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Identifying Influential and Susceptible Members of Social Networks

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Identifying social influence in networks is critical to understanding how behaviors spread. We present a method for identifying influence and susceptibility in networks that avoids biases in traditional estimates of social contagion by leveraging in vivo randomized experimentation. Estimation in a representative sample of 1.3M Facebook users showed that younger users are more susceptible than older users, men are more influential than women, women influence men more than they influence other women, and married individuals are the least susceptible to influence in the decision to adopt the product we studied. Analysis of influence and susceptibility together with network structure reveals that influential individuals are less susceptible to influence than non-influential individuals and that they cluster in the network, which suggests that influential people with influential friends help spread this product.

Peer effects are empirically elusive in the social sciences. Scholars in disciplines as diverse as economics, sociology, psychology, finance, and management are interested in whether children's peers influence their education, whether workers' colleagues influence their productivity, whether happiness, obesity and smoking are 'contagious' and whether risky behaviors spread via peer influence. The success of intervention strategies in these domains depends on the robustness of estimates of the degree to which contagion is at work during a social epidemic (*1*, *2*). Robust estimation of peer effects is also critical to understanding whether new social media technologies magnify peer influence in product demand, voter turnout, and political mobilization or protest.

The recent availability of population-scale networked datasets generated by email, instant messaging, mobile phone communications, and online social networks enable novel investigations of the diffusion of information and influence in networks (*3*–*9*). Unfortunately, identifying influence in these networks is difficult because estimation is confounded by homophily (the tendency for individuals to choose friends with similar tastes and preferences (*10*, *11*), and thus for preferences to be correlated amongst friends), confounding effects (the tendency for connected individuals to be exposed to the same external stimuli), simultaneity (the tendency for connected individuals to co-influence each other and to behave similarly at approximately the same time), and other factors (*1*, *2*, *10*, *12*–*17*). (Note that assortativity is a more general term for homophily that is used to describe the same phenomena for any kind of node). Although some new methods separate peer influence from homophily and confounding factors in observational data (*11*), controlling for unobservable factors such as latent homophily (correlation amongst unobserved drivers of preferences amongst friends) remains difficult without exogenous variation in adoption probabilities across individuals (*18*). Fortunately, randomized experiments provide a more robust means of identifying causal peer effects in networks (*19*–*21*).

One particularly controversial argument in the peer effects literature is the "influentials" hypothesis—the idea that influential individuals catalyze the diffusion of opinions, behaviors, innovations and products in society (*22*, *23*). Despite the popular appeal of this argument, a variety of theoretical models suggest that susceptibility, not influence, is the key trait that drives social contagions (*24*–*28*). Little empirical evidence exists to adjudicate these claims. Understanding whether influence, susceptibility to influence, or a combination of the two drives social contagions, and accurately identifying influential and susceptible individuals in networks, could enable new behavioral interventions to affect obesity, smoking, exercise, fraud and the adoption of new products and services.

We conducted a randomized experiment to measure influence and susceptibility to influence in the product adoption decisions of a representative sample of 1.3 Million Facebook users by randomly manipulating influence-mediating messages sent from a commercial Facebook application that lets users share information and opinions about movies, actors, directors and the film industry. As users adopted and used the product, automated notifications of their activities were delivered to randomly selected peers in their local social networks. For example, when a user rated a movie on the application, a randomly selected subset of their Facebook friends were sent a message notifying them of the rating with a link to the canvas page describing the application and instructions on how to adopt it. As message recipients were randomly selected, treated and untreated peers of the application user only differed by their treatment status—the number of randomized messages they received. The experiment was conducted over 44 days during which 7730 product adopters sent 41,686 automated notifications to randomly chosen targets amongst their 1.3 million friends, resulting in 976 unique peer adoptions or a 13% increase in demand for the product relative to the number of initial adopters (see tables S1 to S4 and figs. S1 to S4).

Estimates of influence and susceptibility were obtained by modeling time to peer adoption as a function of the treatment—receiving influence mediating messages from peers. An influence-mediating message refers to any communication between peers that could conduct influence (*29*, *30*), for example, wearing a logo advertising a brand or recommending a product to a friend. Our method avoids several known sources of bias in influence identification by randomly manipulating who receives influence-mediating messages.

First, we avoid selection bias by randomizing whether and to whom influence-mediating messages are sent (table S5). In uncontrolled environments users may choose to send messages to peers who are more likely to like the product or to listen to their advice, which confounds estimates of susceptibility to influence by oversampling recipients who are more likely to respond positively. Second, our method eliminates bias created by homophily or assortativity in networks by randomizing the receipt of influence-mediating messages. Even latent homophily is controlled because similarity in unobserved attributes is equally represented across treatment groups. Third, the method controls for unobserved confounding factors because randomly chosen peers are equally likely to be exposed to external stimuli that affect adoption such as advertising campaigns or promotions. Fourth, automatically generated messages include identical information, eliminating heterogeneity in message content and valence which are known to impact responses to social influence (*31*). Differences in adoption between treatment groups can then be attributed solely to the number of influence-mediating messages they received.

