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This study measures the degree of contagion or interpersonal influence in the diffusion of new consumer packaged goods (CPGs). The authors demonstrate that when an individual-level trial hazard model is properly specified to account for potential sources of biases, substantial contagion effects may be detected in the diffusion of many CPGs. Using longitudinal panel data on individual-level trial and repeat purchases of 67 newly introduced CPGs, they show that standard diffusion models fail to detect contagion. However, after extending the model to allow for spatial and temporal heterogeneity in contagion and controlling for various cross-sectional and temporal confounds, they find statistically significant contagion effects in 33 to 40 of the 67 sample products. The empirical evidence of contagion in the diffusion of many CPGs has important implications because most new product trial models for CPGs have assumed a priori that there is no contagion in the diffusion of these products. Moreover, the individual-level simultaneous analysis of the diffusion of 67 newly introduced CPGs provides useful insights into the unobservable network of influences among consumers. Such analysis allows a vendor to identify the most influential early adopters among its customers, who could help diffuse a new product more effectively in the market.

Keywords: consumer packaged goods, diffusion model, contagion, new product launch, temporal heterogeneity, spatial heterogeneity

Measuring Contagion in the Diffusion of Consumer Packaged Goods

Marketing researchers have long been interested in measuring contagion, or the interpersonal influence among members of the target market, in the diffusion of new products (Bass 1969). The vast majority of studies in this area have been carried out in the context of consumer durable goods (for a comprehensive survey of this literature, see Mahajan, Muller, and Wind 2000). The basic premise of contagion is simple: Consumers who have adopted a new product will affect the adoption decisions of those who have not through various channels of direct and indirect influence. This idea has led to the development and testing of many models that explicitly incorporate contagion as a driving force in shaping the diffusion curve (e.g., Mahajan, Muller, and Bass 1990, 1995; Parker 1994). In the diffusion literature, contagion effects are usually captured by modeling consumers' trial hazards (i.e., the likelihood of adoption at a particular time given that adoption has not yet taken place) as an increasing function of the cumulative number of adopters in the market.

In parallel with the development of the diffusion literature, which mostly focuses on consumer durable goods and explicitly allows for contagion effects, many models have been developed and tested for studying the penetration of new consumer packaged goods (CPGs), beginning with Fourt and Woodlock's (1960) work (for a comprehensive review, see Fader, Hardie, and Zeithammer 2003). With few exceptions, models in this literature (hereinafter referred to as the trial-purchase literature) have ignored contagion. Arguments for ignoring contagion in the diffusion of CPGs are mainly twofold. First, conceptually, the conventional wisdom has been that because most CPGs are low-uncertainty, low-risk, and low-involvement products, consumers make their trial decisions independently, regardless of what others may have communicated about the new product (Gatignon and Robertson 1985). Consequently, it has been argued that minimal word-of-mouth effects should be evident in trials

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of new CPGs (Hardie, Fader, and Wisniewski 1998). Second, empirically, researchers have noted that the cumulative adoption curves of CPGs are often concave rather than S shaped, which has long been viewed as a hallmark of contagion. In turn, such a lack of S-shaped aggregate diffusion curves has been interpreted as evidence for a lack of contagion in the diffusion of CPGs.

In contrast with the trial-purchase literature, which studies diffusion in the context of CPGs and ignores contagion a priori, we hold a more agnostic view on the influence early adopters may or may not have on nonadopters in the diffusion of CPGs. The theoretical rationale for our view is that information transmission through word of mouth or other forms of direct interpersonal interaction is a sufficient but not necessary condition for contagion to take place. For example, merely observing or being exposed to other consumers buying or consuming a new product can potentially influence a person's own trial decisions. Such indirect influence may exist because of signaling, bandwagon effect (i.e., the tendency to do things simply because many other people do the same), enhanced awareness, aided recall, or mere exposure effect (i.e., "familiarity breeds liking") (Bell and Song 2007; Burt 1987; Golder and Tellis 2004; Goolsbee and Klenow 2002; Van den Bulte and Lilien 2001). Regardless of the potential sources of contagion for CPGs (word-of-mouth communication, mere observation and exposure at the time of consumption, or even purchase), the behavioral outcome is the same: Consumers' trial decisions are influenced by those of prior adopters. We attempt to empirically determine the significance of the impact of such influence, if any, using a rich data set and a flexible modeling approach.

Empirically, we note that an S-shaped cumulative adoption curve is neither a sufficient nor a necessary condition for the existence of contagion in the diffusion process. For example, Jones and Mason (1990) demonstrate that an S-shaped cumulative adoption curve may be caused by growth in retail distribution in the first few weeks after product launch. In other words, without a properly specified model, S-shaped cumulative adoption curves may be mistakenly attributed to contagion. We argue that the reverse can also be true: Without a properly specified model, the lack of S-shaped cumulative adoption curves may be wrongly interpreted as a lack of contagion. In summary, our view is that the existence of contagion in the diffusion of CPGs should not be ruled out a priori; rather, it should be determined empirically, after carefully eliminating biases caused by various potential confounding factors.

If contagion does exist in the diffusion of many new CPGs, the managerial implication would be quite significant. By leveraging interpersonal influences among members of the target market, vendors of new CPGs may find more efficient avenues to speed up the diffusion process that would go beyond traditional mass advertising and price and trade promotions. For example, many large retail chains now collect shopper data through shopping clubs or loyalty cards; analysis of adoption patterns across all new CPGs introduced in the recent past can easily identify customers who are consistently among the early adopters of certain sets of products. If the diffusion of these new CPGs is also influenced by contagion and its impact can be measured at the individual level, as we demonstrate in this study, marketers of new CPGs can identify customers who not only are early adopters themselves but also can exert the most influence on others. By targeting early adopters who are also influencers, the firm not only ensures a faster recovery of its investments in new product development and introduction but also takes advantage of the social spillover effects in diffusing their new products. Anecdotal evidence suggests that some marketers of CPGs have already begun exploring such possibilities (e.g., marketing networks such as VocalPoint/Tremor, BzzAgent, and Shespeaks). The main goal of this study is to present a substantial body of empirical evidence, for the first time in the diffusion literature, regarding the existence or lack of contagion in the diffusion of new CPGs. We do so by examining the purchase histories of 5912 households over 124 weeks for a sample of 67 new CPGs. Our key challenge lies in devising an empirical testing strategy that enables us to minimize potential biases and confounds.

OVERVIEW OF OUR EMPIRICAL TESTING STRATEGY

In most diffusion models that allow for contagion, the trial hazard at any time point is modeled as an increasing function of the cumulative number of adopters in the market. Implicit in such a formulation are two key assumptions: First, temporally, the influence of any adopter will last forever, and second, cross-sectionally, every adopter will have the same influence on every nonadopter. Borrowing the terminology of Strang and Tuma (1993), we refer to these two assumptions as temporal homogeneity and cross-sectional homogeneity, respectively. We argue that both assumptions may prove invalid, especially in the context of CPGs.

The temporal homogeneity assumption disregards the notion that an adopter's influence may diminish over time. Intuition suggests that adopters should be more likely to "show and tell" right after the adoption than long afterward, when the novelty and excitement of the adoption wear off. In addition to diminishing postadoption contagion, which is likely to be true for any new product, the time window for contagion may be particularly short for CPGs because these products are consumed and disposed of much faster than durable goods, limiting the opportunity for contagion caused by exposure and observation. A way to allow for such a pattern of dwindling contagion is to limit the time window during which an adopter may influence nonadopters. The length of the "contagion window," R, determined empirically, indicates the extent to which contagion effects are temporary. The traditional way of modeling the effect of contagion as an increasing function of the cumulative number of adopters is equivalent to assuming an unlimited contagion window (i.e., $R \rightarrow \infty$). If the time window for contagion is truly temporary but its impact is assumed to last forever, the resulting empirical measure of contagion will be downward biased.

The cross-sectional homogeneity assumption (i.e., every adopter will have the same influence on every nonadopter) ignores the notion that the chances for direct and indirect interactions among consumers may not be the same across the population. For example, all other things being equal, we would expect an adopter to be more likely to influence his or her neighbors than those living across or out of town because, on average, neighbors should have more chances than nonneighbors to interact with each other through all sorts of venues, directly or indirectly. A way to allow for such

a pattern is to vary the influencer group of each person cross-sectionally, considering only the K-nearest neighbors of the focal consumer as potential influencers of his or her trial decisions. The number K, determined empirically, indicates the extent to which contagion effects are indeed local. The traditional way of modeling the effect of contagion, assuming that prior adopters located anywhere in the marketplace will influence the focal consumer equally, is equivalent to setting K to the size of the population, N. If an adopter's sphere of influence is truly local (i.e., $K << N$) but researchers model contagion as having global impact (i.e., $K = N$), the resulting empirical measure of contagion will again be downward biased.

Given that downward biases could potentially result from assuming temporal and cross-sectional homogeneity, especially in the context of CPGs, our proposed modeling framework relaxes these two key assumptions and instead determines empirically the extent to which interpersonal influences among members of the target market are temporary and local. In particular, instead of including all prior adopters in the influencer group, regardless of how far away they may live from the focal consumer or how long ago their most recent purchase may have taken place, we only consider the K-nearest neighbors of the focal consumer as potential influencers. Furthermore, for any given period, among the K-nearest neighbors, we include in the focal consumer's influencer group only those who have made at least one purchase in the most recent R periods. Note that our definition of the influencer group is individual, product, and time specific and consists of not only first-time purchasers but also repeat purchasers, in acknowledgment that most CPGs are bought and consumed with relatively high frequency and that both types of buyers can potentially influence trial decisions of the remaining nonadopters. Finally, because both R and K are determined empirically, our definition of the influencer group subsumes the standard definition of the influencer group in the diffusion literature (i.e., all prior adopters).

With this more flexible definition of the influencer group, the basic premise of our empirical testing strategy is as follows: If there is any contagion among consumers, the trial hazard of a nonadopter in any given period should be positively correlated with the size of his or her influencer group in that period. Intuitively, such positive correlation should manifest through two patterns in the observed diffusion data. Within a given neighborhood, all other things being equal, we should observe higher trial rates in periods when we observe more buyers of the new CPG in the recent past. Similarly, within a given period, all other things being equal, we should also observe higher trial rates of the new CPG in neighborhoods in which there were more recent buyers of the product. Thus, the existence of these temporal and spatial patterns in observed diffusion data can be viewed as empirical evidence that contagion may have played a significant role in the diffusion process.

THE CHALLENGE OF IDENTIFICATION

To implement the empirical testing strategy successfully with individual-level diffusion data, researchers must carefully address the challenge of identification, which arises because it is often difficult to separate mere correlation in observed behavior among consumers from genuine contagion effects caused by direct or indirect interpersonal influences. As Manski (1993) and Moffitt (2001) formally lay out, for any nonexperimental analysis of social interaction, the primary confounding factors include (1) endogenous group formation, (2) simultaneity, and (3) correlated unobservables. Hartmann and colleagues (2008) provide a review of these issues, and Nair, Manchanda, and Bhatia (2010) offer some potential solutions in the context of examining social interactions in physician prescription behavior.

For the current study, we argue that the first two confounding factors should be less of a concern because we simultaneously analyze adoptions of multiple new products within the same panel of consumers. In our context, endogenous group formation would be a confound only if people's decision of where to live (and, thus, the composition of influencer groups) was somehow affected by the introduction of new CPGs, which reason would deem improbable, especially because we examine multiple packaged goods spanning a diverse set of product categories. Similarly, simultaneity is a nonissue in our context because it would be a confound only if we could not determine from observed data whether purchases by members of the influencer group preceded trial decisions by the focal consumer. This is not a problem in our study, given how we define the influencer group (i.e., on the basis of decisions in the previous R periods) and given that our empirical analysis uses longitudinal individual-level data across multiple products.

In contrast, the third factor, correlated unobservables, can be a serious challenge in the context of our study. Many unknown or unknowable variables could affect both the trial hazard of a nonadopter in a period and the size of his or her influencer group (i.e., the number of recent purchasers among his or her nearest neighbors). Without properly controlling for these common underlying variables, researchers could misinterpret positively correlated trial hazards and recent purchasers among nearest neighbors as a sign of contagion effects when none actually exist. Next, we outline various potential sources of correlated unobservables, organized into three broad categories, along with a brief discussion of our strategies to control for them, which are essential to rule out the notion that the contagion effects we detect might be spurious.

