Creating Social Contagion through Viral Product Design: A Randomized Trial of Peer Influence in Networks

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We examine how firms can create word-of-mouth peer influence and social contagion by designing viral features into their products and marketing campaigns. Word-of-mouth (WOM) is generally considered to be more effective at promoting product contagion when it is personalized and active. Unfortunately, the relative effectiveness of different viral features has not been quantified, nor has their effectiveness been definitively established, largely because of difficulties surrounding econometric identification of endogenous peer effects. We therefore designed a randomized field experiment on a popular social networking website to test the effectiveness of a range of viral messaging capabilities in creating peer influence and social contagion among the 1.4 million friends of 9,687 experimental users. Overall, we find that viral product design features can indeed generate econometrically identifiable peer influence and social contagion effects. More surprisingly, we find that passive-broadcast viral messaging generates a 246% increase in local peer influence and social contagion effects, while adding active-personalized viral messaging only generates an additional 98% increase in contagion. Although active-personalized messaging is more effective in encouraging adoption per message and is correlated with more user engagement and sustained product use, passive-broadcast messaging is used more often enough to eclipse those benefits, generating more total peer adoption in the network. In addition to estimating the effects of viral product design on social contagion and product diffusion, our work also provides a model for how randomized trials can be used to identify peer influence effects in networks.

Key words: Peer Influence, Social Contagion, Social Networks, Viral Marketing, Information Systems, Randomized Experiment.

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"Causal knowledge requires controlled variation."

– Armin Falk and James Heckman

1. Introduction

It is widely believed that social contagion and word of mouth (WOM) "buzz" about products drive product adoption and sales and managers are increasingly relying on "network" and "viral" marketing strategies to maximize returns to marketing investments (Garber et al 2004, Hill et al 2006, Van den Bulte and Joshi 2007, Manchanda et al 2008, Nam et al 2010, Van der Lans et al 2010). If firms can proactively manage WOM communication, they may be able to engineer the viral spread of products to achieve widespread adoption (Mayzlin 2006, Godes and Mayzlin 2009). Yet, two issues critical to the success of viral marketing efforts have been systematically understudied in the WOM literature – *viral product design* and *the econometric identification of peer influence*. We simultaneously address both topics by conducting a large scale randomized field experiment to test whether viral product features create peer influence and social contagion in new product diffusion.

Viral product design – the process of explicitly engineering products so they are more likely to be shared amongst peers – has existed at least since the first chain letter was sent in 1888.[1] Today, information technologies (IT) enable firms to design products with features that make them more likely to be virally shared. IT-enabled products regularly facilitate the spread of product awareness using automated broadcast notifications of peer activities and by enabling users to actively and personally invite peers to adopt products. Yet, there is almost no empirical evidence on the effectiveness of viral features in generating social contagion and product adoption. We therefore investigate two basic research questions: Can firms add viral features to products so they are more likely to be shared amongst peers? If so, which viral features are most effective in inducing WOM and peer-to-peer influence in product adoption?

[1] This earliest known example of a chain letter seems to have originated as a part of a philanthropy effort initiated by four women requesting donations for education efforts in the Cumberlands region of New Hampshire. The details of this letter may be found at: http://www.silcom.com/~barnowl/chain-letter/evolution.html. Text of first chain letter can be found at: http://www.silcom.com/~barnowl/chain-letter/archive/ce1888-12_cumberlands_sdq4.htm

Unfortunately, evaluating the effects of viral product design features on social contagion is difficult because peer effects and WOM are typically endogenous (Manski 1993, Godes and Mayzlin 2004, 2009, Hartmann et al 2008, Aral et al 2009). We therefore designed and conducted a randomized field experiment testing the effectiveness of two of the most widely used viral product features – active-personalized referrals and passive-broadcast notifications – in creating peer influence and social contagion among the 1.4 million friends of 9,687 experimental users of Facebook.com. The experiment uses a customized commercial Facebook application to observe user behavior, communications traffic and the peer influence effects of randomly enabled viral messaging features on application diffusion and use in the local networks of experimental and control population users. By enabling and disabling viral features among randomly selected application users, we are able to obtain relatively unbiased causal estimates of the impact of viral features on the adoption rates of peers in the local networks of adopters. Using detailed click stream data on users' online behaviors we also explore whether positive network externalities generated by additional peer adopters inspire further product adoption and sustained product use.

Word of mouth is generally considered to be more effective at promoting product contagion when it is personalized and active. Surprisingly, we find that designing products with passive-broadcast viral messaging capabilities generates a 246% increase in local peer influence and social contagion while adding active-personalized viral messaging capabilities only generates an additional 98% increase. Although active-personalized messaging is more effective in encouraging adoption per message and is correlated with more user engagement and sustained product use, it is used less often and therefore generates less total peer adoption in the network than passive-broadcast messaging. Overall, we find viral product design features generate econometrically identifiable peer influence and social contagion effects and show how randomized trials can identify peer influence in networks.

2. Viral Product Design

Since the early work of Katz and Lazersfled (1955) and others (Coleman et al 1966, Grilliches 1957, Arndt 1967, Engel et al 1969), there has been great interest in how WOM can drive consumer de-

mand, public opinion and product diffusion (Reingen et al 1984, Banerjee 1992, 1993, Foster and Rosenzweig 1995, Bowman and Narayandas 2001, Bell and Song 2007, Reingen and Kernan 1986, Brown and Reingen 1987, Bowman and Narayandas 2001, Godes and Mayzlin 2004, Aral et al 2009). Evidence on the importance of WOM has led researchers to examine if firms can proactively create broad, systematic propagation of WOM through consumer populations. Mayzlin (2006) and Dellarocas (2006) provide theoretical support for the potential effectiveness of firm created WOM by showing that equilibrium strategies exist in which firms would profit from posing as customers to generate favorable online WOM even when customers are aware they might be doing so. Godes and Mayzlin (2009) empirically demonstrate the effectiveness of firm initiated buzz marketing, in which paid "agents" spread the word about products, generating exogenous WOM conversations where "none would have naturally occurred otherwise." (Godes and Mayzlin 2009: 721) Proactive firm efforts also target "influential" individuals who are likely to propagate organic WOM most broadly (Katz and Lazersfeld 1955, Merton 1968, Gladwell 2000, Watts and Dodds 2007, Goldenberg et al 2001, 2009, Reingen et al 1984) and optimize referral programs to create incentives for them to spread the word (Biyalogorsky et al 2001, Libai et al 2003, Ryu and Feick 2007).

Conspicuously absent from this large literature on viral marketing is work on viral product design. As Berger and Milkman (2009: 5) note "macro explanations for diffusion … tend to ignore how individual level processes influence what gets shared … Focusing on network structure … and on the influence of special people provides little insight into why certain cultural items become viral while others do not … Brown and Reingen (1987) note that "an enhanced understanding of social influence processes in consumer behavior may simply be obtained by examining which products or services consumers are more likely to "talk about." (p.361), yet little empirical work has answered this call."

We conceptualize viral product design – the process of explicitly engineering products so they are more likely to be shared amongst peers – as encompassing the incorporation of specific *characteristics* and *features* into a product's design to generate peer-to-peer influence in its adoption process. A product's *viral characteristics* are fundamentally about its content and the psychological effects content can have on

a user's desire to share the product with peers. A nascent literature has begun to identify content characteristics that inspire viral product diffusion including usefulness, topicality, prominence, positive valence and unexpectedness (Berger and Milkman 2009, Stephen and Berger 2009, Berger and Heath 2005, Phelps et al 2004, Heath, Bell and Sternberg 2001). A product's *viral features* on the other hand concern modalities of use with respect to sharing – how features enable and constrain a product's use in relation to other consumers. Products may enable communication between users, generate automated notifications of each other's activities, facilitate personalized invitations to peers or enable hypertext embedding of the product on publicly available websites and weblogs. Two of the most widely used viral product features are personalized referrals and automated broadcast notifications.

Personalized Referrals. Enabled by both front end user interface features and backend database management technologies, personalized referral features enable users to select their friends or contacts from a list and to invite them to join the service with the option to attach a personalized message to the invitation. Companies like Facebook enable users to 'invite their friends' to join the service through personalized referrals. When users send Gmail messages, an automated, pop-up hyperlink enables them to invite recipients to join Gmail. Personalized referrals are targeted at peers the user actively selects and can be personalized even further with the inclusion of a personal message.

Automated Broadcast Notifications. The automated broadcast notification is passively triggered by normal user activity. When a user engages the product or takes an action which triggers the product to take an action (e.g. send a message, update the user's status), these actions can be broadcast as notifications to the user's contacts (whether or not their contacts are current users). When a user of LinkedIn.com joins a new group, changes their profile information, connects to a new contact or takes a new job, their contacts are informed via email about the activity, building product awareness and encouraging users to return to the site to see what their contacts are doing. Facebook notifies friends when a user adopts a new application or achieves some application milestone. Notifications such as these build awareness among friends of new activities or products a user is adopting or engaging with and can encourage friends of cur-

rent application users to become aware of, interested in and to eventually adopt the application themselves.

Referrals are more personalized and targeted than broadcast notifications. Personalization describes the degree to which the output of the viral feature is tailored to each specific peer or more generally aimed at anyone, ranging from 'broadcast' to 'personalized.' For example, personal referrals are more personalized than notifications because the user actively chooses a subset of their social network to which the referral is sent. Referrals can be even more personalized if the user chooses to attach a personal note to the referral.[2] Personalization therefore encompasses both targeting and customization. Targeting specifies whether the feature is directed at the broad population of potential consumers, a subset of consumers like a current user's social network, or a specific person (Hill et al 2006). Customization specifies whether the content of a feature's engagement with the recipient can be tailored to a group of friends or a specific individual with a personalized message that is either actively written by the user or passively generated by the system (Tam and Ho 2005).

Word of mouth is generally considered to be more effective at promoting product contagion when it is personalized and active. When individuals proactively choose to share information about products and services with their friends, they tend to choose to activate their strong tie relationships (Frenzen and Nakamoto 1993, Stephen and Lehmann 2009). Strong ties exhibit greater homophily (McPherson et al 2001, Jackson 2008), greater pressure for conformity (Coleman 1988) and deeper knowledge about one another. We simply know more about the preferences of our close friends and colleagues than we do about our acquaintances. We tend to trust information from close "trusted" sources more (typically our strong embedded ties) (Uzzi 1996) and we tend to respond more often to them out of a feeling of responsibility and reciprocity (Emerson 1962). In addition, the personalization of messages tends to make them more effective, especially in online environments in which we are constantly bombarded with irrelevant

[2] Generic messages can be replaced or complemented with personalized text. For example, an invitation sent from the Flixster Facebook application uses the default text: "Hey - allow this app access so we can test our Movie Compatibility." Users sending the invitation may click the "add a personal message" button to customize the invitation.

information (Tam and Ho 2005, Dijkstra 2008). For these reasons one might suspect that personalized referrals are more persuasive and therefore more effective than broadcast notifications.

