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Journal
Journal of Health and Social Behavior, 57(1)

ISSN
0022-1465

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Publication Date
2016-03-01

DOI
10.1177/0022146515627848

Peer reviewed

HHS Public Access

Author manuscript

J Health Soc Behav. Author manuscript; available in PMC 2019 August 03.

Published in final edited form as:

J Health Soc Behav. 2016 March ; 57(1): 22–38. doi:10.1177/0022146515627848.

How Initial Prevalence Moderates Network-Based Smoking Change: Estimating Contextual Effects with Stochastic Actor Based Models*

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Abstract

We use an empirically-grounded simulation model to examine how initial smoking prevalence moderates the effectiveness of potential interventions designed to change adolescent smoking behavior. Our model investigates the differences that result when manipulating peer influence and smoker popularity as intervention levers. We demonstrate how a simulation-based approach allows us to estimate outcomes that arise (1) when intervention effects could plausibly alter peer influence and/or smoker popularity effects, (2) across a sample of schools that match the range of initial conditions of smoking prevalence in US schools. We show how these different initial conditions combined with the exact same intervention effects can produce substantially different outcomes - e.g., effects that produce smoking declines in some settings can actually increase smoking in others. We explore the form and magnitude of these differences. Our model also provides a template to evaluate the potential effects of alternative intervention scenarios.

Even with recent declines, a fifth of high schoolers identified as smokers in 2009, while nearly half reported ever having smoked (CDC 2010). The vast majority (80%) of adult smokers first smoked as adolescents (Kessler et al. 1997), making this a critical point in the life course. During this period, adolescents begin to spend less time with family and more time with friends (Larson and Richards 1991). Consequently, friendship networks are a key contributor to developing and habituating health behaviors (Haas, Schaefer and Kornienko 2010), including smoking (Kobus 2003). Moreover, the schools adolescents attend are an important context that provides behavioral norms and peer influences that shape health trajectories (Gest et al. 2011).

Interventions have increasingly leveraged friendship-based mechanisms to improve health outcomes (Valente 2012). While peer-based approaches have proven beneficial, their implementation is often limited to a few locales at a time. Recent work on structural interventions has demonstrated that contextual conditions can moderate interventions' effectiveness, leading the same intervention to have substantially different effects across

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sites (Sambrano et al. 2005). In particular, the initial prevalence of a targeted behavior or outcome can substantially alter an intervention's effectiveness (Tobler et al. 2000). This raises the specter that interventions developed in a limited set of contexts may not generate the same beneficial outcomes when carried into new settings. Effects of network interventions in particular may be subject to moderation by initial prevalence. For example, when direct peer influence is strong, adolescents tend to adopt their friends' behavior. But, when peer influence is weaker, other sources of influence, such as global norms, can wield more power. In such a situation the prevalence of the behavior in the broader environment is likely to carry greater consequence. This highlights the need to understand how contextual moderators can help anticipate when and how the same interventions can produce differing results, and when different intervention approaches may be necessary to evoke the same desired outcomes.

We extend previous work that examines how variation in the strength of peer influence and smoker popularity effects cumulate to affect smoking outcomes. Research demonstrates that adolescent friendship dynamics and smoking behavior are closely intertwined (Green et al. 2013; Mercken et al. 2010; Pearson, Steglich and Snijders 2006; Schaefer, Haas and Bishop 2012). Friendships provide pathways to smoking via "peer influence" as adolescents adopt behaviors exhibited by their friends (Haas and Schaefer 2014). Conversely, smoking is a salient aspect of "friend selection," with adolescents often selecting friends whose smoking behavior matches their own (Schaefer, Haas and Bishop 2012). Here, we explore how smoking interventions that target these peer-based processes might differ in effectiveness depending on the initial prevalence of smoking.

To examine this question, we develop a series of empirically-grounded simulations from a statistical model that estimates the coevolution of network change and smoking behavior (Steglich, Snijders and Pearson 2010). These models allow us to isolate the key factors of a network-based intervention - i.e., peer influence on smoking behavior, effects of smoking by popular teens on others in the school, and friendship selection processes (Schaefer, Haas and Bishop 2012) - and apply them to a wider range of schools than would be feasible in field experiments. We parameterize these models with data from the first two waves of the National Longitudinal Study of Adolescent Health (Bearman, Jones and Udry 1997).

Background

Health behaviors are influenced by the contexts and social networks that surround individuals (Haas, Schaefer and Kornienko 2010; Valente 2010). In particular, recent work has sought to disentangle the complex ways that social networks and smoking behaviors are intertwined (Christakis and Fowler 2008; Lakon, Hipp and Timberlake 2010). Numerous processes contribute to the observed associations between one's own smoking behavior and that of their peers (Hoffman et al. 2006); key amongst those are peer influence, homophilous selection, popularity, and social norms (Green et al. 2013; Steglich, Snijders and Pearson 2010). We begin by briefly describing each of these processes in turn.

Smoking Peer Effects

Peer influence is the process by which individuals' change their behaviors over time to more closely resemble the behaviors of their friends (Brechwald and Prinstein 2011; Mercken et al. 2010). Evidence suggests that peer influence on smoking operates both for uptake (Ennett et al. 2006) and cessation (Christakis and Fowler 2008), with recent work suggesting the strength of these processes is asymmetric (Haas and Schaefer 2014). Moreover, adolescents may be more susceptible to such peer influences because of the rapid changes in smoking behavior at that point in the life-course (Mayhew, Flay and Mott 2000). These dynamics are especially important to understand as the teen years are key for setting later trajectories of potential tobacco use (CDC 2008).

While similarities among peers' smoking behaviors have frequently been attributed to interpersonal influence, recent work has highlighted the importance of homophilous selection in accounting for these patterns (Green et al. 2013; Hall and Valente 2007; Schaefer, Haas and Bishop 2012). Homophilous selection occurs when individuals select others who are similar to themselves on various characteristics (McPherson, Smith-Lovin and Cook 2001) - including smoking (Mercken et al. 2010). While one could approach this as a source of endogeneity requiring controls to uncover true influence effects (Manski 1993), homophilous selection has become a process of primary interest among social network scholars (Shalizi and Thomas 2011; Steglich, Snijders and Pearson 2010).

Beyond processes of influence and selection that create similarities between friends, smoking is also often associated with popularity (Moody et al. 2011; Valente, Unger and Johnson 2005). In some schools smokers may be unpopular (i.e., less likely to be chosen as friends than non-smokers) whereas in other school contexts, smokers are more popular than non-smokers as friends. Smoking-based friendship selection can facilitate smoking diffusion, for instance if non-smokers follow the lead of more popular friends who smoke (Brechwald and Prinstein 2011; Lakon, Hipp and Timberlake 2010; Valente and Pumpuang 2007; Valente et al. 2007).

Peer-Based Interventions

Noting the importance of friendship processes in adolescent smoking dynamics, intervention efforts have increasingly targeted friendship mechanisms (Valente 2012). For example, school-based interventions have focused on reducing adolescent susceptibility to peer influence (Campbell et al. 2008; Lynam et al. 1999; Peterson et al. 2000), with varying levels of success (Hwang, Yeagley and Petosa 2004; Lantz et al. 2000; Wiehe et al. 2005). Similarly, interventions have identified popular adolescents, or "opinion leaders," within school contexts for targeted interventions (Valente and Davis 1999; Valente and Pumpuang 2007). "Information based" interventions (Lynam et al. 1999) can also alter peer influence, selection, and popularity effects, if the popularity derived from smoking decreases as peers learn about the harms of smoking.