Time-Invariant, Cross-Section-Variant Unobservables

These correlated unobservables vary across consumers but are constant over time. Some of them may be tied to inherent personal traits (e.g., a person's intrinsic willingness to try new things early), and others may be more product specific (e.g., high involvement with a particular product category or brand). Regardless of whether it is due to individual differences in inherent personal traits or product preferences, unobserved heterogeneity exists in people's baseline trial propensities toward a new product, independent of external influences, such as contagion and marketing. It is conceivable that consumers with high baseline trial propensities (hereinafter referred to simply as innovators) may live close to other innovators and those with low baseline trial propensities (hereinafter referred to simply as laggards) may be neighbors of other laggards. If that is the case, although people may not influence one another's adoption decisions, the positive correlation between physical proxim-

ity and proximity of adoption time would lead to spurious spatial contagion (i.e., the more of a person's neighbors adopt, the more that person is likely to adopt). This is a problem in studies measuring contagion from the diffusion pattern of a single new product, which makes it difficult (if not impossible) to isolate early adoptions driven by high baseline trial propensity from those caused by strong contagion from neighboring early adopters. However, when contagion is studied simultaneously across multiple products, such as in our study, it is possible to distinguish between heterogeneity in baseline trial propensity and the effects of contagion. This happens because the underlying factors that can drive consumers' intrinsic innovativeness or product preferences should simultaneously affect their baseline trial propensities toward multiple products. In other words, these factors, which may or may not be correlated across space, should lead to trial decisions that are correlated across products. Thus, by tapping into the manifested pattern of correlation in consumers' trial behavior across multiple products, we can infer the common underlying factors, which in turn can serve as controls for unobserved heterogeneity in baseline trial propensities, enabling us to more effectively separate spatially correlated time-invariant, cross-section-variant unobservables from the effects of spatial contagion.[1]

Time-Variant, Cross-Section-Invariant Unobservables

These correlated unobservables represent any temporal trends that can drive both trial hazard and recent purchasers among nearest neighbors (i.e., the size of the influencer group). Without proper control, it is possible to misinterpret temporal covariation between trial rates and recent purchasers among nearest neighbors as a result of contagion. To address this issue, our proposed model incorporates two controls. The first is to make trial hazard a function of time, both linear and log-linear, with the log-linear part capturing potentially nonlinear changes in the initial postlaunch periods. The second control is to include the number of recent purchasers among nonneighbors of the focal household because any time-variant, cross-section-invariant variable should, by definition, have the same impact on the number of recent purchasers among both neighbors and nonneighbors. Thus, including recent purchasers among nonneighbors provides a strong control for time-variant, cross-section-invariant confounds.

Time-Variant, Cross-Section-Variant Unobservables

This is the most difficult confound to control because no study can rule out all possibilities. Differential product availability (e.g., products launched at different stores at different time points) is one example of this potential confound. In our case, we were fortunate to have data on store rollout and individual trial store information to account for this confound explicitly. Other examples include price and promotions, which vary across shoppers depending on where and when they make a purchase. Not accounting for these marketing efforts may show spurious contagion effects. For example, in an extension of Burt's (1987) replication of Coleman, Katz, and Menzel's (1966) classic study on the diffusion of tetracycline, Van den Bulte and Lilien (2001) demonstrate that the contagion effects detected in previous studies disappeared when they controlled for marketing efforts. We also have data to account explicitly for these potential confounds as observables. Other possible time- and cross-section-variant confounds include advertising and competition, which are truly unobservable in our particular application. However, given that our study involves a compact midsize metropolitan area dominated by the focal grocery chain, it is not unreasonable to assume that, on average, neighbors and nonneighbors of each household living in the same metropolitan area are exposed to the same media and share more or less a homogeneous shopping environment offered by the same retail chain. Consequently, buyers among neighbors and nonneighbors are under the influence of these same unobservable confounds, and therefore the inclusion of recent purchasers among nonneighbors in the hazard function provides a good control for most, if not all, advertising and competition that may also have a similar impact on recent purchasers among neighbors.

In summary, our empirical testing strategy fully recognizes potential threats to the identification of contagion effects and addresses them systematically, following the treatments outlined previously. Because of the richness of our data (geocodes of household residence to separate neighbors from nonneighbors, detailed rollout and trial store information to control for spatial heterogeneity in availability, price and promotion data, and individual purchase history across dozens of new product introductions spanning multiple product categories) and the flexibility and robustness of our modeling framework, we ensure that whatever contagion effects we detect empirically have ruled out alternative explanations. In the next section, we present a multiproduct individual-level diffusion model that takes into account all the potential sources of biases in measuring contagion discussed previously, including the following:

- *Temporal heterogeneity*: We consider the possibility that only adopters who have recently tried or repurchased the product can potentially influence nonadopters, which has been ignored in the marketing literature.
- *Spatial heterogeneity*: We consider that only adopters who are among the nearest neighbors might exert influence on a nonadopter, leading to local (rather than global) contagion effects, which have been ignored in aggregate diffusion models.
- *Correlated unobservables*: We address these confounds by (1) estimating the diffusion model across multiple products to rule out time-invariant, cross-section-variant unobservables; (2) incorporating time trends (linear and log-linear) to account for time-variant, cross-section-invariant unobservables; (3) considering the effects of price and sales promotions to account for time- and cross-section-variant unobservables; and (4) incorporating purchasers among nonneighbors to account for time-variant unobservables.
- *Unobserved individual differences in baseline trial propensity*: An important and critical distinction between our empirical testing strategy and extant literature is that we take advantage of information obtained from the same consumers across dozens of new product introductions spanning multiple categories, which enables us to infer baseline trial propensity at the individual level by tapping into the pattern of cross-product correlation in trial behavior. This enables us to more effectively

[1]Separating contagion from this type of potential confounds is only possible when the analysis is based on adoption behavior observed across a large number of new products. Moreover, the products included in the analysis must be related as a group, in the sense that it is reasonable to assume that their purchases are influenced by a common set of consumer-specific factors.

disentangle spatial correlation in unobserved individual baseline trial propensities from the effects of spatial contagion. In contrast, most previous attempts to measure contagion at the individual level are based on trial history of a single new product and thus depend on only one observed spell per consumer to identify contagion and rule out alternative explanations.

After presenting our proposed individual-level diffusion model, we apply it to purchase data for 67 newly introduced CPGs, obtained from a panel of 5912 grocery shoppers in one midsize metropolitan area over 124 weeks. We calibrate several versions of the proposed model, including the classic discrete-time proportional hazard model not allowing for contagion, which is nested under our more general formulation, to investigate how the potential sources of biases mentioned previously affect the measurement of contagion in the diffusion of CPGs. To validate our empirical testing strategy further, we compare the predictive performance of our proposed model with that of the well-known exponential-gamma hazard model with covariates that Fader, Hardie, and Zeithammer (2003, p. 395) recommend, which incorporates all the predictors considered in our proposed model, except for the covariates related to contagion.

Because we model individual-level adoption behavior among a defined population across multiple new products spanning multiple categories, we can measure the degree of implicit influence each consumer exerts on all others within the same geographic market. We use this implicit influence measure to construct an adjacency matrix that reflects the network of influences derived from our empirical measure of contagion, which in turn enables us to study the implicit social network manifested through the spatial and temporal patterns of diffusion of the 67 products in our sample. Analysis of this implicit network, along with individual-level estimates of baseline trial propensities obtained from our model, enables us to identify the innovators for specific products, as well as those who are more likely to exert influence on others. Targeting influential innovators can lead to more effective diffusion of new products in the future.

AN INDIVIDUAL-LEVEL TRIAL HAZARD MODEL FOR MULTIPLE NEW PRODUCTS

We follow the common practice in the literature to model individual adoptions with a discrete-time proportional hazard model (Greve, Strang and Tuma 1995; Van den Bulte and Lilien 2001), where the trial hazard (λ_{ijt}) represents the likelihood that consumer i, who has not yet tried product j at the beginning of the *t*th postlaunch period, will do so in that period. In particular, we formulate the log-hazard rate as follows:

$$\text{(1)} \quad \ln(\lambda_{ijt}) = \alpha_{ij} + \beta_j X_{ijt} + \delta_{1j} t + \delta_{2j} \ln t + \gamma_j N_{ijt}^{RK} + \theta_j M_{ijt}^{RK},$$

where α_{ij} = the baseline propensity of consumer i adopting product j, which can be a function of various time-invariant factors associated with consumer i and product j (e.g., the intrinsic innovativeness of consumer i, or i's preference for the brand of product j);

X_{ijt} = marketing efforts (e.g., price discounts, display, and feature promotions) received by consumer i from product j in the *t*th postlaunch period;

β_j = consumers' responsiveness to product j's marketing efforts in making their trial decisions;

δ_{1j} and δ_{2j} = linear and nonlinear time trends, respectively, in the log-hazard rate. Other things being equal, positive (negative) δ_j indicate increasing (decreasing) trial propensity over time;

N_{ijt}^{RK} = the number of prior adopters who can potentially influence consumer i's trial of product j in period t. Unlike traditional diffusion models, which treat all prior adopters as potential influencers of consumer i in period t, our formulation includes in the influencer group of consumer i in period t only the K-nearest neighbors of consumer i who bought product j at least once in the R periods before t (excluding t). R and K are determined empirically;

γ_j = the contagion coefficient for product j, indicating the extent to which consumers' trial hazards in a period are tied to the sizes of their influencer groups in that period. If contagion does play a role in the diffusion of product j, γ_j should be positive and statistically significant;

M_{ijt}^{RK} = the number of non-K-nearest neighbors of consumer i who bought product j at least once in the R periods before t (excluding t). We include M_{ijt}^{RK} in the hazard function as a control for many time-variant correlated unobservables. For example, advertising in mass media may simultaneously drive up λ_{ijt} and N_{ijt}^{RK}. If advertising is not explicitly included in Equation 1, the estimate of γ_j would be biased upward, creating an illusion of contagion. Thus, M_{ijt}^{RK} serves as a strong control in such a situation, to the extent neighbors and nonneighbors of consumer i are, on average, exposed to more or less the same media environment; and

θ_j = the coefficient for the number of recent purchasers among nonneighbors (M_{ijt}^{RK}). If any interpersonal influence consumer i receives comes predominantly from recent purchasers among his or her K-nearest neighbors (i.e., contagion is indeed local), M_{ijt}^{RK} should have no positive impact on consumer i's trial hazard. In other words, an insignificant or negative θ_j, in contrast with a positive and significant γ_j, would provide strong evidence of discriminant validity for the hypothesis that contagion exists and is local because otherwise (i.e., if contagion turns out to be nonexistent or global) we would expect both the γ_j coefficient (on neighbors) and the θ_j coefficient (on nonneighbors) to be of similar magnitude and direction.

The trial hazard rate specified in Equation 1 is agnostic about the mechanisms through which contagion occurs. Contagion may arise because of any combination of direct and indirect interpersonal influences a nonadopter may receive from prior adopters, including (but not limited to) word of mouth, signaling, bandwagon effects, enhanced awareness, aided recall, and mere exposure effect. Equation 1 assumes only that the degree of contagion, if any, should be reflected in a hazard rate that is positively correlated with

the number of recent purchasers among nearest neighbors (N_{ijt}^{RK}), because as N_{ijt}^{RK} increases, so should the chances for direct and indirect interpersonal influences. Therefore, a positive and significant estimate of γ_j can be interpreted as the presence of interpersonal influence when consumers make their trial decisions for product j.

As we discussed previously, allowing contagion to be temporary and local is particularly important in the context of CPGs. To implement this condition, both R and K in Equation 1 must be determined empirically. If a small R turns out to provide the best fit, it would indicate that the time window for contagion is indeed temporary. As R approaches the full observation window, Equation 1 degenerates to the traditional assumption of permanent influence, or temporal homogeneity. Similarly, if a small K turns out to fit best, it would indicate that the sphere of interpersonal influence is indeed local. As K approaches the full population size (N), Equation 1 degenerates to the traditional assumption of global influence, or spatial homogeneity.