However, the argument that personalization is more likely to be effective is based on the persuasiveness of the message (and thus likelihood of a positive response or the marginal effectiveness of the feature 'per message') and neglects the effort required by the user to actively select and invite peers to the application. It could be that while personalized viral features are more effective at persuading a recipient to respond positively, the time and energy costs of using the feature prevent its widespread use, limiting its effectiveness in encouraging increases in total overall adoption. It is therefore important to also consider the level of activity a feature requires of users. Activity describes the degree to which users must actively initiate the viral feature and ranges from ‘active’ to ‘passive.’ Typically, when invites are sent by users to their peers suggesting they adopt a product, the user actively chooses which friends to invite and what type of message to send to them as part of the invitation – each of these actions requires the user’s judgment and active participation. In contrast, features that automatically notify peers of a user’s activity with regard to the product without any active choice on the part of the user (e.g. Facebook notifications) are passive in that users’ judgment and active participation are not required to initiate the notification.

When combined, the two dimensions of activity and personalization describe a continuous space of viral product features that ranges from broadcast features generally aimed at anyone to personalized features targeted and tailored toward specific peers, and from active features that require active user engagement to passive features that generate automated actions on behalf of the user. Figure 1 graphically describes this space, with the personalization dimension increasing from the left to the right and the activity dimension increasing from the bottom to the top of the figure.

*** Figure 1 About Here ***

Personalized referrals and notifications, the two examples we empirically evaluate in this paper are denoted in grey boxes, while other examples are denoted in white boxes. Personalized referrals are the most personalized of our examples and also require the most effort by users, while notifications require very little effort beyond normal use of the product and are not personalized but instead broadcast to a

wider population. Each of these examples could also 'move' in the space depending on the specific instantiation of the viral feature. For instance, notifications that only target a subset of the population, a user's personal social network or specific individuals (through collaborative filtering for example) would each appear further to the right of the automated notification example noted in the diagram. Examples of personalized referrals include Facebook, Gmail and Plaxo invitations. Examples of automated broadcast notifications include Facebook notifications, FourSquare geo-location notifications and the failed Facebook Beacon system which broadcast users' product purchases to peers.

The viral product feature space is not limited to personalized referrals and automated broadcast notifications. Take for example hypertext embedding. By allowing anyone to embed a video or video link into their own website, weblog or social networking profile page, YouTube.com enables and encourages uses of their product that facilitate sharing and peer-to-peer transfers of awareness. After friends, acquaintances and strangers watch videos embedded on websites and weblogs, they are shown a piece of code that enables them to embed the video in their own website and a link that enables them to share the video personally via email with their friends. Other prominent products also use this design feature. For example, Slide.com and RockYou.com allow users to create and embed slideshows of pictures or other content on their websites, weblogs and social networking profile pages. As other users browse those items on the web, hyperlinks allow them to download the products or services themselves. In this way, embedding enables users to spread awareness and to provide a path to product adoption for other users.

Generalized hypertext embedding requires more effort to post than notifications which are automatically generated on behalf of the user, but once embedded reach many people. Personalized hypertext embedding, such as profile box installations on Facebook are more personalized than generalized hypertext embedding because they target a user's personal social network rather than the general population of Internet browsers. Collaborative bookmarking sites like Delicious.com are personalized but also include an element of algorithmic activity. In that sense the user is only partly responsible for activating the feature and so we place collaborative bookmarking to the right of hypertext embedding as it is more personalized. The automated targeted notifications box represents a feature that could exist if notifications were

targeted toward specific individuals using collaborative filtering. As we do not know of any features that combine these two processes, we have used a dotted line to mark the box on the figure.

In summary, viral features range along the activity dimension of the viral product feature space from active to passive and along the personalization dimension from personalized to broadcast. Active-personalized viral features are generally considered to be more effective at promoting product contagion because personalized targeted messages are thought to be more persuasive. On the other hand, they require more effort which may curtail their use. The relative effectiveness of these viral features is ultimately an empirical question. We therefore designed and conducted a randomized experiment to estimate the effects of these viral product design features on product adoption and use.

3. Empirical Methods

3.1. Identification of Peer Influence in Social Networks

Evaluating the effects of viral product design features on social contagion is difficult because peer effects and WOM are typically endogenous. Several sources of bias in both cross sectional and longitudinal data on interactions and outcomes among peers can confound assessments of peer influence and social contagion including simultaneity (Godes and Mayzlin 2004), unobserved heterogeneity (Van den Bulte and Lilien 2001), truncation (Van den Bulte and Iyengar 2010), homophily (Aral et al. 2009), time-varying factors (Bemmaor 1994, Van den Bulte and Lilien 2001), and other contextual and correlated effects (Manski 1993). If uncorrected, theses biases can lead researchers to incorrectly attribute observed correlations to the influence of individuals on their peers resulting in misinterpretations of the treatment effects of viral marketing campaigns or viral product design choices.

Several approaches to the identification of peer effects have been proposed in various literatures including peer effects models and extended spatial autoregressive models (e.g. Bramoulle et al 2009, Kelejian and Prucha 1998, Lee 2003, 2007, Oestreicher-Singer and Sundararajan 2008), actor-oriented models (e.g. Snijders et al. 2006), instrumental variables methods based on natural experiments (e.g. Sacredote 2001, Tucker 2008), dynamic matched sample estimation (Aral et al. 2009), structural models

(e.g. Ghose and Han 2010) and ad hoc approaches (Christakis and Fowler 2007). However, randomized trials are believed to be the most effective way to obtain unbiased estimates of causal peer effects (Duflo et al 2006, Falk and Heckman 2009).

The logic of randomization is quite simple. In estimating the expected average effect of a treatment on a population of individuals, we cannot observe the expected outcome for a subject in the treatment group had she not been treated. Since in reality most individuals exposed to a treatment typically differ from those who are not, comparing the treated to the untreated without random assignment of the treatment creates a selection bias that reflects differences in the potential untreated outcomes of treatment and comparison groups. Randomization solves this problem because individuals assigned to the treatment and control groups differ in expectation only through their exposure to the treatment (Duflo et. al. 2006). If the potential outcomes of an individual are unrelated to the treatment status of any other individual (Angrist, Imbens and Rubin 1996),[3] simple OLS estimation provides unbiased estimates of the treatment that are internally valid (Duflo et. al. 2006: 8).

3.2. Experimental Design and Procedures

We partnered with a firm that develops commercial applications hosted on the popular social networking website Facebook.com to elicit data on the peer influence effects of enabling viral features using a commercial application built for use on the Facebook platform. Facebook is an ideal environment in which to study peer influence for four reasons. First, experiments on Facebook can capture natural user behavior and do not suffer from a potential loss of external validity that may affect laboratory experiments in which users are removed from their normal daily routines. Second, experiments conducted on Facebook can tap the broad audience of Facebook's user base which consists of hundreds of millions of individuals that interact on a daily basis and collectively participate in tens of billions of relationships. Third, such experiments can log detailed digital records of users' online representations and interactions,

[3] This is known as the "Stable Unit Treatment Value Assumption" (see Angrist, Imbens and Rubin 1996).

such as demographics, preferences, social networks, online behavior, and product adoption decisions. Fourth, the Facebook application development environment can be leveraged to control experimental treatment conditions in exacting detail.

The application we studied provides users the opportunity to share information and opinions about movies, actors, directors and the film industry in general. The firm designed multiple experimental versions of the application in which *personalized invitations* and *broadcast notifications* were either enabled or disabled, and randomly assigned adopting users to various experimental and control conditions. When a user adopted the application, they were randomly assigned to one of the two treatment conditions or the baseline control condition, and the application collected their personal attributes and preferences from their Facebook profiles, as well as data on their social networks and the personal attributes and preferences of their network neighbors.[4]

The basic experimental design enabled experimental group users to use passive-broadcast and active-personalized viral messaging capabilities to exchange viral messages with their neighbors, while disabling these features for the baseline control group. The application then recorded data on the use of these viral features by experimental group users, as well as click stream data on recipient responses to viral messages, and their subsequent adoption and use of the application for all neighbors of experimental and control group users. When an individual adopted as a result or peer influence, their treatment status was also randomized to ensure that the Stable Unit Treatment Value Assumption held (Angrist, Imbens and Rubin 1996). This facilitated analysis of the average treatment effect of enabling viral messaging capabilities on peer adoption and network propagation and allowed detailed analysis of the relative effectiveness of different viral messaging channels, as well as exploration of the mechanisms by which a particular viral channel influenced recipient behavior.

Facebook allows application developers to implement a variety of viral features that send communications to Facebook peers through well-defined channels. The two primary viral features that were

[4] Facebook allows users to specify privacy settings that may restrict an application's access to some or part of their profile. This is unlikely to have a significant effect on the study, as it is estimated that less than 2% of Facebook users alter default privacy settings (Gross et al 2005).

implemented in the application and enabled or disabled for the experimental treatment conditions are described below and illustrated using the Flixster Facebook application, an example application that is similar to the one used to conduct the experiment.[5]

Automated Broadcast Notifications. When enabled, notifications are generated automatically when an application user performs certain actions within the application, such as declaring a favorite movie or writing a movie review. When notifications are generated, they are distributed to a random subset of an application user's peers and displayed in a status bar at the bottom of the peers' Facebook environment as shown in Figure 2. When a peer clicks on the notification, they are taken to an application canvas page where they are given the option to install the application. Because no explicit action is necessary above and beyond the typical use of the application, notifications are classified as low effort on the activity dimension of the viral feature space. Furthermore, because notifications are randomly distributed to a Facebook user's peers and are not accompanied by a personalized message, they are classified as low personalization (broadcast) in the viral feature space.

Figure 2 About Here

Personalized Referrals or Invitations (Invites). When enabled, invites allow an application user to send a personalized invitation to their Facebook peers, inviting them to install the application. A peer then receives the invitation in their Facebook inbox and may click on a referral link contained within the invitation. If they do so, they are taken to the application canvas page where they are given the opportunity to install the application. This process is illustrated in Figure 3. As each invite requires a conscious and deliberate action on the part of the application user above and beyond typical application use, they are classified as higher effort (activity) than notifications in the viral feature space. Furthermore, because invites are targeted to specific Facebook peers and allow the inclusion of a personalized message, they are classified as higher personalization than notifications in the viral feature space.

*** Figure 3 About Here ***

[5] Due to confidentiality considerations, we do not reveal the application or firm.

The experimental design consisted of three treatment groups into which adopters of the application were randomly assigned: *baseline*, *passive-broadcast*, *active-personalized*. Users assigned to the baseline treatment group received a version of the application in which both notifications and invites were disabled. In the passive-broadcast treatment group (passive), only notifications were enabled. In the active-personalized treatment group (active), both notifications and invites were enabled. There were no other differences between baseline, passive and active applications. Throughout the experiment, each adopter of the application was randomly assigned to a treatment group according to the proportions displayed in Table 1. The proportion of users assigned to the baseline was chosen in agreement with the application developer to obtain a population size sufficient to establish a comparative baseline, while limiting potential adverse effects on the overall diffusion of the product that were deemed undesirable by the application developer.[6]

*** Table 1 About Here ***

Throughout the experiment detailed logs of application user activity, adoption times, viral feature use, peer response, and application user and peer profile data were recorded. Additionally, social network relationships for application adopters and mutual ties between peers of application users were recorded. Our experimental design enabled us to measure the effect of the treatment on the adoption response of peers of treated users as displayed in Figure 4. We took care to minimize contamination and leakage effects and describe our methods with regard to these and other considerations in detail in the Appendix.