Our statistical approach used hazard modeling, which is the standard technique for estimating social contagion in economics, marketing, and sociology [e.g., (*32*)]. However, we extended existing techniques to distinguish two types of peer adoption: *spontaneous adoption*—adoption that occurs in the absence of influence, and *influence-driven adoption*—adoption that occurs in response to persuasive messages. This extension is important because adoption outcomes cluster among peers even in the absence of influence as a consequence of homophily, assortativity, simultaneity and correlated effects (*11*, *12*). We estimate the average treatment effects of notifications by aggregating many individual experiments in which messages were randomized within the local networks of the original adopting users (tables S6 and S7).

To estimate the moderating effects of an individual *i*'s attributes on the influence they exert on their peer *j* and distinguish them from the moderating effects of *j*'s attributes on *j*'s susceptibility to influence, we estimate a continuous-time single-failure proportional hazards model. Survival models provide information about how quickly peers respond (rather than simply whether they respond) and correct for censoring of peer responses that may occur beyond the experiment's observation window. We specify the following model:

$$\lambda_j\left(t, X_i, X_j, N_j\right) = \lambda_0(t)\exp\left[N_j(t)\beta_N + X_i\beta^i_{\text{spont}} + X_j\beta^j_{\text{spont}} + N_j(t)X_i\beta_{\text{infl}} + N_j(t)X_j\beta_{\text{susc}}\right] \quad (1)$$

where λ_j is the hazard of a peer *j* of an application user *i* adopting the application (each peer *j* is associated with one and only one application user *i*), $\lambda_0(t)$ represents the baseline hazard, X_i represents a set of individual attributes of an application user *i*, X_j represents a set of individual attributes of peer *j*. $N_j(t)$ represents the number of notifications received by a peer *j* of application user *i*, as a function of time. $N_j(t)$ reflects the extent to which *j* has been exposed to influence mediating messages from their friend. β_N estimates the effect of receiving a notification on the likelihood of peer adoption, holding sender attributes constant. β^i_{spont} estimates the propensity for peers of an application user with attributes X_i to spontaneously adopt in the absence of influence ($N_j = 0$). β^j_{spont} estimates the propensity for a peer *j* with attributes X_j to spontaneously adopt in the absence of influence ($N_j = 0$). β_{infl} estimates the impact of an application user's attributes on her ability to influence her peer to adopt the application above and beyond the peer's propensity to adopt spontaneously. β_{susc} estimates the impact of a peer's attributes on her likelihood to adopt due to influence above and beyond her propensity to adopt spontaneously (for alternative specifications, robustness and goodness of fit, see table S8 and figs. S5 to S12).

Models of dyadic (two-party) relationships between influencers and potential susceptible test whether influence depends on characteristics of the relationship between a given pair, for example, whether women are more influential over men than men are over women. To estimate the effect of dyadic relationships, we employ the following continuous-time single-failure proportional hazards model:

$$\lambda_j\left(t, X_i, X_j, N_j\right) = \lambda_0(t)\exp\left[N_j(t)\beta_N + S\left(X_i, X_j\right)\beta^{i-j}_{\text{spont}} + N_j(t)S\left(X_i, X_j\right)\beta^{i\to j}_{\text{infl}}\right] \quad (2)$$

where X_i represents a set of the individual attributes of the sender, X_j represents a set of the individual attributes of peer *j* (the potential recipient), and $S(X_i, X_j)$ represents a set of dyadic covariates that characterize the joint attributes of the sender-recipient pair. Dyadic covariates estimate for example whether influence is stronger when the sender and recipient are the same or different genders. β_{spont} estimates the effect of a dyadic relationship between an application user *i* and her peer *j* on the tendency for the peer to adopt spontaneously. For example, when the dyadic relationship variable is an indicator of similarity (such as *same age*), β_{spont} captures the extent to which similarity on that dimension predicts the likelihood to spontaneously adopt, and represents the propensity to adopt due to preference similarity and other explanations for correlations in adoption likelihoods between peers that are not a result of influence. β_{infl} estimates the effect of the dyadic attribute (e.g., same age) on the degree to which a sender influences her recipient peer to adopt, above and beyond their likelihood to spontaneously adopt.

On average, susceptibility decreases with age (Fig. 1). People over the age of 31 are the least susceptible to influence (they have an 18% lower hazard of adopting the application upon receiving a notification than people who do not declare their age, $p < 0.05$) (the statistical significance of all estimates are derived from χ^2 tests). In contrast, people in the highest age quartile (> 31) are significantly more influential than people in the lowest age quartile (< 18). People over 31 have a 51% greater instantaneous likelihood of influencing their peers to adopt with an influence mediating message than people younger than 18 ($p < 0.05$).