As the spatial statistics literature indicates, "neighbors" can be defined in many ways. Our choice of the K-nearest formulation results in a fixed sample of potential influencers for each focal consumer. Such a variable-bandwidth (Fotheringham, Brunsdon, and Charlton 2002) form of geographic weighting works more effectively when the population is unevenly dispersed geographically, as is commonly observed in the United States. Alternative formulations include treating the spatial pattern of contagion as a distance-weighted average (e.g., Greve, Strang, and Tuma 1995; Strang and Tuma 1993), which tends to oversample (undersample) areas with high (low) population density, and allowing contagion to take place only among contiguous regions (e.g., Bell and Song 2007; Bronnenberg and Mela 2004), which limits the extent of spatial influence to immediate adjacency. Another reason for the K-nearest neighbors formulation is pragmatism: The distance-weighted average would be infeasible for large sample sizes such as the one we use.

As we discussed previously, any nonexperimental analysis that attempts to infer interpersonal influence from observed behavior outcome data must confront many potential confounding factors. In our context, unobservables correlated with both the hazard rate λ_{ijt} and the size of the influencer group N_{ijt}^{RK} pose the most serious threat; some of these unobservables can be time variant, cross-section variant, or both. We argue that the variables included in Equation 1 can rule out most, if not all, known confounds. For example, making the trial hazard a function of time t and ln(t) provides control over correlated unobservables that are time variant and cross-section invariant. Other time-variant and cross-section-invariant variables can be controlled for by the number of recent purchasers among nonneighbors, M_{ijt}^{RK}. Finally, variables in X_{ijt} are potentially good controls for many unobserved (to the researcher) marketing efforts that are time and cross-section variant, to the extent that they are correlated with X_{ijt}.

The α_{ij} term in Equation 1 deserves more discussion. All other things being equal, higher α_{ij} indicates that consumer i has greater baseline propensity to try product j, independent of external influences such as contagion (N_{ijt}^{RK}) and marketing (X_{ijt}). Making it consumer specific takes into account heterogeneity in consumers' baseline trial propensities, which also provides some control over correlated unobservables that are time invariant but cross-section variant, as we discussed previously. However, because each consumer adopts a product at most once, α_{ij} is not identifiable as a fixed effect and must be treated as a random effect. An alternative is to let $\alpha_{ij} = \alpha_j + \varepsilon_{ij}$, with $\varepsilon_{ij} \sim N(0, \sigma_j^2)$, where α_j represents the average baseline trial propensity, ε_{ij} is the random deviation of consumer i, and σ_j is the extent of heterogeneity.

A key assumption in formulating α_{ij} as $\alpha_j + \varepsilon_{ij}$ with $\varepsilon_{ij} \sim N(0, \sigma_j^2)$ is that ε_{ij} would be uncorrelated with ε_{ik} for $k \neq j$. Such an assumption implies that consumers' baseline propensity to adopt one product is completely independent of their baseline propensity to adopt other products, which is unlikely given that various underlying factors (e.g., category involvement, intrinsic innovativeness) could lead consumers to behave similarly when it comes to adopting many related or seemingly unrelated new products. When the adoption history of multiple new products is available, as is the case in our study, ignoring potential cross-product correlation in adoption behavior would provide inefficient use of the data. To tap into such correlation, we relax the assumption of independence and explicitly model the potential correlation between ε_{ij} and ε_{ik} for $k \neq j$. Doing so also means that the hazard functions of all products included in the analysis must be calibrated simultaneously. However, this would be infeasible when the number of sample products, J, is large (in our case, J = 67) and it is necessary to estimate the full variance–covariance matrix of $\varepsilon_{ij} \sim N(0, \Sigma_{J \times J})$, which has $J \times (J + 1)/2$ variance and covariance terms (2278 parameters in our case) to be estimated.

To strike a balance among feasibility, efficient use of data, and flexibility, we impose the following latent-factor structure on the random-effect term α_{ij}:

$$\alpha_{ij} = \alpha_j + \varepsilon_{ij} = \alpha_j + \Lambda_j Z_i, \quad (2)$$

where Z_i is a P-dimensional random vector, with each element distributed i.i.d. standard normal, and Λ_j is a P-dimensional parameter vector to be estimated for product j. Formally, Equation 2 is equivalent to assuming $\alpha_{ij} = \alpha_j + \varepsilon_{ij}$ with $\varepsilon_{ij} \sim N(0, \Sigma_{J \times J} = \Lambda_{J \times P}\Lambda_{J \times P})$, which has $J \times P$ parameters to be estimated, rather than $J \times (J + 1)/2$. More intuitively, Equation 2 states that each consumer may face certain latent factors Z_i that underlie their baseline trial propensities toward various new products. Depending on the product, Z_i can lead to above-average (if $\varepsilon_{ij} = \Lambda_j Z_i > 0$) or below-average (if $\varepsilon_{ij} = \Lambda_j Z_i < 0$) baseline trial propensity. New products with similar Λs will induce the same group of consumers to adopt early; consumers with similar Zs would manifest similar trial behavior across products.

With the formulation of α_{ij} given in Equation 2, Equation 1 can be rewritten as follows:

$$\ln(\lambda_{ijt}) = \alpha_j + \Lambda_j Z_i + \beta_j X_{ijt} + \delta_{1j} t + \delta_{2j} \ln t + \gamma_j N_{ijt}^{RK} + \theta_j M_{ijt}^{RK}, \quad (3)$$

where Λ and its dimensionality P are empirically determined, along with the other model parameters α, β, δ, γ, and θ. What helps identify Λ, or the latent factor structure, is the main cross-product correlation patterns in consumers' trial behavior.

With a complementary log-log link function and consumer i's log-hazard rate for product j in discrete time period t, $\lambda_{ijt} = \exp(\alpha_j + \Lambda_j Z_i + \beta_j X_{ijt} + \delta_{1j} t + d_{2j} \ln t + \gamma_j N_{ijt}^{RK} + \theta_j M_{ijt}^{RK})$, the likelihood contribution of consumer i condi-

tional on Z_i, given his or her trial history for a sample of J products and other observables during the observation window, can be written as follows:

$$(4)\quad L_i\left(\alpha, \Lambda, \beta, \delta, \gamma, \theta \mid Z_i; O_i, t_{ij \in O_i}, t_{j \notin O_i}, X_i, N_i, M_i\right) = \prod_{j \in O_i}\left\{\left[1 - \exp\left(-\lambda_{ijt_{ij}}\right)\right]\prod_{l=1}^{t_{ij}-1}\exp\left(-\lambda_{ijl}\right)\right\} \times \prod_{j \notin O_i}\left[\prod_{l=1}^{t_j}\exp\left(-\lambda_{ijl}\right)\right],$$

where O_i denotes the set of products adopted by consumer i, t_{ij} denotes the period during which trial took place for consumer i since product j was introduced, and t_j denotes the total window of observation for product j. Note that because Z_i is unobserved and distributed i.i.d. standard normal, it must be integrated out across P dimensions for model estimation. To simulate the P-dimension integration over Z_i, we use a unique Halton sequence for each dimension of Z_i and assign a different set of draws for each individual i. Furthermore, because Z_i, as a vector of latent individual-level random factors, enters the hazard rate of every sample product, parameters for all J products must be obtained simultaneously (thus tapping into not only the diffusion history of each product independently but also the cross-product correlation pattern in trial behavior). When the model is calibrated using the simulated maximum likelihood estimator, an estimate of Z_i can be derived conditional on the model parameters and consumer i's trial history data and other observables. Such an estimate can then be used to identify innovators who have high baseline trial propensities toward certain types of products. For more on how to simulate likelihood functions that involve multidimension integration with draws from Halton sequences, see Train (2003), who also discusses how to obtain posterior estimates of Z_i.

In summary, our proposed trial hazard formulation (Equation 3) allows for the notion that contagion may be temporary (R) and local (K). Meanwhile, it controls for several major sources of confounding factors that are time variant (X_{ijt}, t, ln(t), and M^{RK}_{ijt}) or cross-section variant ($\alpha_{ij} = \alpha_j + \Lambda_j Z_i$). If interpersonal influence does exist in the diffusion of product j, the contagion coefficient (γ_j) should be positive and significant. By examining a large sample of products ($j \in J$), we should be able to establish a substantial body of empirical evidence regarding whether contagion plays a significant role in the diffusion of new CPGs.

Network Effects

If contagion turns out to play a significant role in the diffusion of new CPGs, vendors may target consumers who not only try new products early (i.e., large $\Lambda_j Z_i$) but also have demonstrated in the past to have a disproportionately large influence on others' adoptions. Targeting those influential innovators and converting them into early buyers enable marketers to benefit from their high willingness to try (leading to a faster recovery of product development and launch investments), as well as a spatial spillover effect on marketing efforts.

After our proposed model (Equation 3) has been calibrated and the degree of contagion (γ_j) has been estimated for each product, researchers can use these estimates to infer the extent to which each consumer exerted influence on every other consumer in the past across the multiple new products included in the calibration sample. For example, suppose Consumer A has purchased product j within R periods before Consumer B's adoption of the same product, and A is one of B's K-nearest neighbors. Then, according to our model, the influence of Consumer A had the effect of increasing the hazard rate at the time of Consumer B's trial of product j by $\exp(\gamma_j) - 1$.

Following this rationale, the effective influence of Consumer A on B's adoption decisions can be measured as the increase in the hazard rate that is attributable to contagion from Consumer A, summed across all the products adopted by Consumer B (O_B):

$$(5)\quad W_{AB} = \sum_{j \in O_B}\left\{\exp\left[R_{jAB}\left(\gamma_j K_{AB}\right)\right] - 1\right\},$$

where R_{jAB} is 1 if Consumer A has purchased product j at least once in the R periods before Consumer B's trial of the same product and 0 if otherwise, and K_{AB} is 1 if Consumer A is among the K-nearest neighbors of Consumer B and 0 if otherwise.

Computation of W in Equation 5 for every pair of consumers produces a directed adjacency matrix that can be represented as a network of effective influences among all consumers, which can then be subjected to exploratory social network analyses. Because we are interested in understanding the patterns of influence among all consumers and the resulting pattern of contagion through the implicit social network, we focus on the two most common measures of influence, or centrality (for details, see Wasserman and Faust 1994), within a network.

Out-degree centrality, or the sum of links arising from a consumer, indicates the extent to which the consumer directly influences others. When introducing a new product, marketers might want to pay special attention to consumers with high out-degree centrality because they directly influence other consumers, thus providing "free" and effective promotion for the new product. Out-degree centrality for consumer i in our case can be easily computed as $OC_i = \Sigma^N_{i'=1} W_{ii'}$, where $W_{ii'}$ is defined as in Equation 5.

Eigenvector Centrality

The measure of out-degree centrality (OC_i) described previously takes into account only the direct influence of consumer i on others, without considering that consumers influenced by i can also exert influence on others, so that the overall (direct plus indirect) influence exerted by consumer i will be greater than the direct influence captured by out-degree centrality. The main purpose of the eigenvector centrality measure is to assign a centrality score to a consumer that reflects not only the influence of this consumer on those directly connected to him or her but also the indirect influence exerted through the network of connections. If we define the eigenvector score as x, the measure for consumer i would be proportional to the weighted sum of the centrality scores of the consumers to which he or she is connected (i.e., $\lambda x_i = \Sigma^N_{i'=1} w_{ii'} x_{i'}$). These relationships across all consumers can be written in matrix form as $\lambda X = WX$, where X can be obtained as the first eigenvector solution of $(\lambda I - W)X = 0$, where I represents a diagonal identity matrix. Bonacich and Loyd (2001) present details about this and other measures of centrality. This particular eigenvector measure is only applicable to asymmetric relationships when each consumer in the network has at least one outer connec-

tion. Because our empirical illustration focuses on the main component of the network of influences implied by our model, we decided to use this simpler version of centrality.

EMPIRICAL ILLUSTRATION AND TESTS OF THE PROPOSED NEW PRODUCT TRIAL MODEL

Data Description

To implement our empirical testing strategy, we use longitudinal panel data provided by a major grocery chain in the United States. Households included in the panel all reside in one midsize metropolitan area in the southeastern United States, which has a total area of approximately 75 square miles and an estimated population of more than 130,000 (as of 2007). In the area under study, the focal grocery chain operates eight stores and is by far the dominant vendor of grocery products in the region (in contrast, the second largest grocery retailer in the area operates only one store in the region). The focal chain runs a popular frequent-shopper program, which has a penetration rate of more than 90% in the area under study and accounts for nearly 95% of the chain's total sales in the region; this ensures that our sample provides comprehensive coverage of the target population.