*** Figure 4 About Here ***

Recruitment. At the launch of the experiment, an advertising campaign was designed in collaboration with a second Facebook advertising firm to recruit a population of application users. The advertising firm operates as an ad-exchange agency that delivers advertisements through dedicated advertising spaces within Facebook and Facebook applications that span a broad range of contexts and user bases.

[6] The developer feared too many baseline users could stunt the viral diffusion of the application, which made us even more interested to test whether these features actually caused social contagion. They therefore insisted that the number of baseline users be limited. Limiting baseline users should in no way bias our results as the proportion of baseline users to either treatment group is constant across treatments. Limiting baseline users should only make our estimates more conservative in that analyses comparing a treatment group to baseline will have less power.

The advertising campaign was designed to reach a representative audience of Facebook users and advertisements were displayed to users through advertising space within Facebook and within existing Facebook applications. The campaign was conducted in three waves throughout the duration of the experiment and cost a total of $6000 to recruit 9687 usable experimental subjects, or 62 cents per recruit.[7] The number of impressions, clicks, and installation responses are displayed in Table 2. Summary statistics of the recruited study population are described in § 4. Comparisons to published demographic statistics indicate the sample is representative of the typical Facebook user (see Appendix). Application diffusion in three randomly selected baseline, passive and active users' local networks is shown in Figure 5.

*** Table 2 and Figure 5 About Here ***

4. Analysis and Results

4.1. Data and Descriptive Statistics

The randomized experiment was conducted over a 44 day period during which 9687 users adopted the application with 405 users randomly assigned to the baseline control group, 4600 users randomly assigned to the passive-broadcast treatment group, and 4682 users randomly assigned to the active-personalized treatment group. Users in these groups collectively had 1.4M distinct peers in their local social networks and sent a total of 70,140 viral messages to their peers, resulting in 992 peer adoptions and 682 peer adoptions in direct response to viral messages. Three main observations arise from consideration of the summary statistics of the resultant data displayed in Table 3.

*** Table 3 About Here ***

First, assignment to control and treatment groups was clearly random with no significant mean or distributional differences between users assigned to baseline, passive-broadcast, and active-personalized treatments in terms of their age, gender, network degree (number of Facebook friends), and level of Facebook activity (number of Facebook wall posts), confirming the integrity of the randomization procedure.

[7] The cost per recruited user is several times smaller than the cost-per-user associated with recruitment for lab-based experiments. The low cost of recruitment makes online experiments an excellent source of experimental data.

Second, while their demographics and Facebook activity patterns were the same, measures of peer response in the network neighborhoods of treated users differed significantly across the treatment and control populations. T-tests show that the number and percentage of peer adopters in a user's local network are significantly higher for treated populations than for the baseline population. For example, the number of peer adopters in a treated user's local network is roughly seven times greater for users that received the passive-broadcast treatment and ten times greater for users that received the active-personalized treatment as compared to that of users that received the baseline treatment. Similarly, the percentage of adopters in a user's local network is roughly 450% higher for users that received the passive-broadcast treatment and 750% higher for users that received the active-personalized treatment than in the networks of users that received the baseline treatment. Measures of the speed of adoption in a treated user's local network, as indicated by the time to the first, second, third and fourth adoption events reveal that the treatments increase the rate of adoption in a treated user's local network. For example, the time to the first adopter is roughly 200% shorter for users that received the passive-broadcast treatment and roughly 300% shorter for users that received the active-personalized treatment as compared to users that received the baseline treatment. The extent to which the effect of the treatment leads to adoption beyond a user's immediate local network can be measured by the maximal diffusion depth – the maximum network distance from a treated user to any peer adopter in a linked chain of adoptions. The average maximal diffusion depth is approximately 360% greater for users that received the passive-broadcast treatment and approximately 450% greater for users that received the active-personalized treatment as compared to users that received the baseline treatment. T-tests reveal these differences to be highly significant.

Finally, the extent to which treatment leads to increased application use is measured by users' average application activity. Average application activity is roughly 130% higher for users that received the passive-broadcast treatment and 140% higher for users that received the active-personalized treatment than for users that received the baseline treatment. Two possible mechanisms could explain this increase in treated user activity. First, it could be that a more viral application is simply more interesting and that this directly drives increased application use. Alternatively, it could be that application virality encour-

ages peers of adopters to join them in application use, creating positive network externalities that inspire users to use the application more when their friends are using it. We explore these alternatives and estimate more formal models of peer influence and social contagion in the next several sections.

4.2. Model Specification

Our main statistical approach uses hazard modeling, which is the standard technique for assessing contagion in economics, marketing, and sociology literatures (e.g. Coleman 1967, Van den Bulte and Lilien 2001, Iyengar et al 2009, 2010, Nam et al 2010). This approach typically represents the hazard of adoption of individual *i* at time *t* as a function of individual characteristics and social influence:

$$\lambda\ (t, x, w, y) = f\,(x_i(t)\gamma, \beta \sum_j w_{ij} y_j(t))\,,$$

where $\lambda\ (t)$ represents the baseline hazard of adoption; $x_i(t)$ is a vector of variables unrelated to social influence that affect *i*'s adoption decision; w_{ij} is the social exposure of *i* to peer *j*; $y_j(t)$ is the adoption status of peer *j* at time *t*; and γ and β are parameters to be estimated.

Hazard rate models and binary choice models with duration dependence, which can be derived from utility theory and threshold based network models (Van den Bulte and Lilien 1999), are typically used to estimate such relationships (e.g. Van den Bulte and Lilien 2001, Manchanda et al 2008). However, our circumstances require a slightly different approach as we are interested in estimating the treatment effects of randomly assigned viral features on the adoption of peers in the local networks of focal experimental and control users, rather than the effects of focal users' social environments on their own adoption decisions. We therefore estimate the peer effects of the treatment 'outward' from an individual to their peers rather than estimating the effects of an individual's social environment 'inward' on their own adoption hazard. In order to estimate the effect of a randomly assigned application on the adoption of peers, working from the individual outward to the social environment is the only option. Controlled "treatments" of each user's social environment are too complex and costly to be accomplished reliably in the field and observation of the diffusion of the product requires estimation of the hazards of the adoption of peers, and

of the subsequent adoption of peers of peers. An 'inside-out' strategy estimating the effects of treatment on adoption in a user's social environment (rather than estimating the effects of the 'outside' social environment 'inward' on the user) is therefore the most appropriate modeling approach.

Our approach compares the hazards of adoption in the social environments of users treated with passive and active viral applications to the hazards of adoption in the social environments of users treated with the baseline application. The analysis therefore involves "multiple failure time" data in which multiple failures can occur for the same subject over time (Holmberg 2002). In our case, we want to estimate the hazard of multiple occurrences of peer adoption in the local networks of treated and untreated users as a function of their exposure to different viral features. In multiple failure time data, failure times are correlated within cluster (in our case within users' local networks), violating the independence of failure times assumption required in traditional survival analysis (Ezell et al 2003). The simplest way to analyze multiple failure data is to examine 'time to first event' and several studies in the contagion literature take this approach (Iyengar et al 2010). Other studies estimate the time to the first event and each subsequent event separately, which by construction assumes each sequential adoption event is equal and indistinguishable from the last (Anderson and Gill 1982). However, these specifications overlook potentially relevant data and fail to consider the cascading diffusion effects of multiple adoption events in a network, such as the presence of non-linear network effects or other non-linearities inherent in diffusion processes. We therefore employ a variance-corrected proportional hazards approach which adjusts the covariance matrix of the estimators in the model to account for the lack of independence among the multiple clustered failure times in the data, but allows the baseline hazards to vary by adoption event in order to account for the possibility that adoption hazards vary across stages of a diffusion process from first peer adopters to second peer adopters and so on.

Failure times in our adoption data are ordered, meaning there is a natural sequential ordering of event times such that the time of the first adoption in a local network by definition precedes the time of the second adoption and so on. If t_{ik} is the adoption time for the k^{th} adoption in i's network, adoption

times are sequential such that $t_{ik} \geq t_{ik-1}$. As we observe time stamped adoption of the application in minutes and seconds, our data are ordered sequentially and no two events happen at the same time. Another important characteristic of the process that produced these data is that the number of adoption events K_i for individual *i* is a random component of the data generating process and is therefore informative of the distribution of recurrence times (Chang and Wang 1999). As the social process of contagion can be affected by prior adoptions in a local network, for instance if network externalities are present, we assume that the baseline hazard function varies over adoption occurrences, such that it differs from first adoption to second adoption to third adoption and so forth. We therefore estimate the following variance-corrected stratified proportional hazards model:

$$\lambda_k(t, X_{ki}) = \lambda_{0k}(t)e^{X_{ki}\beta},$$

where stratification occurs over the *K* adoption events, $\lambda_{0k}(t)$ represents the baseline hazard of the k^{th} adoption event (*i*'s k^{th} friend adopting); X_{ki} represents a vector of covariates affecting the adoption of *i*'s neighbors (including *i*'s viral treatment status (active, passive or baseline), a measure of *i*'s level of activity on the application (Application Activity), peer notifications sent (Notifications), and invites sent (Invites); and β is a vector of unknown parameters to be estimated (Prentice et al. 1981). We assume *i*'s k^{th} friend does not adopt until their $k-1$ friend adopts as this is the case for all our data. Therefore the conditional risk set at time *t* for event *k* consists of all subjects under observation at time *t* who have experienced a $k-1$ adoption event (Cleves 1999). We estimate β using standard maximum likelihood estimation and adjust the covariance matrix to account for non-independence across individuals *i* using the following robust covariance matrix where G is a matrix of group efficient residuals:

$$V = I^{-1}G'GI^{-1}$$

Results are presented in Table 4. Robustness to different model specifications is shown in the Appendix.

*** Table 4 About Here ***

4.3. Effects of Viral Product Design on Peer Influence and Social Contagion

Table 4, Model 1 displays the average treatment effects of passive-broadcast and active-personalized viral treatments on peer influence and social contagion in the local networks of treated users above and beyond control group users who received the baseline application. Users of the passive-broadcast application experienced a 246% increase in the rate of application adoption by peers compared to the baseline group, while adding active-personalized viral messaging capabilities only generated an additional 98% increase (active-personalized users experienced a 344% increase over the baseline group). Models 2-4 decompose the variance in local network adoption rates explained by these treatments by estimating how intermediate variables such as overall application activity, notifications and invites explain the resultant increases in peer adoption. Model 3 shows that a significant amount of the treatment effects are explained by correlated increases in users' use of the application and the viral messages their use generates. Users assigned to passive-broadcast and active-personalized applications use their applications more and send more messages (invites and notifications) that generate greater peer adoption in their local networks. Model 4 reveals that invites have a greater marginal impact on the adoption rate of peers than notifications. One additional personal invite increases the rate of peer adoption by 6%, while one additional notification increases the rate of peer adoption by 2% on average, confirming that more personalized active features have a greater marginal impact on the rate of peer adoption per message than passive broadcast features.

