) - p_2$; $R(x) = -q_2(1-w)$.

$F_2 = 1$ is a potential solution for this Ricatti equation. We use the transformation:

$$z = \frac{1}{F_2 - 1} \quad \Rightarrow \quad F_2 = \frac{z+1}{z}, \quad \text{and} \quad \frac{dF_2}{dt} = -\frac{1}{z^2}\frac{dz}{dt}.$$

For F_2 continuous in $[0, 1]$, z is continuous in $(-\infty, -1]$. Substituting in Equation (A.2.1):

$$\frac{dz}{dt} = q_2(1-w) + z(p_2 + q_2(1 - we^{-p_1 t})). \tag{A.2.2}$$

Equation (A.2.2) is of the form: $d\mu/dx + P_1(x)\mu = Q_1(x)$, with $\mu = z$; $x = t$; $P_1(x) = -p_2 - q_2(1 - we^{-p_1 t})$; $Q_1(x) = q_2(1-w)$. The general solution for this equation is

$$\mu = \frac{\int u(x) Q_1(x)\, dx + c}{u(x)}, \tag{A.2.3}$$

where $u(x) = \exp(\int P_1(x)\, dx)$ is the integrating factor. Because

$$\int P_1(x)\, dx = -p_2 t - q_2 t - \frac{q_2}{p_1} we^{-p_1 t},$$

we get

$$u(x) = \exp\left(-p_2 t - q_2 t - \frac{q_2}{p_1} we^{-p_1 t}\right),$$

and hence

$$\int u(x) Q_1(x)\, dx = \int \exp\left(-p_2 t - q_2 t - \frac{q_2}{p_1} we^{-p_1 t}\right) q_2(1-w)\, dt.$$

Now let us define

$$I = q_2(1-w) \int \exp\left(-p_2 t - q_2 t - \frac{q_2}{p_1} we^{-p_1 t}\right) dt. \tag{A.2.4}$$

To solve this integral, we do another transformation:

$$a = e^{-p_1 t} \quad \Rightarrow \quad t = -\frac{1}{p_1} \ln a \quad \Rightarrow \quad dt = -\frac{1}{p_1 a} da.$$

Equation (A.2.4) then becomes

$$I = -\frac{q_2}{p_1}(1-w) \int a^{(p_2+q_2)/p_1 - 1} \exp\left(-\frac{q_2}{p_1} wa\right) da,$$

with the solution

$$I = \frac{q_2}{p_1}(1-w)\left(\frac{q_2}{p_1} w\right)^{-(p_2+q_2)/p_1} \Gamma\left(\frac{p_2+q_2}{p_1}, \frac{q_2}{p_1} wa\right), \tag{A.2.5}$$

where $\Gamma(\eta, k)$ is the "upper" incomplete gamma function:

$$\Gamma(\eta, k) = \int_k^{\infty} v^{\eta - 1} e^{-v}\, dv.$$

Substituting $a = e^{-p_1 t}$ in Equation (A.2.5), and then $I = \int u(x)Q_1(x)\,dx$ from Equation (A.2.5) back into Equation (A.2.3), we obtain:

$$z(t) = \frac{I+c}{u(x)} = \frac{\left[\frac{q_2}{p_1}(1-w)\left(\frac{q_2}{p_1}w\right)^{-(p_2+q_2)/p_1}\Gamma\left(\frac{p_2+q_2}{p_1}, \frac{q_2}{p_1}we^{-p_1 t}\right)+c\right]}{[\exp(-p_2 t - q_2 t - (q_2/p_1)we^{-p_1 t})]}. \quad \text{(A.2.6)}$$

Transforming z back to F_2, we get:

$$F_2(t) = 1 + \frac{\exp(-p_2 t - q_2 t - (q_2/p_1)we^{-p_1 t})}{\frac{q_2}{p_1}(1-w)\left(\frac{q_2}{p_1}w\right)^{-(p_2+q_2)/p_1}\Gamma\left(\frac{p_2+q_2}{p_1}, \frac{q_2}{p_1}we^{-p_1 t}\right)+c}. \quad \text{(A.2.7)}$$

As $F_2(0) = 0$, we get

$$c = -\exp\left(-\frac{q_2}{p_1}w\right) - \frac{q_2}{p_1}(1-w)\left(\frac{q_2}{p_1}w\right)^{-(p_2+q_2)/p_1}\Gamma\left(\frac{p_2+q_2}{p_1}, \frac{q_2}{p_1}w\right).$$

Hence:

$$F_2(t) = 1 + \exp\left(-p_2 t - q_2 t - \frac{q_2}{p_1}we^{-p_1 t}\right) \cdot \left(\frac{q_2}{p_1}(1-w)\left(\frac{q_2}{p_1}w\right)^{-(p_2+q_2)/p_1}\left(\Gamma\left(\frac{p_2+q_2}{p_1}, \frac{q_2}{p_1}we^{-p_1 t}\right) - \Gamma\left(\frac{p_2+q_2}{p_1}, \frac{q_2}{p_1}w\right)\right) - \exp\left(-\frac{q_2}{p_1}w\right)\right)^{-1}. \quad \text{(A.2.8)}$$

As $w \to 0$, this expression for $F_2(t)$ reduces to the closed-form solution for the MIM.

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