Contextualizing Peer Effects

Contextual prevalence of a behavior can shape individual behaviors in a number of ways. It can alter how often individuals encounter a behavior in their environment, alter the social

norms about the behavior (Mollborn, Domingue and Boardman 2014), change how peer-effects function (Alexander et al. 2001; Zhang et al. 2014), and can even alter genetic influences on behavior (Boardman et al. 2008). Directly, when prevalence is greater, the chances increase that the members of one's network have engaged in the behavior, increasing one's exposure to it. In low prevalence settings, this might mean the difference between having one smoker as a friend versus none, compared to having some versus mostly smoking friends in higher prevalence settings. These exposure differences can lead to differential likelihood of peer-influence based adoption (or cessation), even if all teens were equally (un-)susceptible to peer influence. Indirectly, social norms about smoking within school contexts have been shown to vary directly with the prevalence of observable smoking (Eisenberg and Forster 2003; Gest et al. 2011). As such, exploring the differential effects of initial prevalence on potential intervention roll-outs is informative. Naively, one might expect that enhancing the strength of influence and popularity processes would simply exacerbate the effects of initial prevalence. We have learned from a number of systems science approaches, however, that such naive extrapolations are often overly simplistic (Levy et al. 2010). This realization has lead to an increased focus on identifying critical thresholds (i.e., at what point do models transition from generating increases to decreases in targeted behaviors) and understanding the shape of those trajectories (e.g., not just direction, but shape and magnitude of any such differences).

A primary way that simple prevalence-based extrapolation of effects may not hold is that empirically, smoking is associated with a number of individual, contextual and relational factors - e.g., age, race, gender, other substance use (Lakon, Hipp and Timberlake 2010; Pearson, Steglich and Snijders 2006), and aspects of network position (Haas and Schaefer 2014; Valente, Unger and Johnson 2005). For instance, smokers are more likely to cluster together in schools with higher smoking prevalence (Alexander et al. 2001). We must consider these associations to be certain that any generated outcome differences derive from prevalence differences themselves, not by other factors confounded with prevalence. Below, we ensure this via a combination of two steps. First, these associations inform the strategy for producing initial smoking prevalence conditions; which we elaborate in modeling step I below. Second, we evaluate the robustness of our primary findings to these initial condition assumptions by conducting a follow-up analysis using two alternative strategies.

In addition to direct exacerbation and confounding, initial prevalence can shape smoking outcomes in a number of other ways that moderate the peer-based effects described above. For instance, diffusion and network clustering constitute two processes of theoretical importance, with substantial complexity in their relationship to outcomes (Koopman 2004). Most diffusion processes follow an S-shaped curve marked by initially slow growth, followed by phase of relatively rapid diffusion, then a plateau at or near some threshold of maximal diffusion (Rogers 1995). While influence and popularity processes shape the steepness of the increase observed in the growth phase of any diffusion process (Valente 1995), differences in initial prevalence influence how early that take-off phase is encountered, and how much of the population is ultimately reached by any diffusing characteristic (Rossman 2012). Moreover, empirical networks exhibit high levels of clustering (Snijders 2011), which can substantially alter how widely any observed behavior diffuses (Salathe and Jones 2010). As such it is key to identify at what point(s) similar

effects may generate divergent outcomes (e.g., increasing versus decreasing) in diffusing behaviors of interest--i.e., identify the “tipping points” that arise from varying initial prevalence levels (Valente 1996). The key theoretical aim therefore is to isolate the effects of particular factors like initial prevalence, which are not empirically separable in “real world” contexts. Finding ways to do so will allow estimation of the independent effects of initial prevalence differences on any generated outcome differences.

Evaluating the potential effects of an intervention across a full range of contexts poses a number of challenges. Practically, interventions are often limited in the number of contexts in which they are implemented, making rigorous statistical comparisons of the multitude of important contextual factors impossible (Valente 2012). This makes it difficult for any single observed intervention effect, or linear models based upon observational data, to account for stochastic perturbations in outcomes that arise from these clustering patterns or other similarly complex dynamic processes. One solution is to make certain that intervention contexts contain heterogeneity on key factors that are predetermined. However, our theoretical knowledge of which contextual factors are most important is notably incomplete (Valente 2012), making it difficult to ensure that included sites represent the relevant dimensions of heterogeneity. Moreover even if theoretically identifiable, social network data —especially that needed for estimating models to assess peer-based interventions—are costly to gather (Marsden 2011). It is therefore rare that such efforts have been evaluated in more than a few locations at a time.

Simulating Interventions Across Multiple Contextual Conditions

Computer simulations are a promising strategy for overcoming some of these limitations (Bruch and Atwell 2014). Simulations allow researchers to investigate and understand the impact of manipulating key attributes of complex systems while holding other features constant (Homer and Hirsch 2006; Levy et al. 2010; Maglio, Sepulveda and Mabry 2014). Simulations provide the unique capacity to individually isolate the multitude of mechanisms that typically comprise an intervention, by selectively manipulating single conditions at a time, which would not be feasible in real-world intervention studies (Levy et al. 2010; Mabry et al. 2010; Maglio, Sepulveda and Mabry 2014). For this paper, we employ agent based models (ABM), which allow us to generate initial behavior prevalence conditions, then model how friendship and behavior dynamics unfold over time. This is critical for considering how interventions—developed and implemented in particular settings—might unfold differently when extended to settings with divergent initial conditions. The 85 Add Health schools that we use in the analyses provide such variation, exhibiting differences in initial smoking prevalence ranging from 15% to 55% (mean=35%).

While we can tailor simulations to specify precise intervention effects, simulations are occasionally criticized for their weak empirical basis. This drawback can be addressed by informing ABMs with empirically-derived conditions and parameter estimates (Ip et al. 2013). That is, simulation “rules” can be tailored to reflect effect magnitudes measured in the natural world (Bruch and Atwell 2014). In an example that guides our approach, Snijders and Steglich (2013) show how the stochastic actor-based model (SABM) can be used to derive estimates of friendship-behavior dynamics from observed data. SABMs have been

used to examine the complex processes linking smoking and friendship in empirical contexts, particularly separating selection from influence processes (de la Haye et al. 2013; Green et al. 2013; Snijders, van de Bunt and Steglich 2010; Steglich, Snijders and Pearson 2010). Because the algorithm used to fit an SABM is a form of agent-based model, simulations that manipulate key rules or conditions are a natural extension of fitting an SABM (Mercken et al. 2010; Schaefer, adams and Haas 2013; Snijders and Steglich 2013). This process require taking an existing set of model parameters and exploring the implications of an alternative set of parameter values and/or initial conditions. This is the approach we employ for seeding our agent based models with empirically-grounded estimates of secular changes in smoking behavior, structural and individual initial conditions, and effect sizes for smoking-based selection and peer influence.