Men are 49% more influential than women ($p < 0.05$), but women are 12% less susceptible to influence than men ($p < 0.05$). Single and married individuals are the most influential. Single individuals are significantly more influential than those who are in a relationship (113% more influential, $p < 0.05$) and those who report their relationship status as 'It's complicated' (128% more influential, $p < 0.05$). Married individuals are 140% more influential than those in a relationship ($p < 0.01$) and 158% more influential than those who report that 'It's complicated' ($p < 0.01$). Susceptibility increases with increasing relationship commitment until the point of marriage. The engaged are 53% more susceptible to influence than single people ($p < 0.05$), while married individuals are the least susceptible to influence (Married: N.S.). The engaged and those who report that "It's complicated" are the most susceptible to influence (Those who report that "It's complicated" are 111% more susceptible to influence than baseline users who do not report their relationship status on Facebook $p < 0.05$, and those who are engaged are 117% more susceptible than baseline users, $p < 0.001$).

People exert the most influence on peers of the same age (97% more influence on peers of the same age than the baseline, $p < 0.01$, Fig. 2). They also seem to exert more influence on younger peers than on older peers though this difference is not significant. In non-dyadic susceptibility models, we found that women were less susceptible to influence than men (Fig. 1). Dyadic models (Fig. 2) further reveal that women exert 46% more influence over men than over other women ($p = 0.01$). Finally, individuals in equally (and more) committed relationships than their peers (e.g., those who are married compared to those who are engaged, in a relationship or single) are significantly more influential (Equally Committed: 70% more influential than baseline, $p < 0.05$; More Committed: 101% more influential than baseline, $p < 0.05$).

Comparing spontaneous adoption hazards to influenced adoption hazards reveals the potential roles that different individuals play in the diffusion of a behavior (Fig. 3). For example, in the case of the movie product we studied, both single and married individuals adopt spontaneously more often than baseline users (Single: 31% more often, $p < 0.05$; Married: 36% more often, $p = 0.06$), are more influential than baseline users (Single: 71% more influential, $p < 0.01$; Married: 94% more influential, $p < 0.001$, from Fig. 1), and have peers who are no more likely to adopt spontaneously than the baseline (N.S.; N.S.). This suggests that influence exerted by single and married individuals positively contributes to this product's diffusion without any need to target them. On the other hand, women are poor candidates for targeted advertising because they are likely to adopt spontaneously and are 22% less influential on their peers than baseline ($p < 0.05$). Those who claim their relationship status is complicated are easily influenced by their peers to adopt (35% more susceptible than baseline, $p < 0.05$), but are not influential enough to spread the product further (N.S.). These results have implications for policies designed to promote or inhibit diffusion and illustrate the general utility of our method for informing intervention strategies, targeted advertising and policy making.

Figure 4 shows the joint distributions of influence and susceptibility in anetwork revealing the correlation of influence and susceptibility across all individuals and the assortativity of influence and susceptibility across all individuals and their peers in the network. We calculated individual influence and susceptibility scores as the product of the estimated hazard ratios of individuals' attributes for a broader sample of 12M users with 85M relationships. The analysis combines the estimated impact of each demographic attribute on influence and susceptibility to calculate individuals' overall influence and susceptibility scores. For example, a 35-year-old single female has an influence score equal to $\exp[\beta_{infl,>31} + \beta_{infl,single} + \beta_{infl,female}]$.

Our results indicated that highly influential individuals tend not to be susceptible, highly susceptible individuals tend not to be influential and almost no one is both highly influential and highly susceptible to influence (Fig. 4, Panel I). This implies that influential individuals are less likely to adopt the product as a consequence of natural influence processes (i.e.,in the absence of targeting), making targeting influentials with low propensities to adopt spontaneously a potentially viable promotion strategy. Second, the influentials and susceptibles hypotheses are orthogonal claims. Both influential individuals and non-influential individuals have approximately the same distribution of susceptibility to influence among their peers, meaning that being influential is not simply a consequence of having susceptible peers (Panel II). Both influence and susceptibility play a role in the peer-to-peer diffusion of the product. Combining studies of influence with studies of susceptibility will therefore likely improve our understanding of the diffusion of behavioral contagions. Third, there are more people with high influence scores than high susceptibility scores (Panel I), which suggests that, in our context, targeting should focus on the attributes of current adopters (e.g., giving individuals incentives to influence their peers) rather than attributes of their peers (e.g., giving individuals with susceptible peers incentives to adopt). Fourth, influentials cluster in the network. Panel III reveals the existence of influential individuals connected to other influential peers who are approximately twice as influential as baseline users. In contrast, we find a tendency for less susceptible users to cluster together and no clusters of highly susceptible users (Panel IV). The clustering of influentials suggests there may be a multiplier effect from infecting a high-influence individual. However, individuals with high influence also tend to have peers with low susceptibility, making predictions about which effect would dominate difficult without more evidence. Additional empirical and simulation studies should therefore examine how the assortativity of influence and susceptibility effect the diffusion of behaviors, products and diseases.

Analyzing the heat maps alone is not sufficient to identify optimal intervention targets because more information is needed about the network structure around candidate targets in each region. For example, an individual with high influence and high peer susceptibility in the upper right quadrant of Panel II may seem like a good target, but may be of low degree or may be isolated. The network figures to the left of the heat maps in Fig. 4 show the assortativity of influence and susceptibility in ego networks from different regions combined with information on their network structures, such as network degree and the distribution of influence and susceptibility across peers in the network. Analyzing networks in different regions of the heat maps, such as those displayed in Fig. 4, can suggest optimal targets. For example, node C is not only highly influential, highly susceptible and has peers who are themselves influential and susceptible, but is also of above average degree in its region and has many peers who are susceptible rather than one highly susceptible peer driving the average susceptibility in their network. These characteristics in combination make C a good target.