Our sample includes 5912 households, whose purchase history of 67 new CPGs launched in the first 50 weeks of a 124-week observation window is available. The average length of observation for the 67 new products is 107 weeks. On average, each household tried 8.3 of the 67 sample products. Table 1 presents the frequency distribution of sample households in terms of number of products tried during the observation window. In addition, we have weekly data on these products' display and feature activities, as well as promoted prices (i.e., regular price less any discount received by the customer). Table 2 presents descriptive information about these new products and the associated marketing mixes. (For confidentiality, we can only provide information about the categories to which the new products belong.)

Table 1
FREQUENCY DISTRIBUTION OF SAMPLE HOUSEHOLDS IN NUMBER OF PRODUCTS TRIED

Number of Trials	*Frequency*	*Percentage*	*Cumulative Percentage*
0	62	1.05	1.05
1	157	2.66	3.70
2	289	4.89	8.59
3	379	6.41	15.00
4	447	7.56	22.56
5	529	8.95	31.51
6	556	9.40	40.92
7	563	9.52	50.44
8	456	7.71	58.15
9	415	7.02	65.17
10	381	6.44	71.62
11	331	5.60	77.22
12	297	5.02	82.24
13	211	3.57	85.81
14	196	3.32	89.12
15	134	2.27	91.39
16	116	1.96	93.35
17	99	1.67	95.03
18	75	1.27	96.30
19	55	.93	97.23
20	42	.71	97.94
21	30	.51	98.44
22	27	.46	98.90
23	19	.32	99.22
24	14	.24	99.46
25	12	.20	99.66
27	4	.07	99.73
28	7	.12	99.85
29	1	.02	99.87
30+	8	.14	100.00

The coefficient of variation for price, defined as the ratio of the standard deviation to the average of weekly prices, indicates the degree of price variation for each product. In terms of weekly display and feature activities, because they are highly collinear for many of our sample products, we use a combined index to capture the intensity of these marketing efforts. Finally, for each household, in addition to weekly purchase records of the 67 sample products throughout the observation window, we have their geocodes (longitude and latitude), plotted in Figure 1, which enable us to calculate the geodistances between any two households, a necessary element for capturing spatial heterogeneity in contagion.

In applying the proposed trial hazard model to our particular data set, we stress a critical potential confound: differential availability.[2] Even for the same grocery chain in the same metro area, we cannot assume that new products will be available to all consumers at the same time. Without proper control, such differential availability could pose a serious confound because all purchases, trial or repeat, are conditional on products being available. In other words, when availability is time and cross-section variant, it can positively correlate with both λ_{ijt} and N_{ijt}^{RK}—thus, the threat of spurious contagion. Unfortunately, most previous attempts to measure contagion in the diffusion literature have largely ignored the differential availability issue.

In the current study, several unique aspects of our empirical setting should help minimize this potential confound. First, we have access to the following three pieces of information: (1) the week in which each new product was launched at each of the eight sample stores; (2) for a household that adopted a product during the observation window, the week and the store in which the household made the trial purchase; and (3) the "favorite" store of each sample household, as designated by the focal chain.

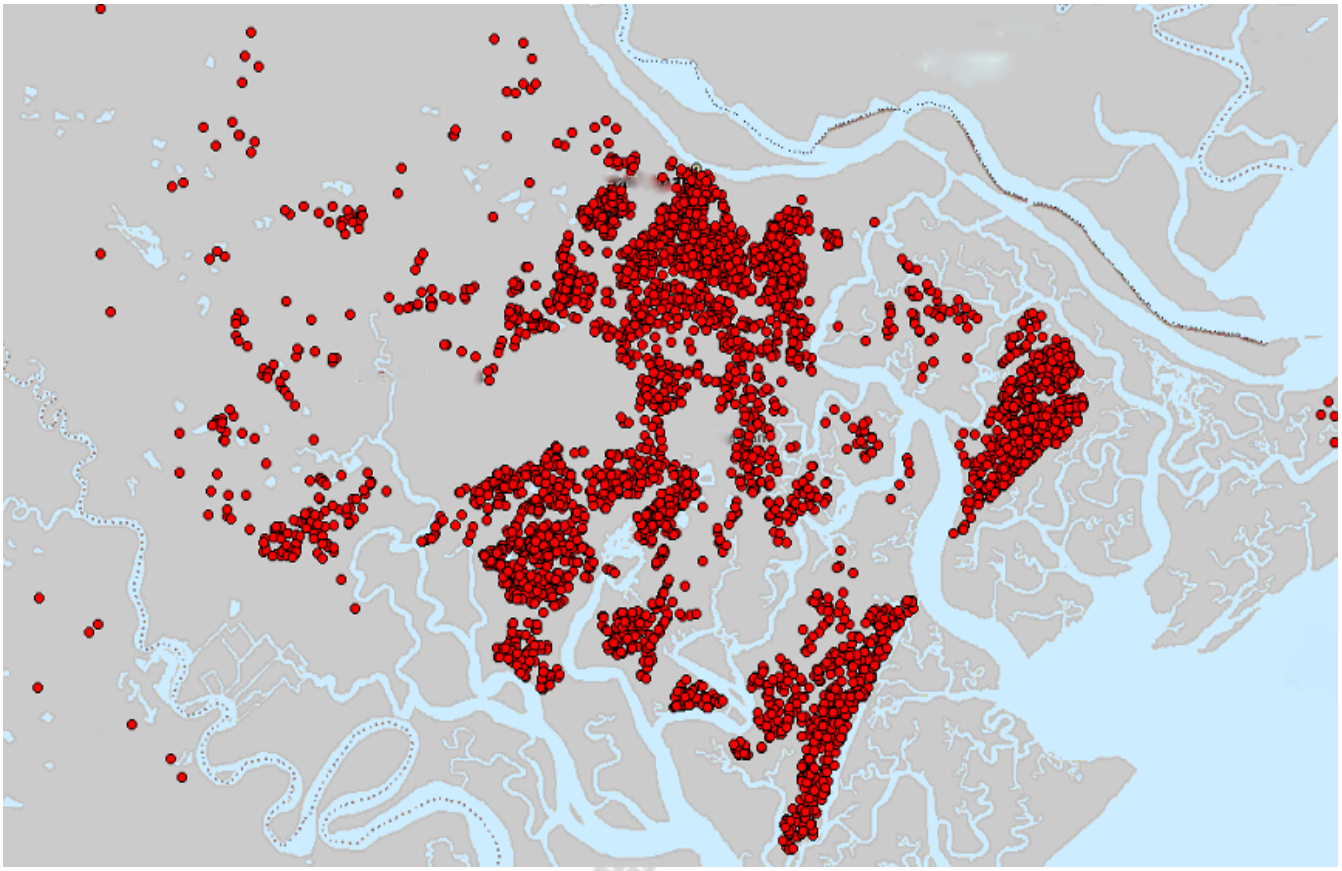
Equipped with these data, for an adopter, we define time to trial (t_{ij} in Equation 4) as the number of weeks passed before adoption since the new product was introduced at the store in which the trial purchase actually took place. Similarly, for an adopter, we use marketing-mix information (X_{ijt}) from the actual trial store. However, for a nonadopter, because by definition no trial took place (and, therefore, no trial store), we define time since launch as the number of weeks passed since the new product was introduced at his or her favorite store. In addition, for a nonadopter, we use marketing-mix information from his or her favorite store. Although this is admittedly not a perfect solution for the nonadopters (because a household may visit nonfavorite stores), we note that according to our data provider, a shopper's favorite store accounts for, on average, more than eight of every ten of his or her transactions. (This is further corroborated by our finding that 86% of the trial purchases

[2]We thank the anonymous reviewers for alerting us to this important issue.

Table 2
DESCRIPTIVE INFORMATION ABOUT THE NEW PRODUCT SAMPLE

Product	*Length of Observation*	*Penetration Rate (%)*	*Average Time to Trial (Among Adopters)*	*Coefficent of Variation for Price*	*Display/ Feature Index*
BAKERY	90	10.1	35	.01	.00
BAKERY	124	9.7	44	.03	.00
BAKING MIXES	102	10.9	42	.03	.01
BAKING MIXES	102	8.7	44	.03	.00
CANDY	110	20.3	42	.19	.26
CANDY	109	19.0	48	.19	.28
CANDY	108	14.5	39	.17	.13
CANDY	109	13.8	46	.18	.27
CANDY	110	13.6	43	.18	.24
CANDY	102	11.5	43	.19	.19
CANDY	80	9.8	22	.24	.23
CANDY	112	9.5	40	.24	.13
CANDY	110	9.1	48	.17	.26
CHARCOAL	76	13.0	31	.11	.30
CHARCOAL	76	8.1	34	.03	.08
CKY/CRKR/SNK	124	29.9	40	.05	.29
CKY/CRKR/SNK	124	29.9	40	.05	.29
CKY/CRKR/SNK	108	27.0	44	.15	.20
CKY/CRKR/SNK	104	14.5	49	.21	.21
CKY/CRKR/SNK	120	15.2	44	.00	.01
CKY/CRKR/SNK	120	10.2	42	.04	.07
CKY/CRKR/SNK	120	9.4	57	.18	.17
CNV BREAKFAST	103	9.4	42	.07	.08
CONDIMENTS & SAUCES	118	19.4	49	.14	.01
CONDIMENTS & SAUCES	118	19.0	54	.17	.01
FACIAL TISSUE & NAPKIN	111	9.9	46	.16	.09
FACIAL TISSUE & NAPKIN	111	6.2	51	.17	.06
FACIAL TISSUE & NAPKIN	111	8.9	46	.16	.04
FACIAL TISSUE & NAPKIN	113	8.6	44	.17	.03
FROZEN GROCERY	118	13.1	48	.05	.12
FROZEN GROCERY	119	10.9	44	.05	.09
FROZEN GROCERY	88	9.0	37	.15	.11
FROZEN GROCERY	121	9.1	47	.10	.07
FROZEN GROCERY	121	8.3	51	.09	.03
HISPANIC FOODS	114	8.7	46	.10	.00
NEW AGE	121	9.3	52	.18	.11
NUTS	89	11.6	36	.11	.31
PKG MEAT	109	15.0	37	.11	.03
PKG MEAT	112	11.9	51	.19	.03
PKG MEAT	111	11.3	45	.11	.04
PKG MEAT	110	9.8	45	.11	.04
PKG MEAT	111	9.4	43	.11	.04
PKG MEAT	111	9.4	45	.11	.04
PKG MEAT	114	8.2	51	.14	.06
PKG MEAT	101	8.8	42	.13	.06
PKG MEAT	119	8.7	52	.15	.03
REFRIG GROCERY	121	10.3	56	.14	.08
REFRIG GROCERY	102	10.4	47	.14	.09
REFRIG GROCERY	92	8.8	50	.15	.04
REFRIG GROCERY	100	8.3	40	.16	.10
REFRIG GROCERY	102	8.5	44	.10	.02
SALAD MIX	124	30.3	47	.13	.15
SALAD MIX	112	27.2	45	.11	.09
SALAD MIX	123	25.3	53	.12	.15
SALAD MIX	100	15.2	40	.18	.10
SALAD MIX	112	15.0	44	.12	.15
SALAD MIX	100	11.1	38	.18	.08
SHELF STABLE VEGETABLES	87	9.6	40	.14	.03
SHELF STABLE VEGETABLES	88	8.4	38	.17	.05
SOFT DRINKS	106	14.8	35	.16	.56
SOFT DRINKS	110	14.6	31	.14	.43
SOFT DRINKS	87	13.5	27	.03	.01
SOFT DRINKS	106	10.0	44	.03	.00
SOFT DRINKS	105	9.1	45	.14	.39
SOFT DRINKS	108	9.1	40	.16	.21
TEAS	120	11.4	51	.10	.01
TURKEY GRINDS	78	9.8	26	.14	.03
YOGURT	108	9.2	43	.10	.06

Figure 1
GEODISTRIBUTION OF SAMPLE HOUSEHOLDS



observed in our data took place in the adopters' favorite stores.[3])