Data

We use data from the National Longitudinal Study of Adolescent Health (Bearman, Jones and Udry 1997). Add Health data contains information on complete networks for more than 100 schools at 1 wave, and 16 schools at 3 waves. Each wave of data also contains information on adolescent smoking and other individual attributes. Following Schaefer et al. (2012; 2013), smoking behavior is specified by 3 levels of reported smoking frequency in the past 30 days: 0 = never, 1 = 1–11 days, 2 = 12 or more days. We use one school with longitudinal network data, with two waves one year apart, to fit an SABM. For extending the model developed by Schaefer et al. (2013, SAH hereafter), we identify Add Health schools with complete network data, acceptable response rates (>75%) and sufficient presence of smoking (>15% ever smoked). This results in a sample of 85 schools with cross-sectional network data, from which we generate the empirical distribution of smoking and its association with friendship networks. Table 1 presents descriptive statistics for this sample of schools on the key smoking and network variables included in our models.

Modeling Approach

Our model is a direct extension of SAH, which used an SABM reported by Schaefer et al. (2012) to simulate how smoking outcomes would differ if actors in one school followed slightly different rules for choosing friends and being influenced by friends' smoking behavior. These rule manipulations are intended to reflect the peer-based intervention scenarios described above that generate alterations in the strength of peer influence and/or smoker popularity effects. SAH demonstrate a strategy for modeling how such social-network based interventions can alter trajectories of population-level smoking prevalence, initiations and cessations. We apply this same sort of model to a wider range of initial smoking prevalence conditions to examine how those differences moderate the effects of peer influence and smoker popularity effects on smoking outcomes. Our model proceeds in 4 steps, which correspond to theoretical concerns about such models elaborated by Bruch and Atwell (2014):

I. Generate baseline conditions to represent a range of prevalence distributions;

II. Fit the SABM model to obtain parameter estimates for simulated intervention rules;

III. Manipulate peer influence and smoker popularity across baseline conditions;

IV. Record outcome(s) of interest for each simulation of network and smoking co-evolution.

I - Generate Baseline Conditions

This step is the key analytic focus of our paper, and therefore is the only step in the modeling process that differs significantly from SAH. We need to establish a means for generating smoking prevalence and frequency distributions, which represent what Bruch and Atwell (2014) characterize as "input uncertainty" aspects of the model. As mentioned above, we must be mindful of the association between smoking and other factors, particularly those related to friend selection and influence. Ideally, these smoking prevalence inputs would vary while leaving associations between smoking and other factors constant. We examined how these school-level measures varied according to the prevalence of smoking in our sample of Add Health schools. We found that network autocorrelation on smoking (i.e., smoking homophily) increases with greater prevalence (Figure S2) and the correlation between smoking and popularity ranges from negative in middle schools to positive in high schools (Figure S3). In other words, smoking prevalence itself is highly correlated with many other factors incorporated in the model we develop below.

Stemming from this observation, we rely on a set of empirically observed networks as baseline conditions to fix these associations as they are distributed within the cross-sectional sample of 85 schools from Add Health described above. This approach has the advantage of also fixing associations between smoking and any important factors that are *unobserved*. These schools naturally vary in smoking prevalence, and represent a range of associations between smoking and network structure (see Table 1). Below, we also report robustness checks for our results that rely on two alternative strategies for generating initial conditions.

II - SABM Estimation

Step II allows us to ensure our model appropriately replicates the SABM from Schaefer et al. (2012) to specify what Bruch and Atwell (2014) label agent behavior. Briefly, the goal is to model endogenous changes in network characteristics and smoking behavior. The SABM contains a "behavior" function to model change in smoking due to individual and network factors (i.e., friends' smoking) and a network function to model change in friendships based on individual factors, dyadic attributes (e.g., homophily), and network processes (e.g., reciprocity). This estimated SABM empirically derives the strength of parameters associated with observed changes in smoking and friendships for those individuals who are observed over time. More details of the general SABM approach are available from Snijders and colleagues (Mercken et al. 2010, Snijders, van de Bunt and Steglich 2010, Steglich, Snijders and Pearson 2006), and for the replicated model from Schaefer et al. (Schaefer, Haas and Bishop 2012). We estimate all SAB and simulation models in RSiena 1.1–232 (Ripley et al. 2014).

One finding of the replicated SABM model is that "adolescents influenced each other's smoking frequency and selected friends with similar levels of smoking. Thus, both selection and peer influence contributed to similarity on smoking among friends" (Schaefer, adams

and Haas 2013, p. e16). Of primary interest is the peer influence effect, which is estimated as 2.89. All else being equal, this indicates how a one-unit difference in smoking similarity between ego and his or her friends corresponds to change in the log odds of adjacent levels of smoking behavior. For example, if increasing one's smoking behavior increases the similarity between oneself and one's friends by 1, then the odds of increasing smoking are 4.24 times greater (exp[beta/smoking range] = exp[2.89/2]; see Ripley et al., 2014) than maintaining one's current smoking level. Turning to smoker popularity, we replicated the finding that students with higher levels of smoking were more likely to be selected as friends than students with lower smoking levels (beta=0.14). The smoker popularity parameter can also be interpreted as the effect of a one-unit difference, this time in alter's smoking level on the log-odds of a tie. The observed parameter of .14 indicates that the odds of befriending a moderate smoker are 1.15 (exp[.14]) times greater than befriending a non-smoker (or a regular vs. moderate smoker), all else being equal. Full details of this model are reported in the Supplementary Information (Table S1) and described in Schaefer et al. (2012).

Our simulation uses these effects and corresponding parameter estimates from the estimated model as the rules governing changes in friend selection and smoking behavior. With two exceptions (detailed below in step III) the actors in our simulations base their friend selection and smoking level decisions on the same factors as adolescents in the observed school. This allows us to estimate how differences in baseline smoking prevalence moderate the effects produced by *the same* model-based mechanisms.

III - Parameter Manipulation and Simulation

This step isolates the manipulations of peer influence and popularity effects that approximate the intervention scenarios described above. Doing so allows us to properly control the aspects of model uncertainty included in the ABM (Bruch and Atwell 2014). We use the parameters from the model fit in section II. We then manipulate the targeted "intervention" parameters (altered peer influence and smoker popularity effects) in the same manner described by SAH, ensuring that values: (a) are centered on observed values from Schaefer et al. (2012) - reported above, (b) include 0, to account for the possibility of null effects, (c) allow for negative values on the smoker popularity effects, to account for the possibility of smokers being *less* popular than nonsmokers, and (d) extend an equal distance in the positive direction (i.e., estimating substantially stronger than observed effects). This results in modeled peer influence ranging from 0 to 6 in increments of 1, with higher values indicating a stronger tendency to adopt a smoking level closer to one's friends' average. Smoker popularity effects range from −0.4 to 0.8 in increments of 0.2, where values indicate the association between peers' smoking behavior and their likelihood of being selected as a friend. During the simulation, actors are given multiple opportunities to change their network ties and smoking based on the model and parameter estimates from the observed data, and manipulations for peer influence and smoker popularity parameters, described above. Applying this model to a single school, Schaefer et al. (2013: 29s) found:

> "…changes in [peer influence] and smoker popularity can affect smoking behavior, but their effects are contingent on one another. Changing smoking-based popularity only affected smoking prevalence when [peer influence] was present. Likewise, the impact of changing the [peer influence] effect was dependent on the strength of

> smoker popularity. Higher levels of [peer influence] increased smoking when smokers were popular, but decreased smoking when smokers were unpopular."