Our method is the first to use randomized experiments to identify influential and susceptible individuals in large social networks; however, the work does have limitations. Our method avoids bias by randomizing message recipient selection and holding message content constant. However, recipient selection and message content may be important aspects of influence and should therefore be estimated in future experiments. Furthermoreit is still not clear whether influence and susceptibility are generalized characteristics of individuals or rather depend on which product, behavior, or idea is diffusing. While our estimates should generalize to the diffusion of similar products, they are not conclusions about who is more or less influential in general. Future work will be needed to determine whether there is something "different" about people who do not provide some information (e.g., age) (table S1). Our experimental methods for influence identification however are generalizable and can be used to measure influence and susceptibility in the diffusion of other products and behaviors in a variety of settings.

Previous research has taken an individualistic view of influence—that someone's importance to the diffusion of a behavior depends only on their individual attributes or personal network characteristics. In contrast, our results show that the joint distributions of influence, susceptibility and the likelihood of spontaneous adoption in the local network around individuals together determine their importance to the propagation of behaviors. Future research should examine how the co-distribution of influence, susceptibility and dyadic induction in networks affects the diffusion of behaviors, the development of social contagions, and the effects of policies intended to promote or contain behavior change. More generally, our results show the potential of methods based on large scale in vivo randomized experiments to robustly estimate peer effects and identify influential and susceptible members of social networks.

References and Notes

1. S. Aral, Identifying social influence: A comment on opinion leadership and social contagion in new product diffusion. *Mark. Sci.* **30**, 217 (2011). doi:10.1287/mksc.1100.0596
2. S. Aral, D. Walker, Identifying social influence in networks using randomized experiments. *IEEE Intell. Syst.* **26**, 91 (2011). doi:10.1109/MIS.2011.89
3. G. Kossinets, D. J. Watts, Empirical analysis of an evolving social network. *Science* **311**, 88 (2006). doi:10.1126/science.1116869 Medline
4. D. Lazer *et al*., Computational social science. *Science* **323**, 721 (2009). doi:10.1126/science.1167742 Medline
5. N. Eagle, M. Macy, R. Claxton, Network diversity and economic development. *Science* **328**, 1029 (2010). doi:10.1126/science.1186605 Medline
6. S. A. Golder, M. W. Macy, Diurnal and seasonal mood vary with work, sleep, and daylength across diverse cultures. *Science* **333**, 1878 (2011). doi:10.1126/science.1202775 Medline
7. S. Aral, M. W. Van Alstyne, The diversity-bandwidth trade-off. *Am. J. Sociol.* **117**, 90 (2011). doi:10.1086/661238
8. E. Sun, I. Rosenn, C. Marlow, T. Lento, in *Proceedings of the Third International Conference on Weblogs and Social Media* (AAAI Press, Menlo Park, CA, 2009).
9. J. Leskovec, L. A. Adamic, B. A. Huberman, The dynamics of viral marketing. *ACM Trans. Web* 10.1145/1232722.1232727 (2007).
10. M. McPherson, L. Smith-Lovin, J. M. Cook, Birds of a feather: Homophily in social networks. *Annu. Rev. Sociol.* **27**, 415 (2001). doi:10.1146/annurev.soc.27.1.415
11. S. Aral, L. Muchnik, A. Sundararajan, Distinguishing influence-based contagion from homophily-driven diffusion in dynamic networks. *Proc. Natl. Acad. Sci. U.S.A.* **106**, 21544 (2009). doi:10.1073/pnas.0908800106 Medline
12. C. F. Manski, Identification problems in the social sciences. *Soc. Methodol.* **23**, 1 (1993).
13. S. Currarini, M. O. Jackson, P. Pin, Identifying the roles of race-based choice and chance in high school friendship network formation. *Proc. Natl. Acad. Sci. U.S.A.* **107**, 4857 (2010). doi:10.1073/pnas.0911793107 Medline
14. H. Noel, B. Nyhan, The "unfriending" problem: The consequences of homophily in friendship retention for causal estimates of social influence. *Soc.*