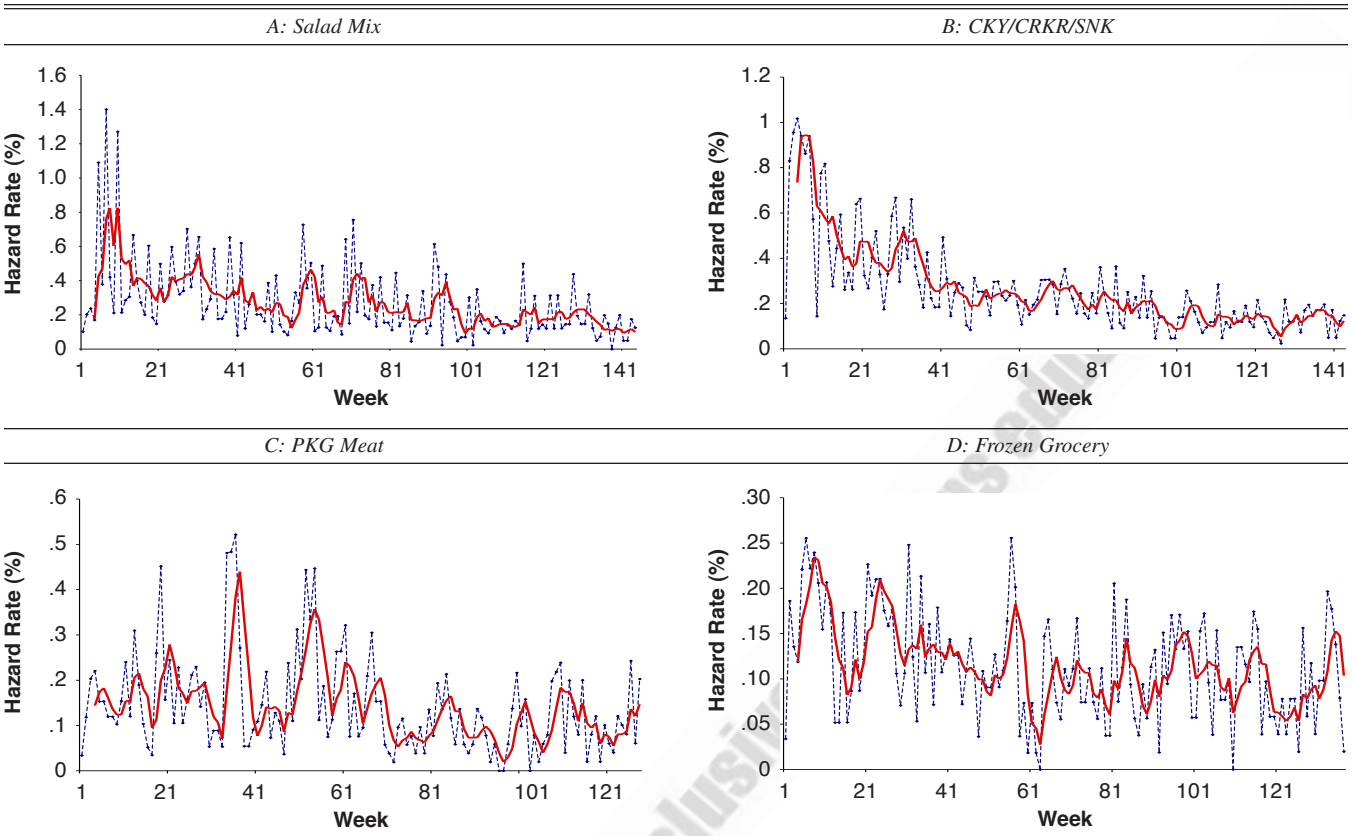
In addition, we note that the market under study is a mid-size metropolitan area, in which the focal chain dominates with eight stores, all located within a ten-mile radius. According to the focal chain, these eight stores use similar planograms and are served by the same set of distribution centers. Not surprisingly, after examining the between-store launch time differences for the 67 products in our sample, we found that the median of maximum launch-time differences (i.e., the gap between the first and the last store launches) is 5 weeks and the average is 8.5 weeks. The vast majority (56) of the new products in our sample were rolled out across the market under study within less than two months. For all practical purposes, this shows a lack of real heterogeneity in distribution time in our case, which, combined with our consideration of product availability differences by store and household (as discussed previously), should render the potential impact of differential availability inconsequential on our results. Finally, we checked and found that all the sample products, after being launched at a store, had been continuously available at the store throughout the observation window.

Given our proposed model, for a particular R and K, we identify the contagion coefficient (γ_j) by tapping simultaneously into cross-sectional and temporal patterns embedded in the diffusion data, after taking into account α_{ij} (heterogeneity in baseline trial propensity), X_{ijt} (observed marketing efforts), t (linear and log-linear time trends), and M_{ijt}^{RK} (any time-variant correlated unobservables that have more or less the same impact on the neighbors and nonneighbors of consumer i). Temporally, contagion is present if higher adoption rates among nonadopters of a new product j are observed in periods when there were more buyers of the product in the previous R periods. Similarly, within a particular period and according to cross-sectional variation, contagion is present if higher adoption rates are observed among nonadopters whose K-nearest neighbors include a larger number of buyers of the new product j in the previous R periods.

Figure 2 shows weekly aggregate empirical hazard rates for four randomly selected products, calculated by dividing the number of new triers in week t by the number of nontriers up to the end of week t – 1. Figure 3 presents cumulative adoption rates for 12 sample products in two categories. At first glance, these longitudinal aggregate rates do not seem to indicate the existence of contagion, because contagion is often associated with increasing hazard rates over time,

[3]The "perfect" solution would require information at the individual shopping trip level (e.g., the day each trip took place, at which store, whether the new product was in stock in that store on that day). We consider that beyond the scope of this project and leave it for further research.

Figure 2
WEEKLY TRIAL HAZARD RATES OBSERVED FOR FOUR SAMPLE PRODUCTS



Notes: The solid line represents the four-week moving average hazard rate, which is defined as the number of new triers during week t divided by the number of nontriers at the beginning of week t.

which in turn lead to S-shaped aggregate cumulative adoption curves commonly found in studies of diffusion of consumer durable goods. In our aggregate data, we rarely observe such patterns, even though we find clear evidence of contagion at the individual level, as we show subsequently. As we indicated previously, aggregate data may lead to wrong conclusions about contagion, due to biases caused by failing to take into account temporal and spatial heterogeneities and various confounds. To tease out the significance of each of these effects on the measurement of contagion, we estimate multiple versions of our proposed individual-level trial model, which allows us to identify the formulation that best represents the adoption behaviors across the 67 products in our sample.

Model Comparisons

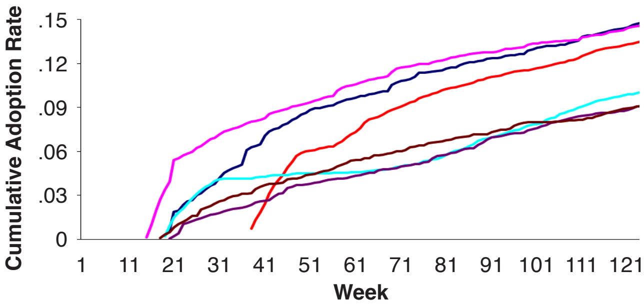
Under our proposed modeling framework, we let the data determine two potential sources of downward biases in the measurement of contagion: (1) spatial heterogeneity, or the number of nearest neighbors as potential influencers (K in Equation 3), and (2) temporal heterogeneity, or the number of lagging weeks over which influences may last (R in Equation 3). When both K and R are equal to 0 (i.e., forcing γ and θ to be zero in Equation 3), our model is equivalent to assuming that no contagion exists in the diffusion process, which we use as a baseline and compare its goodness of fit with that of other models for which we allow K and R to be greater than 0. In particular, we consider five levels of spatial heterogeneity (K = 200, 500, 1000, 1500, and full sample), and three levels of temporal heterogeneity (R = 4, 8, and full observation window), which leads to 16 (1 + 5 × 3) competing model specifications. When K is set to full sample, the model is equivalent to assuming spatial homogeneity (i.e., all consumers have the same influence on one another, regardless of the geographic distance between them), which is a common assumption in standard diffusion models. When R is set to full observation window, the model is equivalent to assuming temporal homogeneity (i.e., the contagion effect of a product trial purchase will last forever, regardless of elapsed time), which is also a common assumption in standard diffusion models.

To determine which K and R combination provides the best fit for our data, we rely on the Bayesian information criterion (BIC) and select the model with the smallest BIC. The left panel of Table 3 reports our findings. We note that when K and R are set to 0 (i.e., disallowing the possibility of contagion), the goodness of fit is the worst (BIC = 676,800, larger than all the other models). Thus, we should not rule out a priori the notion that contagion may play a significant role in consumers' trial decisions toward new CPGs.

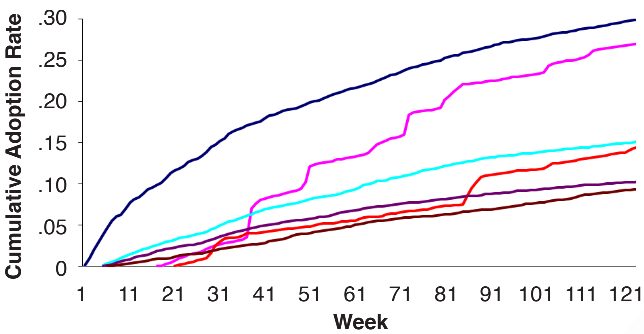
Furthermore, when we compare the BICs across columns, the impact of temporal heterogeneity is evident: Models with R = 4 fit the data better than those with R = 8,

Figure 3
CUMULATIVE ADOPTION RATES OBSERVED FOR SIX SOFT DRINKS AND SIX COOKIES/CRACKERS/SNACKS

A: Soft Drinks



B: CKY/CRKR/SNK



which in turn fit better than models that assume temporal homogeneity (i.e., R = full observation window). We believe that the main reason our data consistently reject temporal homogeneity is the nature of the new products involved in our study (i.e., frequently purchased CPGs, for which contagion is most likely to occur at about the time the product is purchased and consumed, which usually happens within days after purchase).

When we compare the BICs across rows, the impact of spatial heterogeneity is evident: Model fit peaks when K = 1000 and is by far the worst when spatial homogeneity is assumed (i.e., K = full sample). This is consistent with our intuition that the reach of interpersonal influence should be mostly local and the standard assumption of global sphere of influence does not hold in the context of CPGs. Taken together, the preceding model comparisons show that (1) the existence of contagion is supported empirically by our data and (2) temporal heterogeneity and spatial heterogeneity should not be ignored in modeling the diffusion patterns of new CPGs.

In addition to comparing goodness of fit, we compare the number of positive and statistically significant contagion coefficients resulting from each model specification. The right panel of Table 3 reports our findings; when the model is not properly specified, many positive and statistically significant ($p < .01$ and $p < .05$) contagion coefficients become statistically insignificant. When we compare across rows (especially from K = 1000 to full sample), a large number of contagion coefficients turn from positive and significant to insignificant (e.g., 33 at $p < .01$ or 40 at $p < .05$ when K = 1000, and R = 4 versus 11 or 14 when K = full sample and R = 4), highlighting the downward biases that can arise when spatial heterogeneity is assumed away. The difference is less dramatic when we compare across columns (from R = 4 or 8 to full window), indicating that the potential downward biases caused by the assumption of temporal homogeneity may be moderated when we allow for spatial heterogeneity.

Parameter Estimates

Table 4 presents the parameter estimates for the best-performing model (the smallest BIC; i.e., K = 1000 and R = 4). We note that 53 of the 67 price coefficients are negative and statistically significant ($p < .01$), and none are positive and significant. This is consistent with the expectation that consumers in general are responsive to price discounts when making their trial purchase decisions. As for the display/feature coefficients, 30 of 67 are positive and statistically significant, and none are negative and significant. Again, these results are consistent with the notion that display and feature are effective in promoting trial purchases. The number of statistically significant promotion coefficients is smaller than that of price coefficients because not all the 67 products we studied were introduced with much promotion, and for those that were promoted, the promotions tended to be correlated with price discounts. Taken together, the estimates of price and promotion effects (i.e., the β_j's), all having significant and expected signs, provide a strong face validity check on our proposed trial hazard model.