With one exception, all other parameters are held constant at the values from the observed school estimated in step II. The exception accounts for the simulated change in smoking prevalence in each school if all other effects were absent (i.e., the linear and quadratic effects in the smoking function). These are fixed to set each school's default change in smoking prevalence to zero, rather than reproducing the level of change from that one school.

IV - Outcomes

We conducted 100 simulation runs over each combination of peer influence and smoker popularity (7 levels each, producing 49 combinations). For each simulation run, our outcome measure is *change in smoking prevalence*, which is computed as the difference in the proportion of adolescents who smoke at time 2 compared to time 1 - which corresponds to the year over which the observed network dynamics were estimated in step II. This recorded range of outcomes allows us to identify how robust our model's results are to stochastic variability within the model (Bruch and Atwell 2014).

Results

Figure 1 presents the simulated change in school-level smoking prevalence obtained by extending the combination of parameter estimates from the fitted SABM (peer influence =3, smoker popularity =0.2), to the initial conditions of all sample schools - each point represents the resulting mean change in smoking prevalence for a single school (with whiskers denoting the 95% confidence intervals). The schools are arranged along the x-axis according to their initial smoking prevalence. This Figure in essence shows how smoking prevalence would change in the full sample of schools if they each were subject to the observed influence and popularity effects from the school in Schaefer et al. (2012). Two details are worth highlighting. First, the school from which the model was derived (filled circle) generated no change in smoking prevalence. Remember that the aim here is to isolate the differences generated solely from the manipulated (peer influence and smoker popularity) parameters, keeping other observed factors constant (i.e., applying the *same* intervention effects across settings). In the observed school, without holding those other factors constant, a 5% smoking increase was observed. The simulated model generates no change in smoker prevalence when those other factors are held constant, and the targeted intervention scenarios (peer influence and smoker-popularity) are held at their observed values. In other words, this simulated data point allows us to confirm that we can successfully isolate the effects of peer influence and smoker popularity in the desired ways, which can in turn be applied to the other initial conditions. Second, the results for the other schools foreshadow the general pattern in the full results - the *same* process unfolding under different initial conditions leads to divergent smoking outcomes. In this case, schools with lower initial prevalence show *declines* in smoking prevalence, whereas schools with the highest initial prevalence show little simulated change.

Figure 2 provides results for a selection of 16 manipulated parameter effect combinations (results for all 49 parameter combinations are presented in the Supplementary Information).

This figure can be interpreted two primary ways. First, focusing *within* any one panel addresses our primary question: How does the *same* combination of simulated peer influence and smoker popularity parameters produce differing results when applied to varying initial smoking conditions? Second, comparing results down the columns or across the rows demonstrates how smoking outcomes vary depending on *different* manipulations of intervention-based parameters (peer influence in the letter-labeled rows, and smoker popularity in the numeric-labeled columns). These cross-panel comparisons are meaningful both for *individual schools* (e.g., the relative differences for the filled circle in each plot replicate SAH model results) and for differences between the *distributions*.

Focusing on the first interpretation, Panel Aii represents when both peer influence and smoker popularity effects are absent. Under these conditions, simulated prevalence remains virtually unchanged - both for the school on which the model is based (filled circle) and across most of the other observed school conditions (73 of 85 confidence intervals include 0 and mean values are each relatively close to 0). Comparing panel Aii to the others in Row A reveals that when peer influence is absent—regardless of the strength of smoker popularity effects—smoking prevalence remains unchanged for nearly all schools. Turning to the rest of column ii (null smoker-based popularity selection effect), smoking prevalence declines across most initial conditions as peer influence is introduced. This prevalence decline is stronger at moderate than extreme levels of initial smoking as indicated by the “U-shape” curves. Moreover, the strength of this decline intensifies with increases in the peer influence parameter (the deepening of the U-shape moving down the column).

Moving away from outcomes when one (or both) manipulated effect(s) is null, we find that differing initial smoking conditions produce substantially divergent smoking outcomes for several combinations of peer influence and smoker popularity effects. Models with modest *negative* smoker popularity effects (Column i) retain the general pattern from the model with null smoker popularity effects – smoking declines, marked by a consistently below zero U-shape pattern, which amplifies as peer influence increases. For each of the combinations where peer influence and smoker popularity effects are both *positive* (Rows B-D, Columns iii-iv), differences in initial smoking prevalence lead to smoking outcomes marked by an inflection point: low smoking prevalence schools (less than ~30% initial prevalence) exhibit declines; higher smoking prevalence schools (above ~40%) experience increases; and schools with more moderate initial smoking prevalence exhibit no significant changes. Across these four panels, stronger effects change the amplitude, but not general pattern of this S-curve.

The second way to interpret Figure 2 is to compare single schools across panels, which reveals three distinct patterns: (1) In low prevalence schools (less than ~20%), smoking almost always declines, with stronger peer influence amplifying those declines (comparing down the columns). (2) Smoker popularity alters that effect comparatively little (comparing across the rows). At moderate levels of initial smoking (between ~20–40%) effects are mixed between declines and no changes. High peer influence and low/null smoker popularity parameter combinations (i.e., Rows B-D, Columns i-ii) generate some of the largest declines. However, for these schools, increasing smoker popularity effects (across rows) shifts observed outcomes, from prevalence declines, to results mostly

indistinguishable from zero. For schools with low or moderate levels of initial smoking, *no* combination of modeled effects produce significant smoking increases. (3) Schools with the highest initial smoking prevalence (greater than ~40%) generate a complex combination of outcomes. When manipulations made smokers unpopular (Column i), in the presence of peer influence (Rows B-D), smoking decreased, and more strongly as peer influence increased. By contrast, when smokers were relatively more popular we observe the only instances where smoking prevalence increases. Looking down Column iv, as peer influence strengthens, increases in smoking prevalence become more pronounced. This pattern is also evident in column iii, where smoker popularity is weaker, but the confidence intervals indicate that changes are mostly indistinguishable from zero.

Finally, a related way to examine these results is to ask how much initial smoking is necessary for prevalence to increase or decrease (i.e., where are the inflection points), and how does this vary based on the strength of peer influence and smoker popularity. At the lowest levels of peer influence or popularity effects, no threshold pattern emerges, with smoking consistently stagnating in the former or decreasing in the latter. At modest peer influence increases, smoking change either stagnates (in high popularity settings) or declines (in low popularity conditions). At the highest levels of combined smoker popularity and peer influence effects (e.g., cell Div), schools can experience prevalence increases even though initial smoking prevalence is quite low (i.e., less than one-third initial smokers). With strong peer effects, the initial prevalence exhibits a threshold that differentiates schools where the model generates smoking declines (initial low prevalence) from those generating increases (high initial prevalence). Looking at the empirical distribution of schools, these conditions are most likely to be met in schools with positive correlations between smoking and indegree (which could result from smoking-based popularity) and higher smoking autocorrelation (which would occur if peer influence were strong). Approximately one-quarter of the Add Health schools display initial conditions that meet these criteria (see figures S2–S4). Moreover, of the Add Health schools that exhibit strong initial popularity-smoking associations and autocorrelation with smoking, many have moderate initial smoking prevalence (see Figure S4). In other words, those schools with the strongest initial peer-smoking associations are likely to straddle the critical threshold of initial prevalence, leading small (intervention-based) changes being capable of tipping the scales in either direction.