Networks **33**, 211 (2011). doi:10.1016/j.socnet.2011.05.003
15. W. Hartmann *et al*., Modeling social interactions: Identification, empirical methods and policy implications. *Mark. Lett.* **19**, 287 (2008). doi:10.1007/s11002-008-9048-z
16. N. A. Christakis, J. H. Fowler, The spread of obesity in a large social network over 32 years. *N. Engl. J. Med.* **357**, 370 (2007). doi:10.1056/NEJMsa066082 Medline
17. R. Lyons, The spread of evidence-poor medicine via flawed social-network analysis. *Stat. Polit. Policy* 10.2202/2151-7509.1024 (2011).
18. C. R. Shalizi, A. C. Thomas, Homophily and contagion are generically confounded in observational social network studies. *Sociol. Methods Res.* **40**, 211 (2011). doi:10.1177/0049124111404820 Medline
19. S. Aral, D. Walker, Creating social contagion through viral product design: A randomized trial of peer influence in networks. *Manage. Sci.* **57**, 1623 (2011). doi:10.1287/mnsc.1110.1421
20. D. Centola, The spread of behavior in an online social network experiment. *Science* **329**, 1194 (2010). doi:10.1126/science.1185231 Medline
21. S. Leider, M. M. Möbius, T. Rosenblat, Q.-A. Do, Directed altruism and enforced reciprocity in social networks. *Q. J. Econ.* **124**, 1815 (2009). doi:10.1162/qjec.2009.124.4.1815
22. E. Katz, The two-step flow of communication: An up-to-date report on an hypothesis. *Public Opin. Q.* **21**, 61 (1957). doi:10.1086/266687
23. T. W. Valente, *Network Models of the Diffusion of Innovations* (Hampton, Cresskill, NJ, 1995).
24. D. Kempe, J. Kleinberg, É. Tardos, in *Proceedings of the Ninth ACM SIGKDD International Conference on Knowledge Discovery and Data Mining* (Association for Computing Machinery, New York, 2003), pp. 137–146.
25. M. Granovetter, Threshold models of collective behavior. *Am. J. Sociol.* **83**, 1420 (1978). doi:10.1086/226707
26. D. J. Watts, P. S. Dodds, Influentials, networks, and public opinion formation. *J. Consum. Res.* **34**, 441 (2007). doi:10.1086/518527
27. P. S. Dodds, D. J. Watts, Universal behavior in a generalized model of contagion. *Phys. Rev. Lett.* **92**, 218701 (2004). doi:10.1103/PhysRevLett.92.218701 Medline
28. D. Centola, M. Macy, Complex contagions and the weakness of long ties. *Am. J. Sociol.* **113**, 702 (2007). doi:10.1086/521848
29. S. Aral, D. Walker, Social Science Research Network Working Paper Series, http://dx.doi.org/10.2139/ssrn.1564856 (2010).
30. D. Godes *et al*., *Mark. Lett.* **16**, 415 (2005).
31. C. Heath, C. Bell, E. Sternberg, Emotional selection in memes: The case of urban legends. *J. Pers. Soc. Psychol.* **81**, 1028 (2001). doi:10.1037/0022-3514.81.6.1028 Medline
32. R. Iyengar, C. Van den Bulte, T. W. Valente, *Marketing Sci.* 10.1287/mksc.1100.0566 (2010).

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Supplementary Materials
www.sciencemag.org/cgi/content/full/science.1215842/DC1
Materials and Methods
Figs. S1 to S12
Tables S1 to S9

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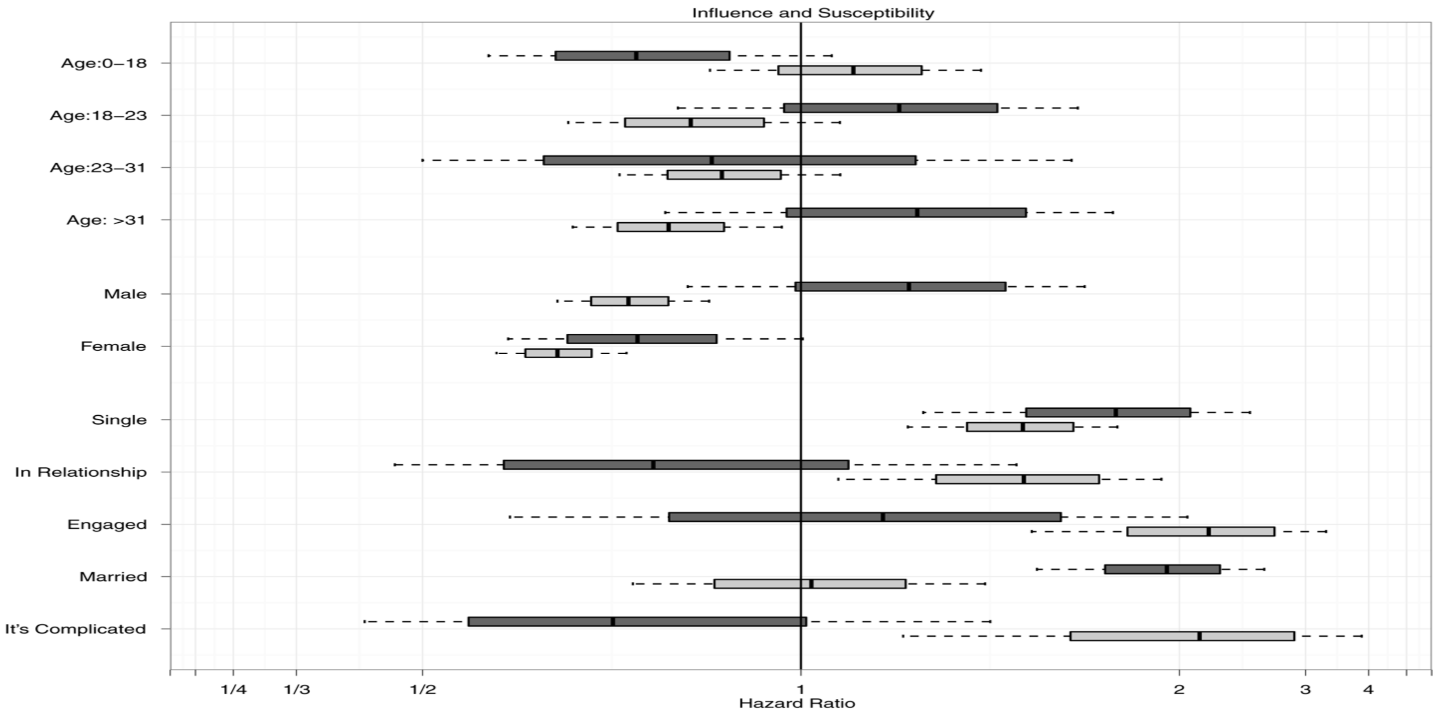