In terms of time trend (i.e., the δ_j's), 40 of the 67 new products show a negative and statistically significant ($p < .01$) linear and/or log-linear trend. Such strong and consistent empirical results suggest that, all other things being equal, the more time elapses since its launch, the less likely consumers are to try a new CPG. It would seem reasonable that, as the product penetrates its target market, there are fewer interested consumers yet to adopt the product and that consumers with a higher propensity to adopt are more likely to have adopted the product previously, leading to this negative trend in aggregate. Nevertheless, we are unsure as to why such negative duration dependence exists, considering all the other time-variant variables we control for in our hazard model. It suffices to say that not taking this trend into account could lead to biases in the measurement of contagion effects.

Of key interest are the estimates of the contagion coefficients (i.e., the γ_j's). That is, 64 of 67 are positive, among which 33 ($p < .01$) to 40 ($p < .05$) are statistically significant; the remaining 3 are negative but statistically insignificant. We interpret this clear pattern as strong empirical evidence that contagion should not be automatically ruled out for the diffusion of CPGs, though our proposed model per se is silent about the specific mechanisms through which contagion may take place. Interpretation of γ_j's aside, because approximately half the new products in our sample have positive and significant contagion coefficients, in modeling individual adoptions of new CPGs, researchers should acknowledge that the probability of trial purchase may increase with the number of recent neighboring purchasers. It is worth stressing again that if the model is not properly specified—that is, spatial or temporal heterogeneity is ignored—many contagion coefficients become statistically insignificant, highlighting the need to allow for these

Table 3

MODEL COMPARISONS BY GOODNESS OF FIT AND POSITIVE AND SIGNIFICANT CONTAGION COEFFICIENTS

	BIC			*Number of Positive and Significant Contagion Coefficients (p < .01)*			*Number of Positive and Significant Contagion Coefficients (p < .05)*		
K\R	*4 Weeks*	*8 Weeks*	*Full Window (i.e., Temporal Homogeneity)*	*4 Weeks*	*8 Weeks*	*Full Window (i.e., Temporal Homogeneity)*	*4 Weeks*	*8 Weeks*	*Full Window (i.e., Temporal Homogeneity)*
0		677,800			0 (i.e., no contagion)			0 (i.e., no contagion)	
200	675,871	675,948	676,151	23	29	24	36	34	29
500	675,672	675,832	676,080	27	29	19	35	36	25
1000	**675,578**	675,711	675,963	**33**	29	13	**40**	36	17
1500	675,605	675,764	675,981	32	28	7	36	38	13
Full sample (i.e., spatial homogeneity)	676,725	676,894	677,081	11	8	4	14	10	5

Table 4
PARAMETER ESTIMATES[a]

selection_id	*Product*	*Intercept*	*Price*	*Promotion*	*Linear Trend*	*Log-Linear Trend*	*Spatial Contagion*[b]	*Non-Neighbors*[b]	*Latent Factor 1*	*Latent Factor 2*
41	BAKERY	–6.495	.262	.634	–.007	–.221	**3.024**	.107	**.209**	.062
55	BAKERY	–3.618	–.810	–.312	.003	**–.453**	**6.782**	.209	**.229**	**–.636**
30	BAKING MIXES	**–7.640**	.852	**.694**	**–.012**	.150	.485	–.273	**.522**	**–.619**
47	BAKING MIXES	**–5.937**	–1.447	.386	–.007	.008	2.047	*1.162*	**.379**	**–.614**
6	CANDY	**–3.087**	**–2.937**	**.841**	**–.014**	–.041	**.644**	–.099	**.780**	**–.418**
7	CANDY	**–3.615**	**–3.071**	**.739**	**–.016**	.189	**1.198**	**–.214**	**.364**	**–.694**
10	CANDY	**–3.861**	**–1.017**	**.746**	.005	**–.279**	**2.287**	**.405**	**.120**	**–.364**
18	CANDY	**–3.952**	**–2.285**	**.718**	**–.013**	.029	**1.277**	**–.531**	**–.164**	**–.523**
19	CANDY	**–4.465**	**–2.134**	**.905**	**–.017**	.024	**1.766**	**–.332**	**.636**	**–.599**
25	CANDY	**–4.576**	**–2.426**	**1.086**	**–.010**	–.048	**1.961**	**–.422**	**1.052**	**–.436**
46	CANDY	**–4.045**	**–1.714**	**.372**	.003	**–.801**	**2.078**	**–.667**	**.609**	**–.884**
50	CANDY	**–4.391**	**–3.125**	**.543**	.004	**–.590**	1.520	–.154	**.660**	**–.869**
53	CANDY	**–4.447**	**–2.300**	**.924**	–.002	**–.273**	*2.392*	–.292	**.343**	**–.687**
21	CHARCOAL	**–2.122**	**–.602**	**.232**	–.011	.036	.336	–.052	**.853**	**–.371**
63	CHARCOAL	**–15.341**	1.553	**.498**	**–.022**	.236	2.074	–.726	**.744**	**–.367**
2	CKY/CRKR/SNK	**–2.340**	**–.508**	**.228**	–.002	**–.286**	**.526**	.004	**1.425**	**–.560**
3	CKY/CRKR/SNK	**–2.819**	**–1.681**	**.678**	**–.006**	–.069	**.761**	**–.377**	**.142**	**–.763**
12	CKY/CRKR/SNK	**–4.604**	**–.809**	**.527**	**.016**	**–.437**	.317	**.485**	**1.023**	**–.666**
15	CKY/CRKR/SNK	**–6.526**	.037	.345	–.006	–.119	*1.663*	.354	**.826**	**–.589**
35	CKY/CRKR/SNK	**–6.416**	–.032	.106	–.007	.008	**1.795**	–.602	**1.226**	**–.164**
52	CKY/CRKR/SNK	**–6.283**	**–.476**	.201	–.005	.072	*2.277*	–.294	**–.539**	**–.920**
40	CNV BREAKFAST	**–5.921**	–.573	**.309**	–.004	–.114	1.386	–.123	**.935**	**–.826**
8	CONDIMENTS & SAUCES	**–5.022**	**–.875**	.281	–.005	–.042	**2.481**	*.420*	**.749**	**–.440**
9	CONDIMENTS & SAUCES	**–3.859**	**–1.010**	.241	.001	**–.265**	**1.755**	.384	**.712**	**–.462**
17	FACIAL TISSUE & NAPKIN	**–4.099**	**–1.231**	**.359**	–.004	**–.297**	**3.572**	.287	**–.946**	–.038
36	FACIAL TISSUE & NAPKIN	**–5.140**	**–.989**	.159	.000	–.262	–.785	**1.826**	**–.975**	–.015
43	FACIAL TISSUE & NAPKIN	**–5.261**	**–.855**	.039	.004	**–.404**	**4.939**	–.294	**–1.597**	**–.407**
66	FACIAL TISSUE & NAPKIN	**–5.862**	**–.582**	.262	.002	**–.282**	**4.970**	–.598	**–1.575**	**–.299**
23	FROZEN GROCERY	**–7.435**	.593	**.581**	–.002	–.141	**3.056**	*–.833*	**1.463**	–.036
33	FROZEN GROCERY	**–5.682**	–1.209	**.609**	–.004	–.105	**1.807**	–.685	**1.550**	**.213**
42	FROZEN GROCERY	**–2.861**	**–.953**	.018	–.002	**–.320**	1.691	–.127	**.286**	**–.657**
48	FROZEN GROCERY	**–4.276**	**–.390**	.258	**–.011**	.098	1.269	**–2.050**	**–.278**	**–.764**
65	FROZEN GROCERY	**–4.271**	**–.396**	–.045	–.008	.039	1.618	*–1.653*	**1.044**	**–.415**
61	HISPANIC FOODS	**–6.501**	–.386	–.214	**–.014**	.282	**5.148**	**–1.905**	**–.227**	**–.899**
60	NEW AGE	**–3.705**	**–2.515**	**.640**	**–.010**	.074	1.323	.019	**.529**	**–.690**
29	NUTS	**–2.595**	**–.914**	**.247**	**–.012**	–.205	**2.778**	.353	**–.210**	**–.725**
13	PKG MEAT	**–1.751**	**–4.460**	**.412**	**–.012**	.088	**1.891**	*–.489*	**1.286**	**–.172**
24	PKG MEAT	**–2.933**	**–2.011**	.313	**–.009**	.116	1.661	**–.785**	**.364**	**–.819**
27	PKG MEAT	**–4.160**	**–1.385**	.084	**–.010**	**.242**	1.182	.096	**.742**	**–1.328**
37	PKG MEAT	**–3.672**	**–1.329**	–.106	–.002	.030	.932	**–1.099**	**.706**	**–1.375**
44	PKG MEAT	**–2.917**	**–1.697**	–.234	**–.009**	.091	1.232	–.356	**.787**	**–1.394**
49	PKG MEAT	**–3.606**	**–1.574**	–.063	–.007	.121	–1.395	.687	**.776**	**–1.429**
56	PKG MEAT	**–3.811**	**–1.067**	.080	.002	–.111	*2.928*	.362	–.124	**–1.046**
59	PKG MEAT	**–4.550**	**–.818**	–.298	.004	**–.498**	*3.105*	.631	**1.029**	**–.877**
64	PKG MEAT	**–5.681**	**–.312**	**.422**	.000	–.074	1.094	–.858	–.046	**–.571**
32	REFRIG GROCERY	**–3.378**	**–1.758**	–.017	–.006	–.027	–.045	.545	.107	**–.781**
34	REFRIG GROCERY	**–4.934**	**–1.056**	.271	–.005	–.066	**3.330**	.511	**.408**	**–.745**
54	REFRIG GROCERY	**–5.106**	**–.752**	.224	.004	–.107	1.529	–.198	**–.777**	**–.920**
62	REFRIG GROCERY	**–3.477**	**–1.104**	**.285**	**.011**	**–.506**	.396	.369	**.159**	**–.925**
67	REFRIG GROCERY	**–5.150**	**–.908**	.354	.000	–.125	.789	–.599	**–1.146**	**–.691**
1	SALAD MIX	**–4.950**	**–.481**	**.678**	**–.005**	–.001	**.629**	.024	**–.992**	**–1.110**
4	SALAD MIX	**–2.455**	**–.933**	**.776**	.002	**–.377**	**2.184**	**–.406**	**.689**	**–.528**
5	SALAD MIX	**–5.257**	**–.379**	**1.058**	–.001	**–.240**	**1.029**	*.276*	**–.331**	**–1.033**
11	SALAD MIX	**–3.054**	**–1.588**	–.154	–.003	**–.252**	.851	–.209	**–.612**	**–.600**
16	SALAD MIX	**–1.983**	**–1.573**	.047	.003	–.225	**3.182**	–.269	**–1.297**	**–.893**
31	SALAD MIX	**–4.669**	**–1.168**	.122	**–.012**	–.042	**1.715**	–.368	**–.874**	**–.825**
45	SHELF STABLE VEGES	**–6.058**	**–.864**	.204	.002	–.082	1.914	.600	**–.242**	**–.547**
57	SHELF STABLE VEGES	**–6.279**	–.579	.082	–.003	–.081	**5.510**	.056	**–.357**	**–.566**
14	SOFT DRINKS	**–2.961**	**–.815**	.140	–.007	**–.304**	**1.078**	–.187	**–.501**	**–1.277**
20	SOFT DRINKS	**–2.127**	**–.919**	**.210**	.003	**–.646**	**1.488**	–.015	**–.644**	**–.923**
22	SOFT DRINKS	–1.223	**–3.758**	.356	–.001	**–.353**	*1.190*	–.097	**1.316**	**–.806**
28	SOFT DRINKS	**–1.470**	4.038	.000	.008	**–.669**	1.803	.539	.071	**–1.122**
38	SOFT DRINKS	**–3.296**	**–2.581**	.148	.004	**–.430**	*2.047*	–.129	**–.438**	**–1.332**
51	SOFT DRINKS	**–4.149**	**–2.461**	**.409**	–.003	–.202	1.282	*–.921*	**1.519**	**–.304**
26	TEAS	**–3.576**	**–.838**	.185	.003	**–.374**	**3.592**	*–.926*	**.301**	**–.385**
39	TURKEY GRINDS	**–3.757**	**–.340**	–.139	.006	**–.645**	**4.911**	–.120	**.331**	**–.364**
58	YOGURT	**–7.605**	1.348	**.795**	–.008	–.086	.777	.584	**–.229**	**–.920**

[a]Estimates significant at $p < .01$ are in bold.