Robustness Check

Our approach to manipulating prevalence was to use empirically-observed school network and smoking distributions to specify initial conditions. This strategy has the advantage of retaining the associations between targeted prevalence levels and other contextual characteristics, either known or unknown, that may affect the processes we investigate. To examine how robust the pattern of results described above are to this approach, we explored two alternative strategies for setting initial conditions that rely on slightly different sets of assumptions.

The (a) *random assignment* condition uses time 1 data from the school used in SAH, and randomly assigns individual smoking behavior to match the target initial smoking prevalence

conditions. We base these conditions on a combination of smoking-related factors observed in the sample of 85 Add Health schools. First, initial prevalence conditions range from 15% to 75% initial smokers in increments of 10%, which extends the observed distribution to incorporate some potential range of measurement error. Second, for a given prevalence level, we specify the number of non-smokers (identified as smoking level 0), and distribute smokers across levels 1 and 2 to create a *target distribution*. We took an empirical approach to creating the distribution rule. For each Add Health school in our sample we calculated the proportion of students at each of the 3 smoking levels. From the observed proportions, we regressed the proportion of students at smoking level 1 on the proportion of nonsmokers, obtaining a reasonable fit (r^2 = .61). We then fed each target level of non-smokers into this model to predict the number of smokers at level 1. To illustrate, the estimated model has the form: $smokers_1$ = .48 - .39($smokers_0$). For prevalence level .75, in which the proportion of smokers at level 0 is .25, this evaluates as .48 – .39(.25) = .38, indicating that the proportion of smokers at level 1 should be .38. The remaining 37% of actors are at smoking level 2 (i.e., 1 - .25 - .38 = .37). Once we have these target distributions, random assignment of smoking status to match the distributions of initial smoking prevalence is a straightforward approach. However, random assignment ignores any empirically-observed associations between smoking and network structure, as well as correlations between smoking and other individual attributes.

The (b) *model-based* approach seeks to identify key structural patterns, then reproduce them when manipulating baseline conditions. We identify two classes of associations: 1) correlations between smoking and other individual attributes, and 2) associations between smoking and network structure. To maintain associations with other individual attributes, we fit a multinomial model that predicts smoking based on sex, age, alcohol use, and GPA. For each actor, we use this model to calculate the predicted likelihood of being at each smoking level. For a given target smoking distribution, actors with the greatest probability of being non-smokers are assigned smoking level 0; actors with the greatest probability of being at smoking level 2 are thus assigned, and the remaining actors are assigned smoking level 1. This assignment contains a stochastic element; thus, an actor with a low probability of being a smoker may still be assigned to be a smoker. After assigning the smoking distribution, we check that the assignment had its intended effect of maintaining the correlation between smoking and these individual attributes (if correlations fall outside the observed 95% CI, the assignment is rejected and a new distribution of smoking values is generated).

Then, to maintain the observed association between smoking and network structure, we fit an exponential random graph model (ERGM, Robins et al. 2007) to the observed school. The ERGM provides estimates of purely structural properties (e.g., triadic closure, reciprocity) and associations between smoking and the network (smoking autocorrelation and smoker popularity). These estimates provide parameters from which we create a distribution of networks with the same number of actors, ties, and key properties as the observed network. From this distribution, we retain those networks where density, smoking autocorrelation, and the correlation of smoking and indegree fall within the 95% CI of the observed distributions of associations.

Figure 3 compares these two additional strategies for setting initial conditions to the results presented above; for simplicity we present only the fitted loess curves for mean values; the full set of results, with 95% confidence intervals and all 49 parameter value combinations, are available in Appendix Tables S5–S7. By and large, the results are robust to the strategy employed for generating the initial conditions. The lone exception is that the model-based conditions produce somewhat different effects at the highest levels of peer-influence and smoker popularity effects (lower, right cells of Figure 3). Namely, the shape and magnitudes of the curves across conditions remain relatively similar, however the model-based condition exhibits increases in smoking prevalence at lower levels of initial prevalence than the other two conditions. This suggests that in schools with the same structural features as those from observed schools, but lower initial smoking prevalence (e.g., derived from the population declines in smoking prevalence observed since Add Health data were collected), we may observe smoking increases similar to those in the higher prevalence schools observed in these data. Alternatively, this deviation for the model-based condition could also indicate that the model is missing one or more important aspects of structure—aspects that are captured by the empirically-based conditions.

Discussion

Frequently, fielded interventions intentionally target sites that differ in strategic ways (e.g., to see if they can produce similar health improvements in “high” and “low” risk settings). Short of massive-scale intervention rollouts, in which contextual factors are carefully matched, we are limited in our efforts to disentangle differences in targeted intervention effects from differences generated by contextual variation. The combination of empirical and simulation models demonstrated here enhances our capacity to isolate the precise mechanisms driving behavior changes. In essence the logic of our approach is that if only contextual factors vary across settings—while intervention mechanisms operate *identically*—any differences in outcomes are attributable to those differences in contextual factors. This allows more principled consideration of the potential range of effects that can be anticipated from intervention efforts, and assists in identifying the (combinations of) factors that are likely to provide the greatest probability for desirable outcomes. Our results show that reaching similar health outcome targets in different initial contextual conditions may require very different intervention designs.

Schaefer, adams and Haas (2013) demonstrated the utility of SABMs for anticipating outcomes of peer-network interventions targeted at observed friendship network and smoking dynamics for a single school. Here, we explore how those same intervention effects can generate differential consequences dependent upon initial conditions. Our key finding is that even if an intervention could generate the *exact same* mechanistic change across schools (e.g., altering peer influence in *exactly* the same way), the effects on smoking outcomes could substantially differ, depending on initial prevalence. The differences we identify are not simple extrapolations from one school to another: effects that reduce smoking in some school conditions can lead to increases in others. Moreover, it was not simply the case that initially predominate behaviors ultimately take over. Rather, prevalence seems to moderate the effects of peer influence and smoker-popularity changes. For example, in schools with relatively low initial smoking prevalence (< ~25%), an intervention designed to reduce peer-

influence susceptibility would be detrimental to smoking reductions. This is because in low prevalence schools, peer influence can serve a protective function by preventing (or reducing) smoking.

The consequences of manipulating these intervention levers are quite different in contexts with high initial smoking prevalence. Contrasting above, interventions designed to reduce adolescents' susceptibility to peer influence would have beneficial results in schools that start with higher smoking prevalence. If high initial prevalence were coupled with high smoker popularity, then reducing peer influence would inhibit the spread of smoking (a positive population health outcome). In such a situation, smokers are relatively more influential because the network positions they occupy provides greater connectivity to others. Thus, weakening the magnitude of peer influence, thereby making actors more autonomous, restricts smoking diffusion. Contrastingly, in a similarly high prevalence context, but lacking strong smoker popularity effects, reducing peer influence would generate more modest smoking declines because smokers do not occupy the same influential network positions.