Fig. 1. Effects of age, gender, and relationship status on influence and susceptibility. Influence (dark grey) and susceptibility to influence (light grey) are shown along with standard errors (boxes) and 95% confidence intervals (whiskers). The figure displays hazard ratios (HR) representing the percent increase (HR > 1) or decrease (HR < 1) in adoption hazards associated with each attribute. Age is binned by quartiles. Each attribute is shown as a pair of estimates, one reflecting influence (dark grey) and the other susceptibility (light grey). Personal relationship status reflects the status of an individual's current romantic relationship and is specified on Facebook as: *Single, In a Relationship, Engaged, Married*, and *It's Complicated*. Estimates are shown relative to the baseline case for each attribute, which is the average for individuals who do not display that attribute in their online profile.

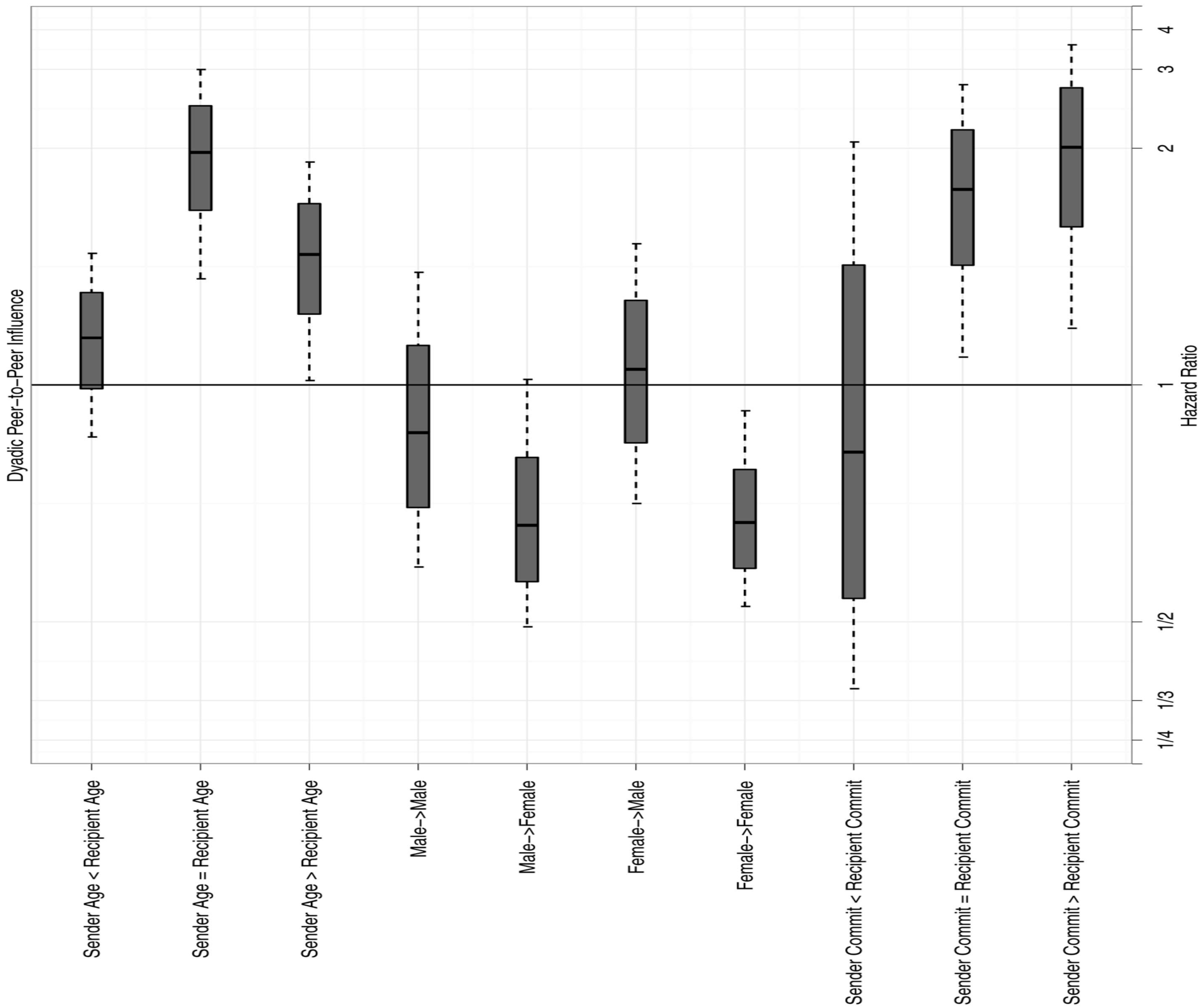