[b]For these two parameters, estimates significant at $.01 < p < .05$ are in italics; to increase the number of effective digits displayed within three decimal places, we normalize the numbers of neighbors and nonneighbors by 100.

heterogeneities when empirically measuring contagion with diffusion data.

Recall that M_{ijt}^{RK}, defined as the number of non-K-nearest neighbors of consumer i who bought product j at least once in the R periods before t, is included in the hazard function as a control for time-variant unobservables (e.g., competition) that can affect λ_{ijt} and, at the same time, affect both N_{ijt}^{RK} (neighbors) and M_{ijt}^{RK} (nonneighbors). Having M_{ijt}^{RK} in the hazard function provides a strong proxy for such correlated unobservables. Most of the coefficient estimates for M_{ijt}^{RK} (i.e., θ_j's) turn out to be statistically insignificant (53 with $p > .01$ or 45 with $p > .05$). The remaining θ_j's are mostly negative (11 at $p < .01$ and 16 at $p < .05$), with only 3 being positive at $p < .01$ and 6 being positive at $p < .05$.

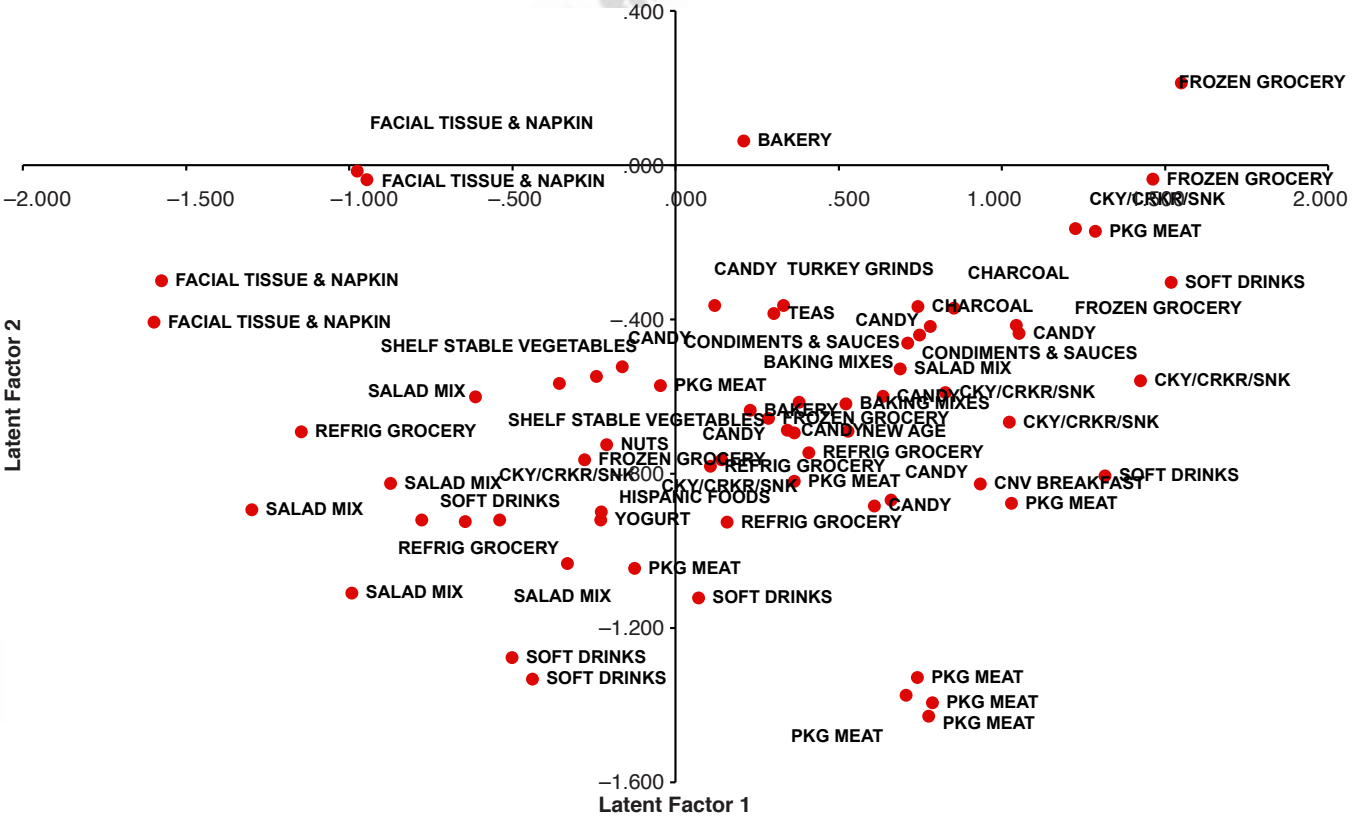
If contagion has no role or when it does and the impact is global (i.e., no difference between neighbors and nonneighbors), we should expect no systematic difference between the coefficient estimates for N_{ijt}^{RK} (neighbors) and those for M_{ijt}^{RK} (nonneighbors). However, that is not what we found out empirically; rather, we learned that the vast majority of the coefficient estimates for M_{ijt}^{RK} are either insignificant (45 with $p > .05$) or negative (16 at $p < .05$), forming a stark contrast with the coefficient estimates for N_{ijt}^{RK} (i.e., our measure of contagion effects), which are almost all positive (64 of 67), with more than half being statistically significant (40 at $p < .05$). Such a contrast adds discriminant validity to the hypothesis that contagion plays a role in the diffusion of many new CPGs, and when it does, the interpersonal influence consumers receive comes predominantly from recent purchasers among their neighbors (i.e., contagion has a local sphere of influence) rather than from nonneighbors (M_{ijt}^{RK}).[4]

The last two columns of Table 4 present the estimated loadings of two latent factors (i.e., Λ_j's), which we plot in Figure 4 for ease of interpretation.[5] Two products that have Λ_j's pointing in the same direction are likely to be tried early by the same group of consumers. Overall, the estimated loadings make intuitive sense. For example, 64 of 67 loadings on Latent Factor 2 are negative and statistically significant, and none are positive and significant. All other things being equal, consumers with larger (more negative) factor scores (Z_i's) on this dimension have higher baseline trial propensities for nearly all new products in our sample and therefore are more likely to try them earlier. In other words, Latent Factor 2 could be interpreted as an intrinsic willingness to try new products early, regardless of the num-

[4]The negative coefficient estimates for nonneighbors (as we discovered for 11 of the 16 new products, $p < .01/.05$) are largely a reflection of the natural negative correlation between aggregate trial rate (which declines over time) and the number of prior adopters and customer base for repeat purchases (which increases over time). The t and ln(t) variables included in the hazard function capture most but not all of this negative correlation, and what is left is captured by M_{ijt}^{RK} (which in general also increases over time, more so immediately after launch).

[5]We also estimated the proposed model with three and four factors. The three-factor solution is slightly better than the two-factor solution, which is about the same as the four-factor solution. For the sake of simplicity and ease of interpretation, we report here only the results based on the two-factor solution.

Figure 4
SCATTERPLOT OF LATENT FACTOR LOADINGS



ber of early adopters in the market. In addition, note that the factor loadings for many products from the same category are similar (e.g., FACIAL TISSUE and NAPKIN, PKG MEAT, SALAD MIX), indicating that the amounts of time it took consumers to try these products were highly correlated, which is intuitive because individual consumers' baseline propensities to try new products from a particular category should be largely driven by their familiarity and involvement with that category.

Predictive Validity Test

Compared with existing models in the literature, our proposed model includes two additions: the latent factor structure ($\alpha_{ij} = \alpha_{ij} + \Lambda_j Z_i$), intended to control for unobserved heterogeneity in baseline trial propensity, and the covariates N_{ijt}^{RK} (recent purchasers among neighbors) and M_{ijt}^{RK} (recent purchasers among nonneighbors), intended to detect contagion effects. Although the main goal of our proposed model is to measure contagion, rather than forecasting trial sales, to ascertain the risk of overfitting and the model's predictive validity, we benchmarked our model against the exponential-gamma hazard model with covariates, which has been identified as the best-performing model for forecasting new CPG trial sales (Fader, Hardie, and Zeithammer 2003). More specifically, we calibrated both our model and the exponential-gamma model using data from the first 84 weeks of the 124-week observation window, with the exponential-gamma model ignoring contagion and including X_{ijt}, t, and ln(t) as the covariates. We then used the calibrated models to predict, in the holdout 40-week period, trial probability for each nonadopter in each week. To evaluate performance, we compared the predicted weekly aggregate trial rates with their observed counterparts, across the 67 products in our sample.

Table 5 displays the mean absolute error (MAE), comparing the predicted number of weekly trials, according to either of the two competing models, for the last 40 weeks in our observation window with the actual number of weekly trials. For ease of interpretation, we sorted the 67 new products by the decreasing order of improvement in MAE produced by our proposed model relative to the exponential-gamma model, which ignores contagion.

Table 5 shows that our proposed model produced better predictive performance for 52 of the 67 products, with up to 33% improvement (in terms of producing smaller MAE) and a median and mean improvement of 5% and 7.5%, respectively, over the exponential-gamma model. Such improvements are nontrivial given that we are benchmarking against a model that represents the state of the art in forecasting new CPG trial sales, according to Fader, Hardie, and Zeithammer (2003). We interpret this as additional evidence that information on neighbors' past purchases helps predict the focal consumer's trial decision because contagion plays a significant role and should not be automatically ignored when studying the diffusion of CPGs.

In addition to contagion, another key difference between our model and the exponential-gamma (and all the other popular CPG adoption models Fader, Hardie, and Zeithammer [2003] examine) is that our model is calibrated simultaneously using data from all 67 products (thus taking advantage of cross-product correlation in observed trial behavior), whereas all the existing models can only be applied to each product independently (one product at a time). Using trial history from a single product, existing models have little information to identify individual baseline trial propensity. The only information about the nonadopters is that they have not tried the focal product by the time of prediction. Exponential-gamma and other models would essentially predict the same trial probability for every nonadopter in the holdout period because there is no information to distinguish one nonadopter from another on the basis of trial history for a single product. In contrast, our model enables us to borrow information (through the latent factor structure) from trial histories of all the other 66 products in making predictions for the focal product. In other words, with trial histories from 67 products, we have enough information to identify heterogeneity in baseline trial propensity at the individual level, which is captured by the estimated consumer-specific latent factor scores. These scores, reflecting trial histories across 67 products in the calibration period, help predict the focal consumer's trial probabilities in the holdout period. (Intuitively, for two consumers who have not tried the focal product at the time of prediction, if we know that one has tried many other new products in the calibration period and the other has tried none, our model would predict higher trial probability for the first consumer.)

In short, by comparing the holdout predictive performance of our proposed model with that of a well-known hazard model that uses the same data, we ensure that our detection of contagion effects is not due to overfitting the data; if that were the case, our model would produce poorer holdout predictions than a simpler model that ignores contagion. The results from our predictive validity test should further enhance the credibility of our unconventional findings.