If interventions could alter peer influence and popularity effects *simultaneously*, the outcomes are even more complexly related to the initial conditions in the school. For example, an intervention designed primarily around demonstrating the harmful effects of smoking might be expected to reduce both smoker-popularity (e.g., by stigmatizing the behavior) and peer influence effects (by increasing the weight given to information vs. peers in decision-making). The potential outcomes from such a case can be estimated by comparing a single school's outcomes moving diagonally up and to the left across Figure 2. For schools with the highest initial smoking prevalence, this would produce the most consistently health-promoting shifts. However, for schools with low to moderate initial smoking prevalence, such shifts appear to have little impact. Indeed, in schools with low to moderate initial smoking, *amplifying* the effectiveness of peer influence appears to be the more health-beneficial approach. In sum, neither the initial conditions nor the manipulated parameters solely accounted for the generated outcomes. This suggests that interventions must be tailored to contextual conditions to which they are to be applied, in ways that are aware of the initial prevalence of smoking behavior.

Beyond the scope of the particular questions here, these results also re-emphasize the need to consider how context shapes observed behavioral patterns and may shape the efficacy of any interventions designed to improve health behaviors and outcomes. Agent based models provide a framework to isolate particular components of such models in ways that are not available in real world interventions. This can be especially useful for predicting the implications of interventions that target those modeled characteristics (El-Sayed et al. 2012, El-Sayed et al. 2013). In a network context, the SABM approach used here could also be extended to examine multiple behaviors at once - e.g., how smoking and alcohol use are interrelated (Kiuru et al. 2010). While these are important advances for network scholarship and understanding health behaviors, there are also important implications for intervention work. In particular, future interventions could maximize their desired outcomes by targeting contexts where the model suggests the most beneficial outcomes. That is, if an existing intervention is able to produce identifiable peer influence (Campbell et al. 2008) and/or

popularity effects (Valente and Pumpuang 2007), our models can be used to identify initial smoking prevalence conditions in which those effects would maximally decrease smoking outcomes. More generally, as future health interventions are developed, a simulation modeling strategy like the one developed here can help identify optimal intervention roll-out scenarios that would best leverage the effects any particular intervention is able to generate.

Limitations and Extensions

By combining principled simulation scenarios with an empirical basis in a large nationally-representative sample of schools, we demonstrate how different initial conditions lead to different intervention outcomes, even when derived from the same behavioral mechanisms. We emphasized smoking prevalence as a key contextual moderator of known peer-influence processes. The relative consistency in our findings across the different initial prevalence conditions in our follow-up analysis (which included natural, systematic, and random manipulation of prevalence) demonstrates the robustness of our findings. However, prevalence is only one of many contextual factors that may shape the efficacy of smoking outcomes. Of utmost importance is further theoretical development regarding the contextual factors that moderate friendship-behavior dynamics, coupled with empirical tests. The model and framework developed here could be readily extended to evaluate the effects of initial smoking prevalence versus other contextual factors.

Our modeling strategy can also be extended to consider alternate intervention scenarios. First, our manipulations are based on relatively blunt-instrument intervention levers thought to alter peer and, in turn, smoking outcomes. More nuanced intervention levers should be explored, e.g., interventions administered to subsets of students--perhaps based upon particular characteristics, such as being an opinion leader (Valente and Pumpuang 2007; Valente et al. 2007). The results here present only the cumulative aggregation of net change across the simulated scenarios, masking the fact that even the scenarios producing the largest smoking decreases contain some initiations, and those with large increases include some cessations. Moreover, recent work demonstrates that smoking cessation and initiation may function via different processes (Haas and Schaefer 2014). The current model could be extended to manipulate peer influence and or smoker popularity effects *separately* for smoking cessation and smoking initiation. This would be especially useful given that interventions aimed at developing “refusal” skills likely have a stronger effect on inhibiting increases in smoking than on promoting smoking cessation.

Second, our observed change statistics only represent one year’s duration. Extending the amount of time over which we allow smoking and friendships to evolve within the model may alter some of the similarities/differences we observe across examined scenarios. Unfortunately, we need more and different data than are frequently available to inform data-based models for such longer duration effects. Third, given secular decreases over the past two decades, we can speculate that the low prevalence schools from the Add Health era more closely represent the conditions present in schools today. However, the adolescent smoking landscape may have changed enough over this time that even apparently similar conditions may embody different friendship-smoking dynamics that would produce dramatically different outcomes than we can estimate.

Conclusion

Researchers cannot simply export an intervention developed in one (or a few) context(s) into other locales and assume that it will produce the same outcomes, even if the mechanisms are reproduced in the same way. Altering peer processes, such as peer influence and/or smoking-based friend selection—even if reproduced identically across schools—can produce widely divergent smoking outcomes, depending on the schools' initial smoking levels. This contextual conditioning of potential intervention effects has implications for anyone attempting to alter adolescent smoking, or health behavior more generally. One must know both the assumed mechanisms by which the intervention is expected to function *and* the initial contextual conditions to which they are targeted, if interventions' potential effects are to be adequately predictable. We have demonstrated an empirically-grounded, simulation-based approach useful for estimating models that include such conditions and dynamic smoking and peer effects. We encourage other researchers to extend and modify this approach to investigate friendship-behavior processes that depart from those examined here (e.g., through other targeted intervention mechanisms).

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

This research was funded by the Eunice Kennedy Shriver National Institute of Child Health and Human Development (R21-HD071885) and a Loan Repayment grant from the National Institute of Minority Health and Health Disparities (L60-MD003181). We thank three anonymous JHSB reviewers, Michael Bader and Patrick Krueger for helpful comments on previous drafts. We used data from Add Health, a project directed by Kathleen Mullan Harris and designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill and funded by the Eunice Kennedy Shriver National Institute of Child Health and Human Development (grant P01-HD31921), with cooperative funding from 23 other federal agencies and foundations. Information about how to obtain the Add Health data files is available on the Add Health web- site (http://www.cpc.unc.edu/addhealth).

Biography

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References

Alexander C, Piazza M, Mekos D and Valente Thomas W. 2001 "Peers, Schools, and Adolescent Cigarette Smoking." Journal of Adolescent Health 29(1):22–30. [PubMed: 11429302]

Bearman Peter S., Jones Jo and Udry J. Richard. 1997 “The National Longitudinal Study of Adolescent Health: Research Design.”

Boardman Jason D., Saint Onge Jarron M., Haberstick Brett C., Timberlake David S. and Hewitt John K.. 2008 “Do Schools Moderate the Genetic Determinants of Smoking?” Behavioral Genetics 38:234–246.

Brechwald Whitney A. and Prinstein Mitchell J. 2011 “Beyond Homophily: A Decade of Advances in Understanding Peer Influence Processes.” Journal of Research on Adolescence 21(1):166–79. [PubMed: 23730122]

Bruch Elizabeth and Atwell Jon. 2014 “Agent-Based Models in Empirical Social Research.” Sociological Methods & Research forthcoming. doi: 10.1177/0049124113506405.