The click stream data, which records each time stamped viral message sent and any response to it by peers of experimental and control users corroborate these results. Table 5 displays the number of invitations and notifications sent, the responses to those messages that resulted in click through installations of the application and the resultant adoption rate per message. Invitations, which require the most effort by the user and which are targeted specifically to recipients chosen by the user, are the least used but the most effective per message in creating peer influence and social contagion. Notifications, which require the least effort and are automatically sent to randomly selected peers, generate more messages, but are also less effective per message in converting new users.

*** Table 5 About Here ***

These results together confirm the main findings of the study: viral product design features do in fact generate econometrically identifiable peer influence and social contagion effects. Features that require more activity on the part of the user and are more personalized to recipients create greater marginal increases in the likelihood of adoption per message, but also generate fewer messages resulting in less total peer adoption in the network.

*** Figure 6 About Here ***

Figures 6a) and 6b) plot the cumulative peer adoptions and the fractions of adopters in the local networks of baseline, passive and active treatment users, while 6d) plots the Kaplan-Meier survival estimates for baseline, passive and active treatments respectively.[8] Susceptible peers of users in the passive-broadcast viral treatment group had an approximately seven-fold higher fraction of adopters in their local networks compared to baseline users. Susceptible peers of users in the active-personalized treatment group had over a ten-fold increase in adoption fraction compared to users in the baseline group, and an additional 1.5-fold increase in adoption fraction over peers of users in the passive viral treatment group. These graphs confirm that viral feature design has an economically significant impact on the diffusion of product adoption. Figure 6c) shows that treated users also use the application more than baseline users, suggesting that positive network externalities may be driving social contagion, an explanation we explore in more depth in the next section.

[8] Figure 3b) plots the fraction of susceptible peers that adopt the application t days after they become susceptible in active-personalized, passive-broadcast and baseline treatment and control groups, while Figure 3a) shows the cumulative adoption in each group. To assess the effect of the treatment group on the adoption of application user's peers through any influence-mediating channel, we identify the time of susceptibility to influence for all peers of buy-in users. To account for fixed-time effects, we look at the adoption response of all susceptible peers *t* days after they first became susceptible. We define the adoption fraction as, $A_f(t)$:

$$A_f(t) = \frac{\text{Number of susceptible peers that have adopted t days after becoming susceptible}}{\text{Number of peers that are still susceptible t days after becoming susceptible}}$$

and we plot the adoption fraction as a function of *t* for peers of buy-in users assigned to the baseline, passive, and active viral treatment groups.

4.4. Mechanisms Driving Social Contagion

Although the randomized experiment provides unbiased estimates of social contagion effects caused by viral product design features, several different social mechanisms could explain how viral features create social contagion. For instance, network externalities could generate a positive feedback loop of use and additional peer adoption (Van den Bulte and Stremersch 2004). It could also be that the features themselves make the application more interesting and therefore simultaneously drive application use and peer adoption creating a spurious correlation between peer adoption and product use. Several dimensions of our data shed light on the mechanisms linking viral features to contagion and whether network externalities are driving our results.

First, the hazard rate of adoption is increasing over adoption events in our data, implying a reinforcement effect of prior adoptions on the likelihood of future adoption (Feller 1943, Arbous and Kerrich 1951, Coleman 1964, Allison 1980, Holden 1986, Van den Bulte and Stremersch 2004). The hazard rate of adoption increases faster than exponentially for the first several adoption events then more slowly, suggesting that reinforcement is approximately constant over peer adoptions (Allison 1980). Although we interpret these results with caution because "one cannot distinguish between contagion and heterogeneity only on the basis of statistical properties of the distributional form" (Taibleson 1974: 878), the fact that the hazard rate of adoption is increasing in the *k* adoption events is consistent with a reinforcement effect of prior adoptions on future adoption.

Table 6 and Figure 7 About Here

Second, active-personalized viral features are associated with more sustained product use than both passive-broadcast viral features and the baseline model (see Figure 6c). Table 7 presents results which confirm these findings in OLS regression models estimating correlations between viral features, the number of peer adopters and application use.[9] Table 7 shows that feature inclusion, peer adoption and

[9] As we do not have the benefit of randomization beyond the original application features, we rely on observational analyses to examine the underlying mechanisms that may explain social contagion. Although we interpret these results as strictly correlation rather than causation we feel they shed a good deal of light on the possible mechanisms that explain the relationship between viral features and social contagion.

application use are correlated. Users of active-personalized and passive-broadcast versions of the application exhibit more application use (Table 7, Model 1) and these results hold when controlling for observable differences in users' overall Facebook activity (Table 7, Model 2). Controlling for treatment status, degree and overall Facebook activity, the number of peer adopters a user has is positively associated with their sustained use of the application (Table 7, Models 3). These results are consistent with the existence of positive network externalities – as more of their peers adopt the application users are more engaged and use the application more.[10]

Table 7 About Here

However, several alternative explanations could also account for these results. We depict the potential causal relationships between these covariates in Figure 8. We assessed the potential causal relationships between these three covariates given our data and what our analysis means for the existence of network externalities. The randomized trial confirms that viral features cause peer adoption. We also note that because features are randomized and not controlled by the user, no other covariate can drive the existence of features. If adoption by a user's peers causes an increase in the user's use of the application, then the existence of network externalities is confirmed.

Figure 8 About Here

We first consider the possibility that omitted variables (such as unobserved user heterogeneity) are simultaneously driving application use and peer adoption. While this scenario is possible, because feature inclusion is randomized, the distribution of such unobserved covariates must be the same across different treatment groups and so, omitted variables cannot produce the discrepancy in peer adoption and application use across the different treatment groups that we observe in Figures 6a) and 6c). It is possible that an unobserved covariate that must first be activated by the existence of a particular feature in order to drive peer adoption and application use could exist; however, this too is unsupported by the evidence. As

[10] Although passive-broadcast viral features are associated with more product use than the baseline product in the beginning of the study, this association disappears over time (see Figure 6c). That active-personalized features (which enable users to actively select and invite specific peers) is associated with sustained product use while passive-broadcast viral features (which notify random peers) is not may even suggest a direct network effect from interacting with specific peers with whom users most want to interact on the application.

we observe correlation between adoption and use beyond that which is explained by use of the invite and notifications features themselves (see Table 7), it seems unlikely that a user characteristic that simultaneously drives peer adoption and use would be activated by a viral feature that the user does not use.

Cases (a) and (b) in Figure 8 represent correlations that are not supported by the evidence in Table 7. Cases (a) and (b) are inconsistent with the discrepancy in application use between users that received different viral treatments observed in Figure 6c and Table 7. Cases (c) and (d) in Figure 8 represent the network externalities mechanism, where peer adoption drives increased use of the application by the original adopter. Cases (e) and (f) represent a 'demand effect' in which the correlation between features and application use is explained by an increase in utility for the application user from the existence of features. Finally cases (g) and (h) represent the co-existence of both network externalities and demand effects. If our data reject cases (e) and (f) we have good reason to suspect that network externalities are driving social contagion (at least to some extent).

One variant of the demand effects argument is that the viral features themselves increase users' utility from sustained application use and simultaneously encourage peer adoption. If the mere presence of the invite and notification features was correlated with both application use and peer adoption, and if peer adoption itself was not driving use, the correlation between the number of peer adopters and application use should disappear once we control for the use of invites and notifications. However, when we hold constant application use associated with both notifications and invites there is still a strong positive relationship between the number of peer adopters and application use (Table 7, Model 3), implying that additional application use, beyond that explained by use of the viral features themselves, is correlated with more peer adoptions. This suggests that demand effects do not fully explain the correlation between peer adoption and use. It could still be that the viral state of the application itself makes the application more interesting, however for this story to be true given our data users would have to derive utility from the viral features without actually using them. When the viral states are entered into the regression they significantly predict application activity in the expected directions and magnitudes (Table 7, Model 1). However, when the number of peer adopters is entered into the analysis, these relationships disappear com-

pletely (Table 7, Models 3 and 4), indicating that the viral state of the application or the utility from simply being able to notify or invite friends alone does not predict application use controlling for the amount of peer adoption.

Another alternative explanation is that there is a demand effect from the existence of the viral features which inspires peer adoption – peers' expected utility from adopting the application is higher because they expect to have access to viral features. For example, it could be that because a user received an invitation, they adopted the application because they valued the ability to invite others and expected to have this feature in the product they adopted, creating a demand effect from the expected utility of having viral features turned on. It seems unlikely that a significant portion of the expected utility from adopting the application comes from the existence of the viral features rather than the functions of the application itself. However, to address this alternative demand effects explanation we performed additional analysis. On average, application use by peer adopters is a reasonable proxy for their satisfaction with the product - the extent to which their expectations regarding the product conform to the product they actually received upon adoption. We therefore examined the application use of peers that adopted through a response to a viral message and divided these peers into two groups: those that received (through random assignment) a version of the product with the ability to send viral messages of the type they received from their influencing peer and those who received a version of the product without the ability to send viral messages of the type they received from their influencing peer. T-tests show that the use of the application by those who received applications with the features they would expect to receive and those that were "disappointed" (so to speak) by not receiving an application with the features they would expect to receive show no significant differences in application use (*t-statistic* = 0.9054; S.D. = 8.0377). Given this evidence we feel it is unlikely that adoption is explained by the attractiveness of the applications with viral features.

Table 8 presents correlates of application diffusion which corroborate results of the randomized trial. Models 4-6 confirm that peers of initial adopters also use the application because diffusion depth depends on peers' (and peers of peers) using of the application. Active-personalized and passive-broadcast treatments significantly increase average diffusion depth and these effects are again explained

by application use and the viral features themselves (Model 6). Results in Model 3 also corroborate hazard model estimates, confirming that invitations have a higher marginal impact on peer adoption than notifications. Invitations are three times more effective per message than notifications in inspiring peer adoption on average. Taken together, the distributional properties of the baseline hazards of adoption events and evidence of a strong correlation between the number of adopter friends and application use suggest that network externalities accelerate contagion – as more of a user's friends adopt, they use they application more creating a positive feedback loop.

5. Discussion

We conducted a large scale randomized experiment testing the effectiveness of viral product design features in creating social contagion. We find that viral product design has econometrically identifiable impacts on peer influence and social contagion in new product diffusion. Results of our randomized trial suggest that designing viral features into products can increase social contagion by up to 400%. Surprisingly designing products with passive-broadcast viral messaging capabilities generates more total peer influence and social contagion than adding active-personalized viral messaging capabilities. Although active-personalized messaging is more effective in encouraging adoption per message and is correlated with more user engagement and sustained product use, it is used less often and therefore generates less total peer adoption in the network than passive-broadcast messaging. Data on the distributional form of the diffusion process and on product use are consistent with the existence of positive network externalities which reinforce peer adoption. These results have broad implications for managers attempting to promote viral product diffusion and for theories of social contagion, opinion leadership and viral product design.