MANAGERIAL IMPLICATIONS AND CONCLUSIONS

The empirical analyses reported and discussed in this study call for a shift in marketing researchers' view of how consumers adopt new CPGs. The common practice in the CPG industry has been to ignore the potential for interpersonal influence among consumers and treat new product trials as independent events across consumers, and most existing trial forecast models for new CPGs ignore contagion. Fortunately, with the prevalence of shopping clubs and frequent-shopper programs among retailers, particularly grocery chains, the analyses reported in our study can be readily replicated, resulting in customer-specific measures of innovativeness, or the likelihood to try a product earlier than others ($\Lambda_j Z_i$), and influence, or the capacity to exert influence on others (OC_i, or X_i). To illustrate how a grocery chain could apply the results from our individual-level diffusion model, we use the parameter estimates reported in Table 4 to produce three measures for two product categories, FROZEN FOODS and SOFT DRINKS, which have five and six sample products, respectively:

- $Y_{ic} = \Lambda_c Z_i$ represents consumer i's innovativeness in product category c, or the deviation of consumer i's log-hazard for category c from the sample average. This measure indicates the extent to which consumer i is expected to try a new product in category c earlier than others;
- $OC_{ic} = \Sigma_{i'=1}^{N} W_{ii'c}$ represents the out-degree centrality for consumer i in category c, measuring the direct influence of consumer i on others in adopting new products from category c; and

Table 5
MAEs IN PREDICTING WEEKLY TRIALS IN THE HOLDOUT PERIOD

selection_id	*Product*	*Proposed Model*	*Expoential-Gamma*	*Percentage Error Reduction*	*Improvement*
33	FROZEN GROCERY	1.44	1.93	33.8	1
7	CANDY	2.06	2.76	33.8	1
60	NEW AGE	1.13	1.46	29.7	1
29	NUTS	2.43	2.97	22.1	1
8	CONDIMENTS & SAUCES	1.66	1.99	19.8	1
1	SALAD MIX	2.90	3.47	19.8	1
24	PKG MEAT	2.02	2.41	19.0	1
32	REFRIG GROCERY	2.41	2.85	18.6	1
4	SALAD MIX	3.82	4.48	17.3	1
56	PKG MEAT	1.64	1.91	16.5	1
25	CANDY	2.44	2.84	16.2	1
17	FACIAL TISSUE & NAPKIN	1.62	1.87	15.8	1
36	FACIAL TISSUE & NAPKIN	1.40	1.62	15.8	1
57	SHELF STABLE VEGETABLES	1.61	1.86	15.5	1
28	SOFT DRINKS	2.26	2.60	15.1	1
3	CKY/CRKR/SNK	2.25	2.57	14.5	1
44	PKG MEAT	1.69	1.91	13.3	1
22	SOFT DRINKS	1.47	1.64	11.6	1
11	SALAD MIX	1.80	1.99	10.7	1
53	CANDY	2.00	2.21	10.2	1
45	SHELF STABLE VEGETABLES	2.29	2.52	9.9	1
43	FACIAL TISSUE & NAPKIN	1.71	1.88	9.8	1
66	FACIAL TISSUE & NAPKIN	1.26	1.38	9.8	1
23	FROZEN GROCERY	1.92	2.10	9.4	1
35	CKY/CRKR/SNK	1.48	1.62	9.2	1
5	SALAD MIX	2.84	3.10	9.1	1
54	REFRIG GROCERY	2.10	2.29	8.6	1
10	CANDY	4.58	4.95	8.2	1
6	CANDY	2.68	2.90	8.1	1
2	CKY/CRKR/SNK	2.30	2.45	6.5	1
12	CKY/CRKR/SNK	4.48	4.77	6.4	1
26	TEAS	2.50	2.65	5.8	1
16	SALAD MIX	1.51	1.59	5.1	1
39	TURKEY GRINDS	1.77	1.85	4.4	1
19	CANDY	2.19	2.29	4.4	1
40	CNV BREAKFAST	1.82	1.90	4.3	1
65	FROZEN GROCERY	1.36	1.42	4.3	1
47	BAKING MIXES	1.84	1.92	4.3	1
67	REFRIG GROCERY	1.64	1.70	4.0	1
37	PKG MEAT	1.69	1.75	3.6	1
50	CANDY	1.98	2.04	3.3	1
48	FROZEN GROCERY	1.41	1.46	3.3	1
34	REFRIG GROCERY	2.16	2.23	3.1	1
14	SOFT DRINKS	2.15	2.21	3.1	1
13	PKG MEAT	1.99	2.04	2.9	1
42	FROZEN GROCERY	2.27	2.33	2.3	1
38	SOFT DRINKS	1.84	1.88	2.1	1
27	PKG MEAT	2.19	2.23	1.7	1
62	REFRIG GROCERY	1.60	1.63	1.4	1
20	SOFT DRINKS	1.57	1.58	.8	1
64	PKG MEAT	1.51	1.52	.5	1
55	BAKERY	1.21	1.21	.3	1
18	CANDY	2.55	2.55	–.1	0
59	PKG MEAT	2.01	2.01	–.1	0
58	YOGURT	1.98	1.97	–.6	0
51	SOFT DRINKS	1.70	1.69	–.6	0
9	CONDIMENTS & SAUCES	2.67	2.64	–1.1	0
46	CANDY	1.74	1.72	–1.1	0
15	CKY/CRKR/SNK	1.72	1.68	–2.1	0
49	PKG MEAT	1.93	1.88	–2.2	0
30	BAKING MIXES	1.71	1.67	–2.3	0
52	CKY/CRKR/SNK	1.79	1.74	–2.7	0
41	BAKERY	1.60	1.55	–2.9	0
31	SALAD MIX	2.23	2.16	–3.3	0
63	CHARCOAL	2.07	2.00	–3.4	0
21	CHARCOAL	3.03	2.88	–4.9	0
61	HISPANIC FOODS	1.58	1.48	–6.5	0
	Average	2.03	2.18	7.5	52

- X_{ic} denotes the eigenvector centrality for consumer i in category c, measuring the total (direct and indirect) influence of consumer i on others in adopting new products from category c.

To verify the convergent and discriminant validity of these measures across the two product categories, we computed the rank-order correlations reported in Table 6, which shows statistically significant correlation (.16) in innovativeness between the SOFT DRINKS and FROZEN FOODS categories. Because eigenvector centrality measures total (direct and indirect) influence and out-degree centrality measures direct influence, we should expect these two measures to be correlated within a product category, which again is confirmed by Table 6's results for both product categories (.65 for both SOFT DRINKS and FROZEN FOODS).

Given that innovative shoppers (identified through $\Lambda_j Z_i$) are likely to try new products earlier, they would also have more opportunities to influence others who are connected to them, and therefore we would expect innovativeness to be positively correlated with influence within a product category, which again is confirmed by Table 6's results (.50 and .35 for SOFT DRINKS and .58 and .55 for FROZEN FOODS). However, there seems to be no strong reason to expect that influence in one product category will lead to influence in a seemingly unrelated category, which is confirmed by our results; most of the cross-category correlations between influence measures were not statistically significant, and the only two statistically significant correlations were negative but small.

After the measures of innovativeness and influence discussed previously are obtained for each customer of the grocery chain, managers can use this information to target new product introductions more effectively. We illustrate this process using data for the same two product categories (SOFT DRINKS and FROZEN FOODS), in each of which we had one additional newly introduced product. We assigned our sample customers into nine segments, combining three innovativeness terciles and three influence terciles, using the same innovativeness and out-degree centrality (i.e., direct influence) scores discussed previously. We focus on direct influence in this illustration because the network of direct influences is more easily defined than the complex network of indirect influences. A manager interested in diffusing his or her new product as quickly and deeply as possible among the grocery chain's customers would be interested in reaching two types of shoppers. First, he or she would like to target the most innovative tercile because that would lead to a higher number of early trials faster. Second, he or she would like to target the most influential tercile because that would lead to the greatest spatial spillover effect. To verify the relationship between trial of a new product (not included in our calibration sample) and our measures of innovativeness and direct influence (derived from adoption histories of the products included in the calibration sample), we allocate each observed trial of the holdout new product to the nine innovativeness–influence segments in proportion to the degree of influence ($W_{ii'}$ in Equation 5) of customers in each segment. Table 7 reports the aggregated trials potentially attributable to each segment.

The first line in each cell of Table 7 shows the number of customers belonging to that segment. The segment sizes are unbalanced because customers who are likely to adopt a new product early have more opportunities to be influential, as discussed previously. The second line shows the number of direct trials, observed among members of each segment. For FROZEN FOOD, it is evident that the absolute number (as well as the relative proportion) of direct trials decreases as we move down from the top tercile of innovativeness. The third line in each cell shows the number of indirect trials, observed among consumers who do not belong to the segment but were potentially influenced by members of the focal segment. As we would also expect, the number of influenced trials of the new FROZEN FOOD product decreases as we move down from the top influence tercile. (The same pattern holds for the ratio between influenced trials and segment sizes.) Table 7, Panel A, shows that the manager would be more effective targeting customers in the top tercile of both innovativeness and influence because that segment generates the most direct and influenced trials, in both absolute numbers and relative proportions.

In general, the results shown in Table 7, Panel B, for the holdout new SOFT DRINK product are similar, except that there are more direct trials among households in the bottom tercile of innovativeness than in the middle decile, suggesting that the link between the innovativeness score obtained from the calibration sample of products does not apply as well to the holdout product as in the previous example (Table 7, Panel A). Nevertheless, the general conclusion still holds; the manager is better off focusing on the top-ranked customers in innovativeness and influence because they generate the most direct and influenced trials.

Table 6
CONVERGENT AND DISCRIMINANT VALIDITY OF INNOVATIVENESS AND INFLUENCE MEASURES

	Soft Drinks			*Frozen Foods*		
	Innovativeness	*Outer-Degree Centrality*	*Eigenvector Centrality*	*Innovativeness*	*Outer-Degree Centrality*	*Eigenvector Centrality*
Soft Drinks						
Innovativeness	**1.00**					
Outer-degree centrality	**.50**	**1.00**				
Eigenvector centrality	**.35**	**.65**	**1.00**			
Frozen Foods						
Innovativeness	**.16**	.09	.09	**1.00**		
Outer-degree centrality	.03	.08	**–.13**	**.58**	**1.00**	
Eigenvector centrality	.03	.08	**–.32**	**.55**	**.65**	**1.00**

Notes: Correlations in bold are statistically significant at the .01 level.

Table 7
NUMBER OF TRIALS BY INNOVATIVENESS AND INFLUENCE TERCILES

A: Frozen Foods				
	Influence			
Innovativeness	*Top Tercile*	*Middle Tercile*	*Bottom Tercile*	*Total*
Top Tercile				
Customers	425	205	59	689
Direct trials	42	20	5	67
Influenced trials	51	11	2	64
Middle Tercile				
Customers	215	290	184	689
Direct trials	19	27	16	62
Influenced trials	16	9	2	27
Bottom Tercile				
Customers	49	194	447	690
Direct trials	4	16	28	48
Influenced trials	10	8	6	24
Total				
Customers	689	689	690	2068
Direct trials	65	63	49	177
Influenced trials	77	28	10	115
B: Soft Drinks				
	Influence			
Innovativeness	*Top Tercile*	*Middle Tercile*	*Bottom Tercile*	*Total*
Top Tercile				
Customers	514	266	81	861
Direct trials	66	14	8	88
Influenced trials	65	15	3	83
Middle Tercile				
Customers	183	350	328	861
Direct trials	15	28	21	64
Influenced trials	19	15	5	39
Bottom Tercile				
Customers	164	245	453	862
Direct trials	15	23	33	71
Influenced trials	13	10	4	27
Total				
Customers	861	861	862	2584
Direct trials	96	65	62	223
Influenced trials	97	40	12	149

This example illustrates that a marketer could potentially reach a larger number of triers more effectively by first targeting customers who are the most innovative and influential according to our model-based innovativeness and influence measures. As large retailers continue to expand their loyalty programs, their data on individual customers' innovativeness and influence may be employed as another valuable service to manufacturers. Rather than wasting their budget on mass-marketing campaigns, which tend to reach mostly consumers who are unlikely to adopt the new product or influence others, a manufacturer can tap into retailers' loyalty program databases to more effectively target their new product introductions to customers who are more likely to not only try the new product but also influence others to do the same.

In conclusion, our examination of the purchase histories of a broad range of new CPGs for a large panel of consumers over an extended period shows that, contrary to conventional wisdom, there is empirical evidence of contagion in the diffusion of many CPGs. Although our proposed model is silent about the mechanisms through which contagion may occur, which may not require explicit interpersonal ties, our empirical findings make it clear that in modeling individual adoptions of new CPGs, researchers should understand that the probability of trial can be increasing in the number of previous purchasers.[6] The key is to account for various potential biases properly. In particular, we find that in the context of CPGs, contagion effects are mostly local, temporally (i.e., lasting for only a limited period) and spatially (i.e., reaching only a limited geographic area). We hope that our research will caution both managers and researchers against writing off a priori the potential value of leveraging contagion in the diffusion of CPGs.

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[6]One hypothesis is that in the context of CPGs, contagion may depend less on direct communications, such as word of mouth, than on other indirect interactions, such as by observing or being exposed to purchases and/or consumption among prior purchasers. Such a hypothesis could be tested by making the strength of contagion a function of product social visibility. The empirical challenge lies in how to measure product social visibility accurately.

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