Fig. 2. Dyadic influence models involving age, gender and relationship status, Theresults include the relative age, gender similarity, and commitment level of the relationship status of senders and recipients along with standard errors (boxes) and 95% confidence intervals (whiskers). The figure displays hazard ratios (HR) representing the percent increase (HR > 1) or decrease (HR < 1) in adoption hazards associated with each attribute. The baseline case represents dyads in which the attribute being examined is unreported in the Facebook profile of one or both peers.

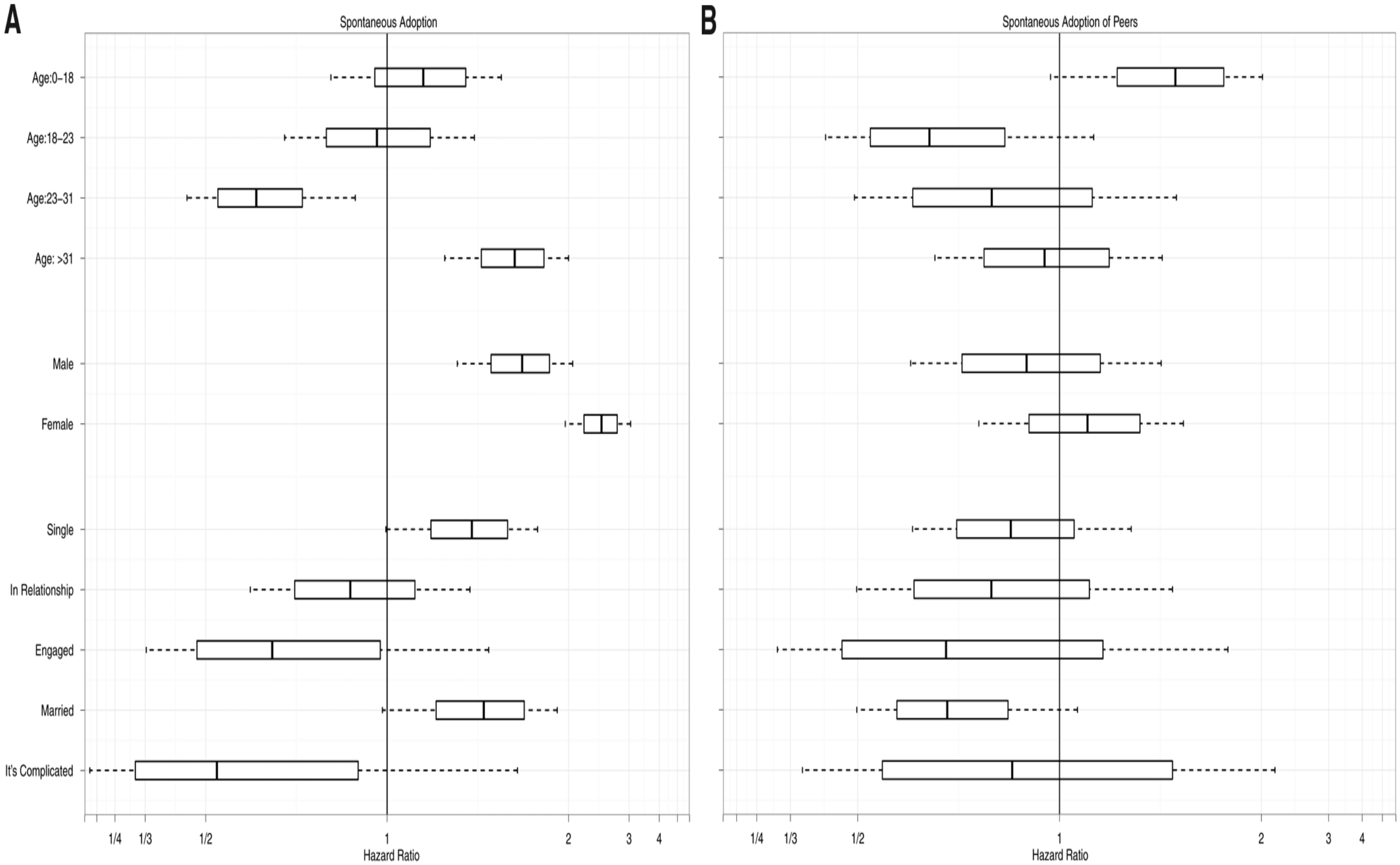

Fig. 3. Left panel: Hazard ratios for individuals to adopt spontaneously as a function of their attributes along with standard errors (boxes) and 95% confidence intervals (whiskers). Right panel: Hazard ratios for individuals to have peers adopt spontaneously as function of their attributes. The figure displays hazard ratios (HR) representing the percent increase (HR > 1) or decrease (HR < 1) in adoption hazards associated with each attribute.