First, our estimates imply that viral product design may be more effective in encouraging new product adoption than traditional marketing strategies alone. Though our experiment does not include a direct comparison to traditional marketing, both notifications and invitations outperformed published statistics on click through rates (CTR) for traditional banner advertising and email marketing campaigns. The 1% CTR on notifications outperforms the CTR for traditional banner advertising (which range from

.10 - .20% in publicly available statistics) and invitations are ten times as effective as traditional banner ads. Compared to email campaign CTR (which range from 2-6% in publicly available statistics), invitations are again more effective at 10%.[11] These comparisons show viral channels to be more effective at generating higher response rates than traditional digital advertising channels.

We also asked the directors of the firm with whom we partnered about their feature implementation and customer acquisition costs and learned that invites can be implemented for a low total cost less than $600. Since implementing viral features incurs a low one-time fixed cost and the expected return is proportional to the increase in adopters the feature generates, viral product design may be a more cost effective strategy than increased spending on traditional digital advertising which incurs costs proportional to impressions or clicks. It may be however that the success of viral product design efforts depends on traditional advertising to the extent that an initial base of users is needed to implement viral marketing. It is also important to consider the social cost of viral messages. Bombarding users with a cacophony of messages from their peers may reduce the effectiveness of viral product design strategies and the overall quality of the user experience. Future work should theorize and estimate the costs of viral product design more comprehensively and consider the implications of both marginal revenue and marginal cost on optimal product design.

Second, given that active-personalized features are more marginally effective but less globally effective than passive-broadcast features, a natural question is how managers can optimize the effectiveness of these viral features? As the main limitation of active-personalized features is that high effort costs curtail their use, one solution may be to couple active-personalized features with referral incentives that encourage their use (Biyalogorsky et al 2001, Libai et al 2003). Optimally designed incentive strategies could encourage users to generate more personalized referrals and to target and personalize viral messages more effectively. It may also be possible to improve the low marginal effectiveness of passive-

[11] Click-through rates on banner ads have declined from 0.33% to 0.19% from 2004 through 2008 (Forrester, Go Big or Go Home Advertising, 2009); DoubleClick reports that in 2008 average CTR in the US was 0.10% for banner ads (DoubleClick, Benchmark Report, 2009). For email campaigns, estimated CTR in 2008 and 2009 remained stable at 5.9%. (Epsilon, October 2009), while Mailer Mailer reports average CTR on email campaigns at 2.80% (Mailer Mailer, June 2009) and Web Market Central reports a "2-3%" CTR (Web Market Central 2007).

broadcast features by automatically targeting and personalizing broadcast messages in smart ways. If there is a social cost to viral messages, product and platform developers could seek to limit impersonal messages in adaptive ways that are tied to the effectiveness of the messages themselves.

Third, in the presence of viral features, network externalities drive a positive feedback loop in which product use drives peer adoption and peer adoption in turn drives product use. Managers should seek to enable this positive feedback loop by designing strong direct and indirect network externalities into their products. That managers should seek to maximize sustained use and minimize customer churn may be obvious, however the effects of sustained use and user churn on product diffusion may be less so. Interactions between network externalities, sustained use and customer churn may change over a product's lifecycle and may vary across products. More work on the relationships between social contagion, sustained use and customer churn over products and product lifecycles will help clarify when viral marketing is most effective.

Our work also has implications for platform developers that seek to benefit from social interactions taking place on their platforms. Platform developers can enable and constrain the viral features that operate in their ecosystem and engineer the user experience to increase sharing, interaction and the virality of products. If one considers the social cost of "spammy interactions," continuous redesign of social interaction features for the purpose of optimizing the user experience is likely a rational, profit maximizing strategy. Optimization from the platform developer's perspective may take place over different variables and constraints, but improving the virality and use of the applications available to users is likely an important goal.

As with any study of social contagion that only considers one product or service, the nature of the product is important. We selected an application that is representative of typical application products developed for the Facebook environment. However, there are some products to which our results may not generalize. We also have coarse data on friendship ties. Our data only record whether two people are Facebook friends. These ties are undirected and unweighted, and there is little indication of their strength or purpose. Future work should analyze how different types of ties and the strength of ties mediate the ef-

fects of viral product features on peer outcomes. We also do not observe the content of communication, nor do we observe channels of communication outside of Facebook. Previous research demonstrates the importance of observing and analyzing content in network data (Aral and Van Alstyne 2009). These omissions limit our ability to explore the social mechanisms and processes that underlie contagion in more depth. Understanding which individuals are influential and which are susceptible in social contagions that arise due to the use of viral product design features may also prove important. Experimental methods can make great strides in helping to resolve recent debates about whether the influentials hypothesis holds (Gladwell 2000, Watts and Dodds 2007). In addition, an important element that has been left in the background in our work is the importance of network structure. As our focus is on the relational elements of social contagion (how one person may influence another) we do not foreground network structure. Important questions around the disproportional influence of hubs and especially the global diffusion properties of viral feature influence propagation are as yet unexplored (Goldenberg et al 2009).

An important goal for managers and researchers is to understand optimal viral product design strategies taking into consideration sustained product use, network externalities, social and economic costs and incentives and the marginal effectiveness of different viral features. The difficulty however is in determining what works and what does not. Numerous statistical challenges prevent clean causal estimation of the relationships between interventions and outcomes in social processes and the likely effects of changes in product design features and platform policy. But, IT-based products and platforms provide a natural arena for randomized experimentation. Given the low cost of conducting such experiments, the rapid development and testing of viral design features and the winner take all nature of markets with network externalities (e.g. Katz and Shapiro 1985), we believe that this type of experimentation and testing will only increase in the future and eventually become commonplace in the development of many IT-based products, platforms and beyond.

7. Conclusion

We examined how firms can create word of mouth peer influence and social contagion by incorporating viral features into the design of their products. We then designed and conducted a randomized field experiment testing the effectiveness of passive-broadcast and active-personalized viral messaging capabilities in creating peer influence and social contagion among the 1.4 million friends of 9,687 experimental users of Facebook.com. Results show that viral product features can in fact generate econometrically identifiable peer influence and social contagion effects. Features that require more activity on the part of the user and are more personalized to recipients create greater marginal increases in the likelihood of adoption per message, but generate fewer total messages creating countervailing effects on peer influence. On average, passive-broadcast viral messaging capabilities, which are less personalized but also require less user effort, generate a 246% increase in local peer influence and social contagion while adding active-personalized viral messaging capabilities, which are more personalized but require more user effort, only generates an additional 98% increase. Although active-personalized messaging is more effective in encouraging adoption per message and is correlated with more sustained product use, it is used less often and therefore generates less total peer adoption in the network than passive-broadcast messaging. Our data suggest that initial peer adoptions in users' local networks create network externalities that accelerate contagion. These results shed light on how viral products can be designed to generate social contagion and how randomized trials can be used to identify peer influence in networks.

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Tables and Figures

Table 1. Stratification Across Treatment Groups		
Baseline Control	**Passive-broadcast Treatment**	**Active-personalized Treatment**
5%	47.5%	47.5%

Table 2. Recruitment Statistics Describing the Initial Advertising Campaign				
Wave	**Impressions**	**Clicks**	**Advertising Related Installs**	**Installs**
1 (Day 0)	18,264,600	12,334	3,072	3,714
2 (Day 15)	20,912,880	25,709	2,619	3,474
3 (Day 20)	19,957,640	7,624	3,219	4,039
Total	59,135,120	45,667	8,910	11,227

Table 3. Summary Statistics and Mean Comparisons of Active, Passive and Baseline Users						
	1	2	3	4	5	6
	Baseline (N = 405)	Passive (N = 4600)	Active (N = 4682)	*t*-statistic (B-P)	*t*-statistic (B-A)	*t*-statistic (P-A)
	Mean (SD)	*Mean (SD)*	*Mean (SD)*	*t-statistic (SE)*	*t-statistic (SE)*	*t-statistic (SE)*
Age	31.51 (13.80)	30.81 (13.31)	29.94 (13.27)	.46 (13.35)	1.03 (13.31)	1.45 (13.24)
Gender (1=Male)	.25 (.44)	.33 (.47)	.32 (.47)	-1.57 (.47)	-1.42 (.46)	.40 (.47)
Degree†	171.79 (223.88)	170.25 (278.64)	166.97 (248.77)	.09 (275.13)	.32 (247.15)	.55 (263.82)
Number of Facebook Wall Posts	40.52 (79.89)	36.45 (94.16)	37.07 (246.76)	.46 (93.11)	.15 (238.20)	-.09 (188.31)
Number of Adopters in User's Local Network	.01 (.12)	0.07 (.35)	0.10 (.44)	-2.84*** (.34)	-3.60*** (.43)	-3.64*** (.40)
Percentage of Adopters in User's Local Network	.02 (.002)	.09 (.01)	.15 (.01)	-1.92* (.01)	-2.35** (.01)	-2.83*** (.01)
Maximum Diffusion Depth	.01 (.11)	.04 (.22)	.05 (.24)	-2.53* (.21)	-3.01*** (.24)	-1.98*** (.23)
Time to 1st Adopter	9.40 (9.71)	4.77 (8.04)	3.17 (6.72)	1.27 (8.07)	2.04** (6.77)	2.45*** (7.30)
Time to 2nd Adopter	---	5.23 (8.17)	4.43 (6.97)	---	---	0.58 (7.45)
Time to 3rd Adopter	---	5.29 (8.07)	3.04 (5.25)	---	---	1.08 (6.33)
Time to 4th Adopter	---	6 (5.83)	1.17 (1.12)	---	---	2.84*** (3.58)
Application Activity	3.17 (4.59)	4.17 (7.24)	4.56 (8.98)	-2.54** (7.08)	-2.89*** (8.73)	-2.20* (8.16)

Notes: ***p<.001; **p<.05; *p<.10; † K-S Tests of Degree Distribution Differences: B-P: .04, p = .80, N.S.; B-A: .04, p = .79, N.S.; .01, p = .94, N.S.

Table 4: Variance-Corrected Proportional Hazards of Contagion in Networks of Baseline, Passive and Active Treatment Groups				
	1	2	3	4
	Hazard Ratio (SE)	*Hazard Ratio (SE)*	*Hazard Ratio (SE)*	*Hazard Ratio (SE)*
Viral State = Passive	3.46*** (1.18)	3.35*** (1.15)	2.50** (.86)	2.51** (.86)
Viral State = Active	4.44*** (1.64)	4.21*** (1.56)	3.33*** (1.24)	3.31*** (1.24)
Application Activity		1.02*** (.004)	1.02*** (.003)	1.02*** (.003)
Notifications			1.02*** (.002)	1.02*** (.002)
Invites				1.06** (.028)
Log Likelihood	-4694.359	-4631.795	-4544.845	-4542.577
X^2 (d.f)	19.34*** (2)	57.41*** (3)	298.78*** (4)	307.47*** (5)
Observations	3929	3929	3929	3929
Notes: ***p<.001; **p<.05; *p<.10; Variance Corrected Proportional Hazards Models are estimated with robust standard errors clustered around users' local network neighborhoods.				