Campbell Rona, Starkey F, Holliday J, Audrey S, Bloor M, Parry-Langdon N, Hughes R and Moore L. 2008 “An Informal School-Based Peer-Led Intervention for Smoking Prevention in Adolescence (Assist): A Cluster Randomised Trial.” Lancet 371(9624):1595–602. [PubMed: 18468543]

CDC, Centers for Disease Control. 2008 “Smoking-Attributable Mortality, Years of Potential Life Lost, and Productivity Losses --- United States, 2000−−2004.” Morbidity and Mortality Weekly Report 57(45):1226–28. [PubMed: 19008791]

CDC, Centers for Disease Control. 2010 “Cigarette Use among High School Students—United States, 1991–2009.” Morbidity and Mortality Weekly Report 59(797–801). [PubMed: 20613702]

Christakis Nicholas A. and Fowler James H.. 2008 “The Collective Dynamics of Smoking in a Large Social Network.” The New England Journal of Medicine 358:2249–58. [PubMed: 18499567]

de la Haye Kayla, Green Harold D., Kennedy David R., Pollard Michael and Tucker Joan S.. 2013 “Selection and Influence Mechanisms Associated with Marijuana Initiation and Use in Adolescent Friendship Networks.” Journal of Research on Adolescence 23:474–86.

Eisenberg Marla E. and Forster Jean L.. 2003 “Adolescent Smoking Behavior: Measures of Social Norms.” American Journal of Preventive Medicine 25(2):122–28. doi: 10.1016/S0749-3797(03)00116-8.

El-Sayed Abdulrahman, Scarborough Peter, Seemann Lars and Galea Sandro. 2012 “Social Network Analysis and Agent-Based Modeling in Social Epidemiology.” Epidemiologic Perspectives & Innovations 9(1):1. [PubMed: 22296660]

El-Sayed Abdulrahman M., Seemann Lars, Scarborough Peter and Galea Sandro. 2013 “Are Network-Based Interventions a Useful Antiobesity Strategy? An Application of Simulation Models for Causal Inference in Epidemiology.” American Journal of Epidemiology. doi: 10.1093/aje/kws455.

Ennett Susan T., Bauman Karl E., Hussong Andrea, Faris Robert, Foshee Vangie A., Cai Li and DuRant Robert H.. 2006 “The Peer Context of Adolescent Substance Use: Findings from Social Network Analysis.” Journal of Research on Adolescence 16(2):159–86. doi: 10.1111/j.1532-7795.2006.00127.x.

Gest ScottD, Osgood D. Wayne, Feinberg MarkE, Bierman KarenL and Moody James. 2011 “Strengthening Prevention Program Theories and Evaluations: Contributions from Social Network Analysis.” Prevention Science 12(4):349–60. doi: 10.1007/s11121-011-0229-2. [PubMed: 21728069]

Green Harold D., Horta Mariana, Kayla de la Haye, Tucker Joan S., Kennedy David R. and Pollard Michael. 2013 “Peer Influence and Selection Processes in Adolescent Smoking Behavior: A Comparative Study.” Nicotine & Tobacco Research 15(2):534–41. [PubMed: 22944605]

Haas Steven A., Schaefer David R. and Kornienko Olga. 2010 “Health and the Structure of Adolescent Social Networks.” Journal of Health and Social Behavior 51(4):424–39. [PubMed: 21131619]

Haas Steven A. and Schaefer David R.. 2014 “With a Little Help from My Friends: Asymmetrical Social Influence on Adolescent Smoking Initiation and Cessation.” Journal of Health and Social Behavior forthcoming.

Hall Jeffrey A. and Valente Thomas W.. 2007 “Adolescent Smoking Networks: The Effects of Influence and Selection on Future Smoking.” Addictive Behaviors 32(12):3054–59. doi: 10.1016/j.addbeh.2007.04.008. [PubMed: 17509770]

Hoffman Beth R., Sussman Steve, Unger Jennifer B. and Valente Thomas W.. 2006 “Peer Influences on Adolescent Cigarette Smoking: A Theoretical Review of the Literature.” Substance Use & Misuse 41(1):103–55. doi: 10.1080/10826080500368892. [PubMed: 16393739]

Homer Jack B and Hirsch Gary B. 2006 “System Dynamics Modeling for Public Health: Background and Opportunities.” American journal of public health 96(3):452–58. [PubMed: 16449591]

Hwang Myunghee Song, Yeagley Kathleen Lux and Petosa Rick. 2004 “A Meta-Analysis of Adolescent Psychosocial Smoking Prevention Programs Published between 1978 and 1997 in the United States.” Health Education & Behavior 31(6):702–19. [PubMed: 15539543]

Ip Edward H., Rahmandad Hazhir, Shoham David A., Hammond Ross, Huang Terry T.-K., Wang Youfa and Mabry Patricia L. 2013 “Reconciling Statistical and Systems Science Approaches to Public Health.” Health Education & Behavior 40:s123–s31.

Kessler David A., Natanblut Sharon L., Wilkenfeld Judith P., Lorraine Catherine C., Mayl Sharon Lindan, Bernstein Ilisa B. G. and Thompson Larry. 1997 “Nicotine Addiction: A Pediatric Disease.” The Journal of Pediatrics 130(4):518–24. doi: 10.1016/S0022-3476(97)70232-4. [PubMed: 9108846]

Kiuru Noona, Burk William J., Laursen Brett, Salmela-Aro Katariina and Nurmi Jari-Erik. 2010 “Pressure to Drink but Not to Smoke: Disentangling Selection and Socialization in Adolescent Peer Networks and Peer Groups.” Journal of Adolescence 33(6):801–12. doi: 10.1016/j.adolescence.2010.07.006. [PubMed: 20732711]

Kobus Kimberly. 2003 “Peers and Adolescent Smoking.” Addiction 98:37–55. doi: 10.1046/j.1360-0443.98.s1.4.x. [PubMed: 12752361]

Koopman Jim. 2004 “Modeling Infection Transmission.” Annual Review of Public Health 25:303–26.

Lakon Cynthia M, Hipp John R and Timberlake David S. 2010 “The Social Context of Adolescent Smoking: A Systems Perspective.” American journal of public health 100(7):1218–28. [PubMed: 20466966]

Paula Lantz, Jacobson Peter D, Warner Kenneth E, Wasserman Jeffrey, Pollack Harold A, Berson Julie and Ahlstrom Alexis. 2000 “Investing in Youth Tobacco Control: A Review of Smoking Prevention and Control Strategies.” Tobacco control 9(1):47–63. [PubMed: 10691758]

Larson Reed and Richards Maryse H.. 1991 “Daily Companionship in Late Childhood and Early Adolescence: Changing Developmental Contexts.” Child Development 62(2):284–300. [PubMed: 2055123]

Levy David T, Mabry Patricia L, Graham Amanda L, Orleans C Tracy and Abrams David B. 2010 “Exploring Scenarios to Dramatically Reduce Smoking Prevalence: A Simulation Model of the Three-Part Cessation Process.” American journal of public health 100(7):1253–59. [PubMed: 20466969]

Lynam Donald R, Milich Richard, Zimmerman Rick, Novak Scott P, Logan TK, Martin Catherine, Leukefeld Carl and Clayton Richard. 1999 “Project Dare: No Effects at 10-Year Follow-Up.” Journal of consulting and clinical psychology 67(4):590. [PubMed: 10450631]

Mabry Patricia L, Marcus Stephen E, Clark Pamela I, Leischow Scott J and Méndez David. 2010 “Systems Science: A Revolution in Public Health Policy Research.” American journal of public health 100(7):1161–63. [PubMed: 20530757]

Maglio Paul P., Sepulveda Martin J. and Mabry Patricia L.. 2014 “Mainstreaming Modeling and Simulation to Accelerate Public Health Innovation.” American journal of public health 104(7):1181–86. doi: 10.2105/AJPH.2014.301873. [PubMed: 24832426]

Manski Charles F. 1993 “Identification of Endogenous Social Effects: The Reflection Problem.” The Review of Economic Studies 60(3):531–42. doi: 10.2307/2298123.