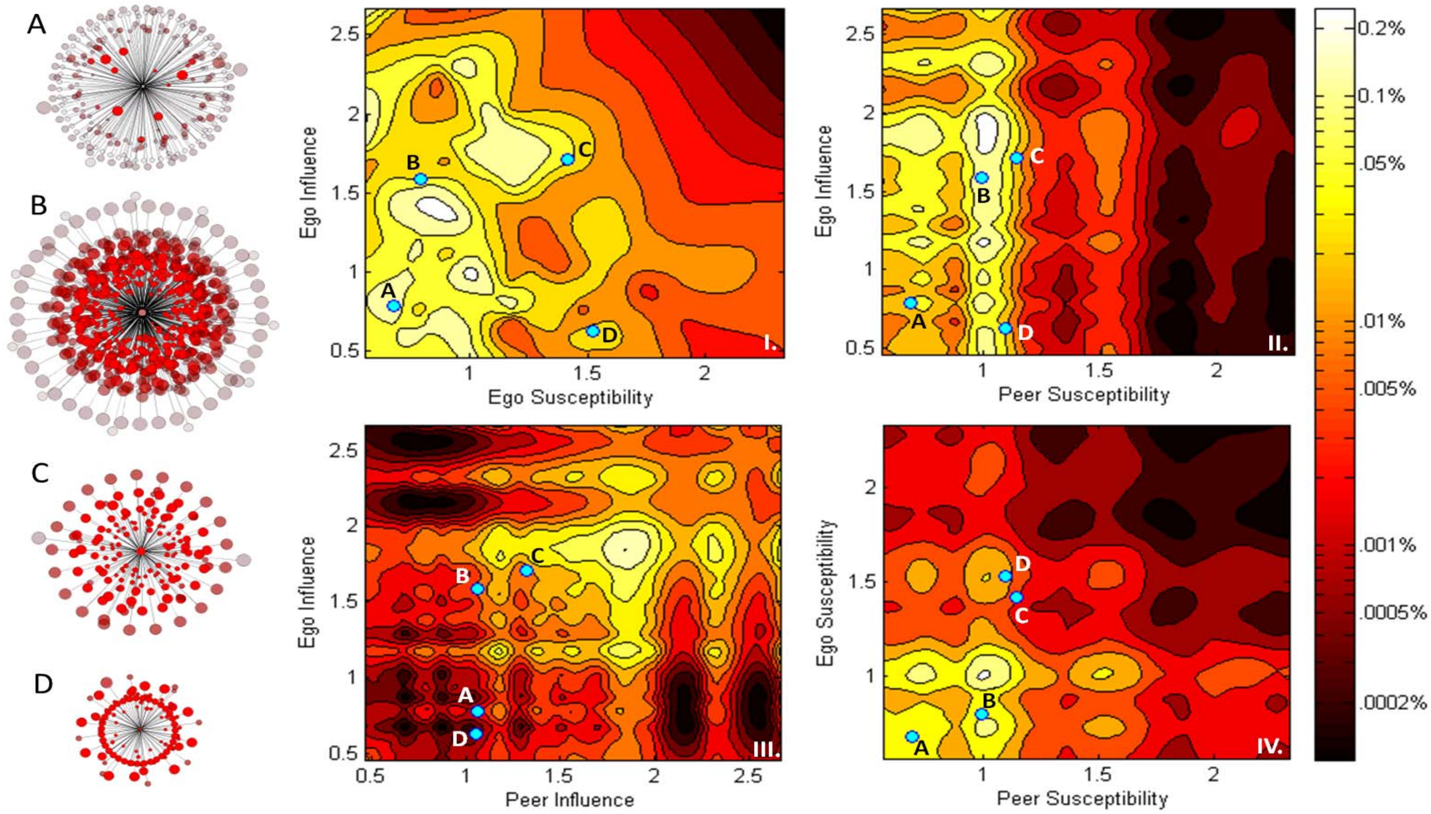

Fig. 4. Heat Maps. Scores for 12M Facebook users (collected from users who installed one of several other Facebook applications developed by the company with 85M relationships are calculated by means of hazard rate estimates relative to the baseline hazard in influence and susceptibility models described in the text. Panel I displays the percentage of people (ego) with predicted influence (y-axis) and predicted susceptibility (x-axis). Panels II-IV display the percentage of ego-peer relationships with (II) ego influence (y-axis) and peer susceptibility (x-axis); (III) ego influence (y-axis) and peer influence (x-axis); and (IV) ego susceptibility (y-axis) and peer susceptibility (x-axis). Network Figures (left): The heat maps do not provide information on network structure, which can be important for informing targeting decisions. The network figures to the left of the heat maps show the assortativity of influence and susceptibility in ego networks drawn from the regions of the heat maps at points A, B, C and D. Nodes in the networks are sized in proportion to their predicted influence (larger nodes are more influential) and shaded and laid out relative to their predicted susceptibility (redder nodes and nodes closer to ego are more susceptible while greyer nodes and nodes further from ego are less susceptible).