Table 5: Click Stream Analysis of Responses to Viral Messages and Adoption			
	1	2	3
	Messages Sent	*Adoptions via Click Through Installation*	*Adoption Rate (Marginal Impact)*
Invitations	160	16	.10
Notifications	69980	666	.01

Table 6: Baseline Hazards Over *k* Events λ_{0k} (*k* = 1…6)				
	1	2	3	4
	Mean (SD)	*Min*	*Max*	*N*
λ_{01}	.0002 (.0001)	.0001	.001	523
λ_{02}	.002 (.001)	.001	.013	128
λ_{03}	.015 (.024)	.005	.14	42
λ_{04}	.034 (.010)	.021	.054	20
λ_{05}	.046 (.008)	.037	.067	15
λ_{06}	.099 (.044)	.053	.14	7

Table 7. Correlates of Application Use				
	1	2	3	4
	Application Use	Application Use	Application Use	Application Use
	Beta (SE)	*Beta (SE)*	*Beta (SE)*	*Beta (SE)*
Viral State = Passive	.129* (.074)	.112 (.079)	.062 (.076)	-.037 (.074)
Viral State = Active	.190*** (.074)	.171** (.079)	.091 (.076)	-.006 (.074)
Degree	-.0001 (.0001)	-.0001 (.0001)	-.0002** (.0001)	-.0002** (.0001)
Facebook Activity		.054*** (.016)	.042*** (.015)	.026* (.014)
Notifications				.022*** (.001)
Invites				.055** (.024)
Number of Adopters			.607*** (.030)	.360*** (.031)
F Value (d.f.)	3.51*** (3)	4.87*** (4)	83.54*** (5)	128.92*** (7)
R^2	.002	.003	.07	.14
Observations	6310	5766	5766	5766
Notes: ***p<.001; **p<.05; *p<.10. Models are estimated with OLS regression with robust standard errors clustered around users' local network neighborhoods.				

Table 8. Correlates of Application Diffusion						
	1	2	3	4	5	6
	Number of Adopters	Number of Adopters	Number of Adopters	Diffusion Depth	Diffusion Depth	Diffusion Depth
	Beta (SE)	*Beta (SE)*	*Beta (SE)*	*Beta (SE)*	*Beta (SE)*	*Beta (SE)*
Viral State = Passive	.078** (.031)	.084** (.033)	.020 (.059)	.045** (.0178)	.048*** (..019)	.020 (.018)
Viral State = Active	.119*** (.031)	.131*** (.032)	.059* (.030)	.057*** (.018)	.063*** (.019)	.033* (.018)
Degree	.0001*** (.00002)	.0001** (.00003)	.0001** (.00002)	.0001*** (.00001)	.00004** (.00002)	.00003** (.00001)
Facebook Activity		.019*** (.006)	.006 (.006)		.013*** (.004)	.007** (.004)
Application Use			.061*** (.005)			.021*** (.003)
Notifications			.010*** (.0004)			.005*** (.0002)
Invites			.035*** (.010)			-.003 (.006)
F Value (d.f.)	12.20*** (3)	11.18*** (4)	157.94*** (7)	9.36*** (3)	10.11*** (4)	85.13*** (7)
R^2	.006	.007	.16	.004	.007	.09
Observations	8910	5766	5766	6310	5766	5766
Notes: ***p<.001; **p<.05; *p<.10. Models are estimated with OLS regression with robust standard errors clustered around users' local network neighborhoods.						

Figures.

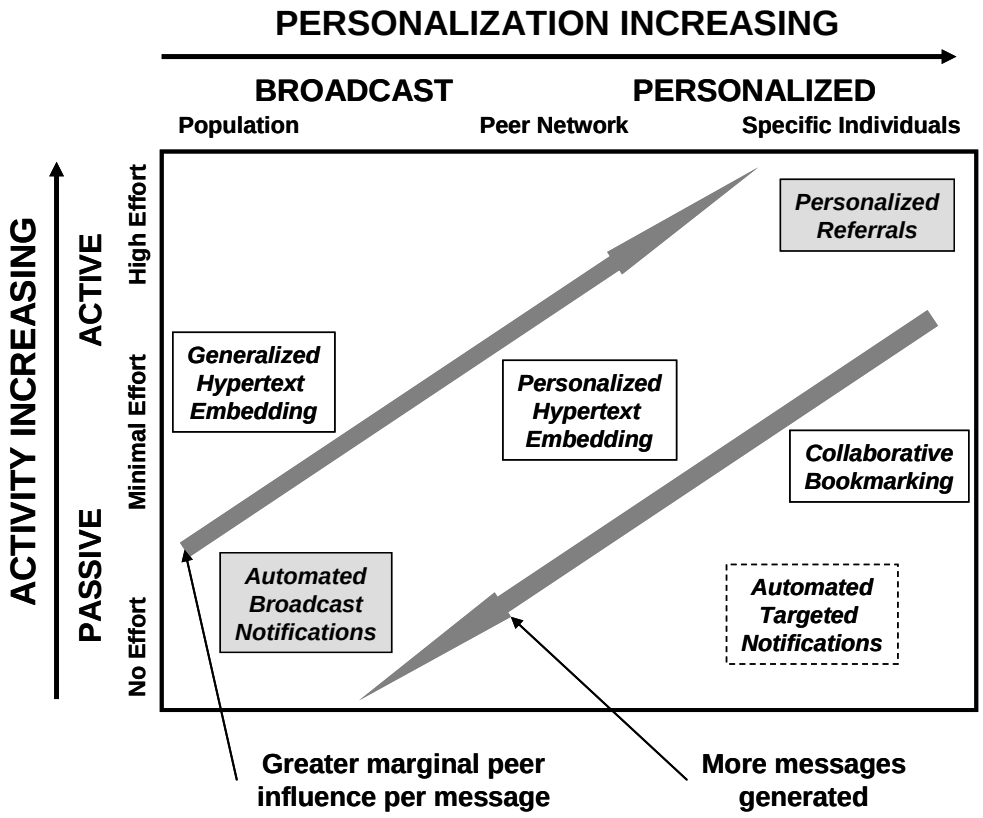

Figure 1. The Viral Product Feature Space.

Figure 2. A Graphical Example of the Interface and Process of the Notifications Feature.

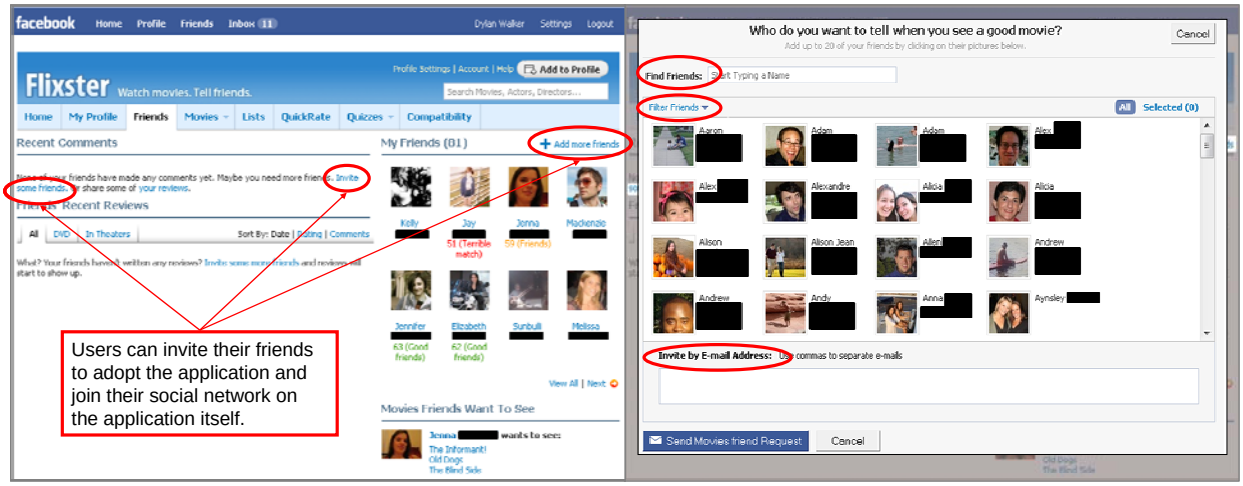

Figure 3. A Graphical Example of the Interface and Process of the Personal Referrals or Invitations Feature.

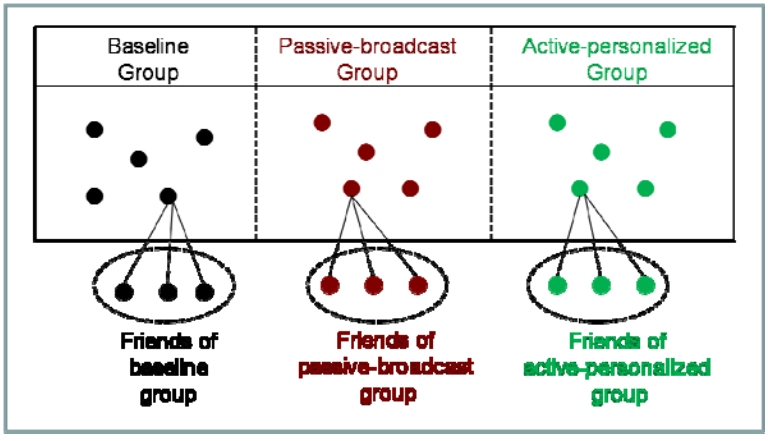

Figure 4. Graphical Representation of the Experimental Comparison

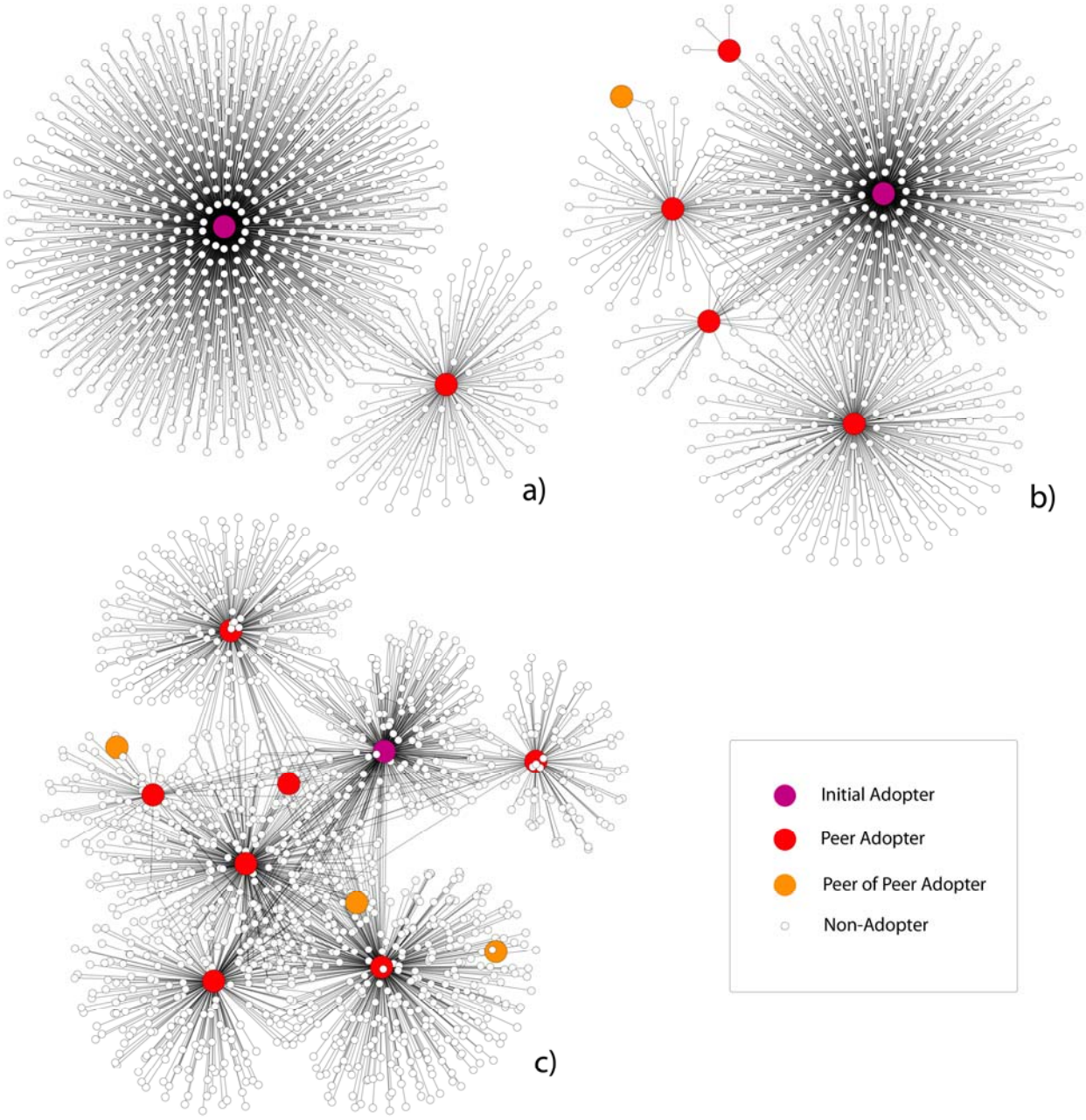

Figure 5. Three Representative Local Networks of a) Baseline, b) Passive and c) Active Users. The Initial experimental adopter (ego) is colored dark purple, peer adopters (friends of ego who adopted) are colored red, peer of peer adopters (friends of friends of ego who adopted) are colored orange. Non-adopters are white.

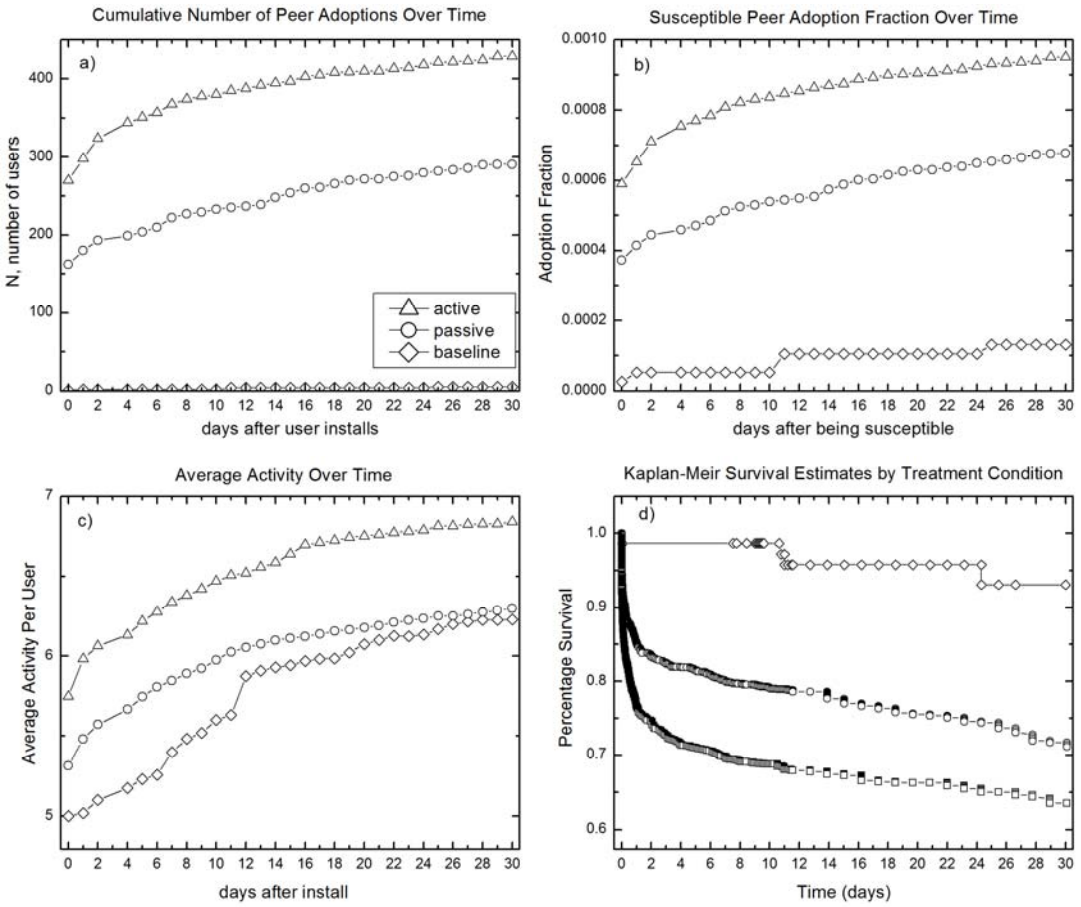

Figure 6. Plots a) the cumulative number of peer adoptions, b) the fraction of susceptible peer adopters, c) the average activity, and d) the Kaplan-Meier Survival Estimates over time for baseline, active and passive users.

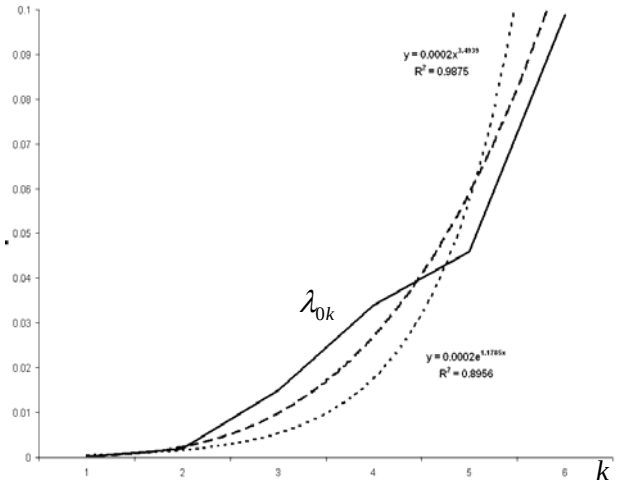

Figure 7. Baseline Hazards (λ_{0k}) for *k* = 1…6 fitted to an Exponential and a Power Function

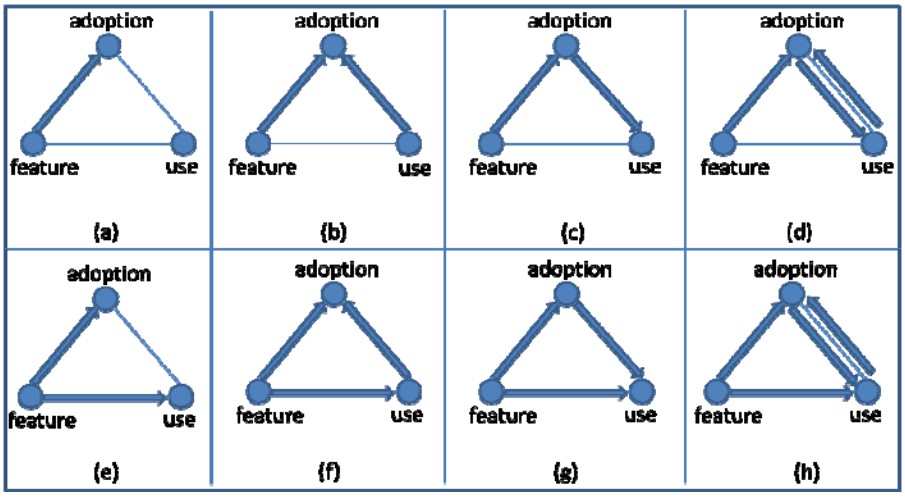

Figure 8: Possible causal relationships between the existence of application features, peer adoption, and application use. Arrows indicate causal direction.

Appendix

1. Inside-Out Design

Randomized trials are traditionally used to estimate the effect of a treatment on the treated. To study the effect of viral feature incorporation on product adoption outcomes, we instead examine the effect of treatment on the peers of treated application users. The difference in these approaches is illustrated in Figure A1. Arrows indicate the potential flow of influence that the experiment is designed to detect. The solid blue circle in the center represents the treated user and the red outlines indicate measurements of treatment effects. In social network environments, a conventional approach is infeasible because it is difficult to comprehensively control the network environments of each user in the study population. It is feasible however to treat a user and observe the effect of treatment on the outcomes of their peers.

The strength of our approach lies in its ability to capture the effect of any form of influence-mediating communication channels between the treated user and her peers including effects that arise through influence-mediating communication channels beyond those that can be explicitly recorded. For example, a treated user could communicate with and influence their peers through offline interactions such as face-to-face communications or telephone conversations as well as unrecorded online communications such as email or external chat conversations. Because we measure the response of peers regardless of how they may or may not have been influenced by treated users, we are able to capture the effect of unrecorded influence-mediating communications on peer adoption.

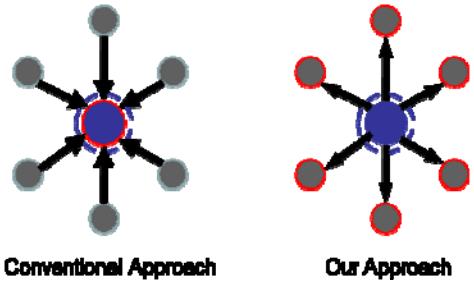

Figure A1

2. Preventing Selection Effects

Selection effects could occur when a user chooses to adopt the experimental application through the recruitment campaign or when they adopt in response to a viral message. We took steps to mitigate and measure both possibilities. The recruitment campaign was designed to reach a representative audience of Facebook users and advertisements were displayed to users through advertising space within Facebook and within existing Facebook applications. Establishing to what extent the recruited population is repre-

sentative of the general Facebook population is somewhat challenging because Facebook does not officially publish demographic statistics of their user base. However, through the use of a recently released social targeting advertisement service provided by Facebook, it is possible to obtain some official demographic statistics. Age and gender demographics sampled through this API and published online by istrategylabs.com are compared to the same demographic ranges for recruited study population users in Figure 2. Though our sample has a slightly higher percentage of women than the Facebook population and users in our sample have a slightly higher average degree, the demographics of our study population are comparable to that of the broader Facebook population and the published Facebook demographics fall within one standard deviation of study population sample means.

In addition to issues of selection surrounding the population of recruited users, application users that adopt due to peer influence may be subject to selection effects and may be fundamentally different from application users that adopted via initial recruitment. It could be that users who use the viral features and peers of users who use viral features are systematically different from randomly selected Facebook users. We avoid these sources of selection bias in our analyses by only considering initially recruited users in the randomized treatment group to which they were assigned. Peers of recruited users only contribute to local network peer adoption of originally recruited users and are not themselves used as test subjects.

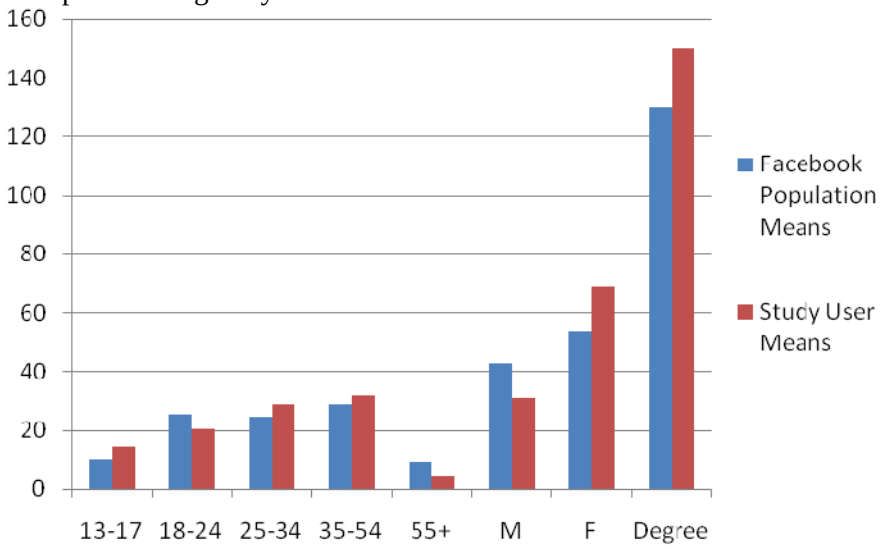

Figure A2

3. Preventing Leakage and Contamination

In randomized trials in network environments, users assigned to different treatment groups may not be strictly isolated from one another. This raises the concern that information leakage through indirect network pathways may contaminate the results of the study. It is important to note that in traditional studies, whether or not the network is measured, relationships may still exist between treatment and control populations that create leakage effects. One benefit of our design is that we systematically observe how individuals in the study are connected, enabling us to measure and prevent leakage.

Several factors reduce the likelihood that leakage is affecting our results. First, because treatment assignment is randomized, any leakage will be uncorrelated with treatment assignment and cannot account for the observed differences in responses to treatments. While it is possible that leakage will on average provide some common information to peers of treated users uniformly across the treatment designations, this effect should only serve to make our estimates across treatment groups more conservative as leakage should reduce differences between control and treatment groups. Second, information flows between individuals in a network are typically proportional to network distance and the decay of information flow and quality with network distance is well documented (Wu et al 2004, Aral et al 2007). While all users may be connected through long friendship paths, leakage will diminish over successive hops in each path.

Nonetheless, leakage effects could downward bias our estimates of treatment effects toward zero and we therefore take several steps to prevent leakage. First, in hazard rate models, we only examine peers of initially recruited adopters. In addition to avoiding potential selection issues mentioned above, this also excludes individuals (and their potential adopter peers) that adopt in chains within a local neighborhood lessening leakage effects. Such individuals are likely to share more and shorter indirect paths with existing adopters than a randomly chosen peer, as a consequence of clustering and mutuality (Newman 2003).

Second, we account for users with multiple treated peers (of similar and/or different treatments). Existence of peers of multiple treated application users leads to two potential complications. First, users may be peers of multiple treated users from different treatment groups making it impossible to link their treatment effects to a single treatment. Second, peers of multiple treated users that belong to the same treatment group are clearly classified as peers of either baseline, active-personalized or passive-broadcast users; however measurements of their response may be incorrectly estimated as a consequence of being subject to influence from multiple treated friends. A peer with multiple treated friends in a given viral treatment group may exhibit an adoption outcome or time to adoption that is systematically different from those of peers with only one treated friend. These two scenarios are displayed in Figure A3.

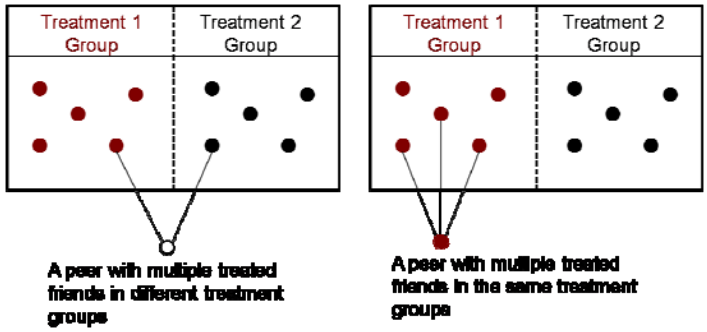

Figure A3

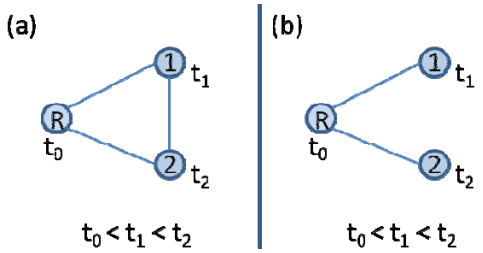

Figure A4

The nature of treatment randomization does not allow us to simultaneously guarantee that all treated friends of a peer will receive the same treatment. Consequently we treat peers with multiple treated friends as contaminated as soon as they become so and exclude them from our analysis. This procedure could underestimate the effect of clusters of adoption on the time to adoption or number of adopters in a local network neighborhood, however if this is the case, it will do so in a manner that is the same for all treatment types. Furthermore, as treatment groups are randomized, there can be no systematic correlation between the type of treatment received by a user and that received by her subsequent adopter peer.

The procedure that we adopt for designating a peer as contaminated is detailed in Figure A4. The initially recruited adopter, labeled R, adopts at time (t_0). Two peers of user R, labeled 1 and 2, adopt at subsequent times t_1 and t_2 respectively. In panel (a) for times $t > t_1$, peer 2 has multiple treated peers (R and 1) that may have been assigned different treatments. Peer 2 is therefore considered contaminated for times $t > t_1$. In panel (b) a similar situation occurs, but no link exists between peers 1 and 2 and consequently neither user is considered contaminated. In our analysis, when a peer is designated as contaminated, she is removed from the hazard rate model for subsequent time periods. This procedure appropriately retains the maximal empirical support for hazard rate estimation and parameterizes our ignorance of what might happen subsequent to a user's contamination. The right-censoring of contaminated subjects has become standard practice in randomized clinical trials where a patient in a randomized treatment group undergoes some characteristic change that is beyond the researcher's control. Furthermore, by including right-censored observations in our data rather than truncating the data, we avoid problems caused by data truncation that could lead to spurious evidence of contagion (Van den Bulte and Iyengar, 2010).

We note that the exclusion of peers with multiple treated friends does not preclude measurement of network externalities. Peers of treated users that become adopters but are not connected are considered uncontaminated and are included in our analysis. For two peers of a treated user that are connected and eventually become adopters, the initial peer adopter is included in our analysis and only the peer that sub-

sequently adopts is considered contaminated and excluded for all times subsequent to contamination. These procedures enable a tightly controlled randomized trial of peer influence that addresses the potential for selection and leakage effects.

4. Robustness Checks for Different Hazard Model Specifications

There are a limited number of survival models that apply to contexts with multiple failures. Among these models, the variance corrected proportional hazards model reported in the paper is the most appropriate specification given the structure of our data and the parameters we estimate. However, we also checked multiple other hazard model specifications in order to test the robustness of our results to changes in model specification and estimation strategy. For good reviews of appropriate specifications of survival models in multiple failure data we recommend Wei and Glidden (1997) and Ezell et al (2003).

Table A1 reports results of different hazard model specifications, all of which are similar to our own. We report the original variance corrected proportional hazards model specification detailed in the paper in Column 1. Column 2 reports an accelerated failure time model with a log-logistic survival distribution. Column 3 reports an exponential regression with log relative-hazard form. Column 4 reports results from a traditional Anderson-Gill model. Column 5 introduces a time-dependent covariate measuring the number of prior adopters to the traditional Anderson-Gill specification to capture the dependence structure among recurrence times which in our original model is captured by the adoption event strata *k*. Column 6 reports a Prentice William and Peterson proportional hazards specification with time dependent strata. Finally, Column 7 reports results of a Wei Lin and Weissfeld marginal risk set model. We note that all specifications produce similar results. However, we are most confident in our original specification which is best suited to our context and data.

Table A1: Robustness Checks for Different Model Specifications

	1	2	3	4	5	6	7
Specification	*VCSPHM*	*AFT*	*EXP*	*AG_1*	*AG_2*	*PWP*	*WLW*
Viral State = Passive	2.51** (.86)	-2.41** (1.16)	1.01*** (.35)	2.60*** (.91)	2.54*** (.87)	2.51*** (.865)	2.00* (.78)
Viral State = Active	3.31*** (1.24)	-3.66*** (1.22)	1.30*** (.39)	3.51*** (1.36)	3.30*** (1.26)	3.31*** (1.24)	2.62** (1.02)
Application Activity	1.02*** (.003)	-.119*** (.039)	.015*** (.003)	1.02*** (.003)	1.02*** (.003)	1.02*** (.003)	1.00 (.002)
Notifications	1.02*** (.002)	-.115*** (.010)	.025*** (.002)	1.02*** (.002)	1.02*** (.001)	1.02*** (.002)	1.01*** (.002)
Invites	1.06** (.028)	-.198 (.259)	.090** (.036)	1.07* (.037)	1.06** (.035)	1.06** (.027)	1.02 (.018)
Prior Adopters					1.50*** (.062)		
Time Dummies	NO	YES	YES	NO	NO	NO	NO
Log Likelihood	-4542.58	-2826.32	-4136.53	-5254.17	-5212.88	-4542.56	-4561.56
X^2 (d.f)	307.47*** (5)	--	1656.60*** (11)	412.65*** (5)	435.88*** (6)	307.60*** (5)	109.17*** (5)
Observations	3929	3929	3929	3929	3929	3929	3929

Notes: ***p<.001; **p<.05; *p<.10; Standard errors are clusters around users' local network neighborhoods. "*VCSPHM*": Variance Corrected Stratified Proportional Hazards Model as specified and reported in the paper; "*AFT*": Accelerated Failure Time Model with Log-Logistic Survival Distribution; "*EXP*": Exponential Regression with Log Relative-Hazard form; "*AG*": Anderson-Gill Model; "*PWP*": Prentice William and Peterson Proportional Hazards Model with Time Dependent Strata; "*WLW*": Wei Lin and Weissfeld Marginal Risk Set Model.

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