Marsden Peter V. 2011 “Survey Methods for Network Data” in The Sage Handbook of Social Network Analysis, edited by Scott J and Carrington PJ. Los Angeles, CA: Sage.

Mayhew Kathryn P., Flay Brian R. and Mott Joshua A.. 2000 “Stages in the Development of Adolescent Smoking.” Drug and Alcohol Dependence 59, Supplement 1(0):61–81. doi: 10.1016/S0376-8716(99)00165-9.

McPherson Miller, Smith-Lovin Lynn and Cook James M.. 2001 “Birds of a Feather: Homophily in Social Networks.” Annual Review of Sociology 27:415–44.

Mercken L, Snijders Tom A. B., Steglich Christian E. G., Vartiainen E and de Vries H. 2010 “Dynamics of Adolescent Friendship Networks and Smoking Behavior.” Social Networks 32:72–81.

Mollborn Stefanie, Domingue Benjamin W. and Boardman Jason D.. 2014 “Norms as Group-Level Constructs: Investigating School-Level Teen Pregnancy Norms and Behaviors.” Social Forces 93(1): 241–267.

Moody James, Brynildsen Wendy D., Osgood D. Wayne, Feinberg Mark E. and Gest Scott. 2011 “Popularity Trajectories and Substance Use in Early Adolescence.” Social Networks 33(2):101–12. doi: 10.1016/j.socnet.2010.10.001. [PubMed: 21765588]

Pearson Michael, Steglich Christian and Snijders Tom. 2006 “Homophily and Assilimation among Sport-Active Adolescent Substance Users.” Connections 27(1):51–68.

Peterson Arthur V, Kealey Kathleen A, Mann Sue L, Marek Patrick M and Sarason Irwin G. 2000 “Hutchinson Smoking Prevention Project: Long-Term Randomized Trial in School-Based Tobacco Use Prevention—Results on Smoking.” Journal of the National Cancer Institute 92(24):1979–91. [PubMed: 11121460]

Ripley Ruth M, Snijders Tom AB, Boda Zsofia, Voros Andras and Preciado Paulina. 2014 “Manual for Rsiena.” University of Oxford: Department of Statistics, Nuffield College.

Robins Garry, Pattison Philippa, Kalish Yuval and Lusher Dean. 2007 “An Introduction to Exponential Random Graph (p*) Models for Social Networks.” Social Networks 29(2):173–91.

Rogers Everett M. 1995 Diffusion of Innovations. New York: The Free Press.

Rossman Gabriel. 2012 Climbing the Charts: What Radio Airplay Tells Us About the Diffusion of Innovations: Princeton University Press.

Salathe Marcel and Jones James H.. 2010 “Dynamics and Control of Diseases in Networks with Community Structure.” PLoS Computational Biology 6(4):e1000736. [PubMed: 20386735]

Sambrano Soledad, Springer J. Fred, Sale Elizabeth, Kasim Rafa and Hermann Jack. 2005 “Understanding Prevention Effectiveness in Real-World Settings: The National Cross-Site Evaluation of High Risk Youth Programs.” The American Journal of Drug and Alcohol Abuse 31:491–513. [PubMed: 16161731]

Schaefer David R., Haas Steven A. and Bishop Nicholas J.. 2012 “A Dynamic Model of Us Adolescents’ Smoking and Friendship Networks.” American journal of public health 102(6):e12–e18.

Schaefer David R., adams jimi and Haas Steven A.. 2013 “Social Networks and Smoking: Exploring the Effects of Peer Influence and Smoker Popularity through Smoking Simulations.” Health Education & Behavior 40(S1):24–32. [PubMed: 22491009]

Shalizi Cosma Rohilla and Thomas Andrew C.. 2011 “Homophily and Contagion Are Generically Confounded in Observational Social Network Studies.” Sociological Methods & Research 40(2): 211–39. [PubMed: 22523436]

Snijders Tom A. B., van de Bunt Gerhard and Steglich Christian E. G.. 2010 “Introduction to Stochastic Actor-Based Models for Network Dynamics.” Social Networks 32:44–60.

Snijders Tom A. B. 2011 “Statistical Models for Social Networks.” Annual Review of Sociology 37:129–51.

Snijders Tom A. B. and Steglich Christian E. G.. 2013 “Representing Micro-Macro Linkages by Actor-Based Dynamic Network Models.” Sociological Methods & Research forthcoming.

Steglich Christian, Snijders Tom A. B. and Pearson Michael. 2010 “Dynamic Networks and Behavior: Separating Selection from Influence.” Sociological Methodology 40(1):329–93.

Tobler Nancy S., Roonia Michael R., Ochshorn Peter, Marshall Diana G., Streke Andrei V. and Stackpole Kimberly M.. 2000 “School-Based Adolescent Drug Prevention Programs: 1998 Meta-Analysis.” Journal of Primary Prevention 20(4):275–336.

Valente Thomas W. 2012 “Network Interventions.” Science 337:49–53. [PubMed: 22767921]

Valente Thomas W. 1995 Network Models of the Diffusion of Innovations. Cresskill, NJ: Hampton Press.

Valente Thomas W. 1996 “Social Network Thresholds in the Diffusion of Innovations.” Social Networks 18(1):69–89.

Valente Thomas W. and Davis Rebecca L.. 1999 “Accelerating the Diffusion of Innovations Using Opinion Leaders.” The Annals of the American Academy of the Political and Social Sciences 566:55–67.

Valente Thomas W., Unger Jennifer B. and Johnson C. Anderson. 2005 “Do Popular Students Smoke? The Association between Popularity and Smoking among Middle School Students.” Journal of Adolescent Health 37(4):323–29. [PubMed: 16182143]

Valente Thomas W. and Pumpuang Patchareeya. 2007 “Identifying Opinion Leaders to Promote Behavior Change.” Health Education & Behavior 34(6):881–96. doi: 10.1177/1090198106297855. [PubMed: 17602096]

Valente Thomas W., Anamara Ritt-Olson Alan Stacy, Unger Jennifer B., Okamoto Janet and Sussman Steve. 2007 “Peer Acceleration: Effects of a Social Network Tailored Substance Abuse Prevention Program among High-Risk Adolescents.” Addiction 102(11):1804–15. doi: 10.1111/j.1360-0443.2007.01992.x. [PubMed: 17784893]

Valente Thomas W. 2010 Social Networks and Health: Models, Methods, and Applications. New York: Oxford University Press.

Wiehe Sarah E, Garrison Michelle M, Christakis Dimitri A, Ebel Beth E and Rivara Frederick P. 2005 “A Systematic Review of School-Based Smoking Prevention Trials with Long-Term Follow-Up.” Journal of Adolescent Health 36(3):162–69. [PubMed: 15737770]

Zhang J Tong L, Lamberson PJet al. 2014 “Leveraging Social Influence to Address Overweight and Obesity using Agent-Based Models: The Role of Adolescent Social Networks.” Social Science and Medicine forthcoming.

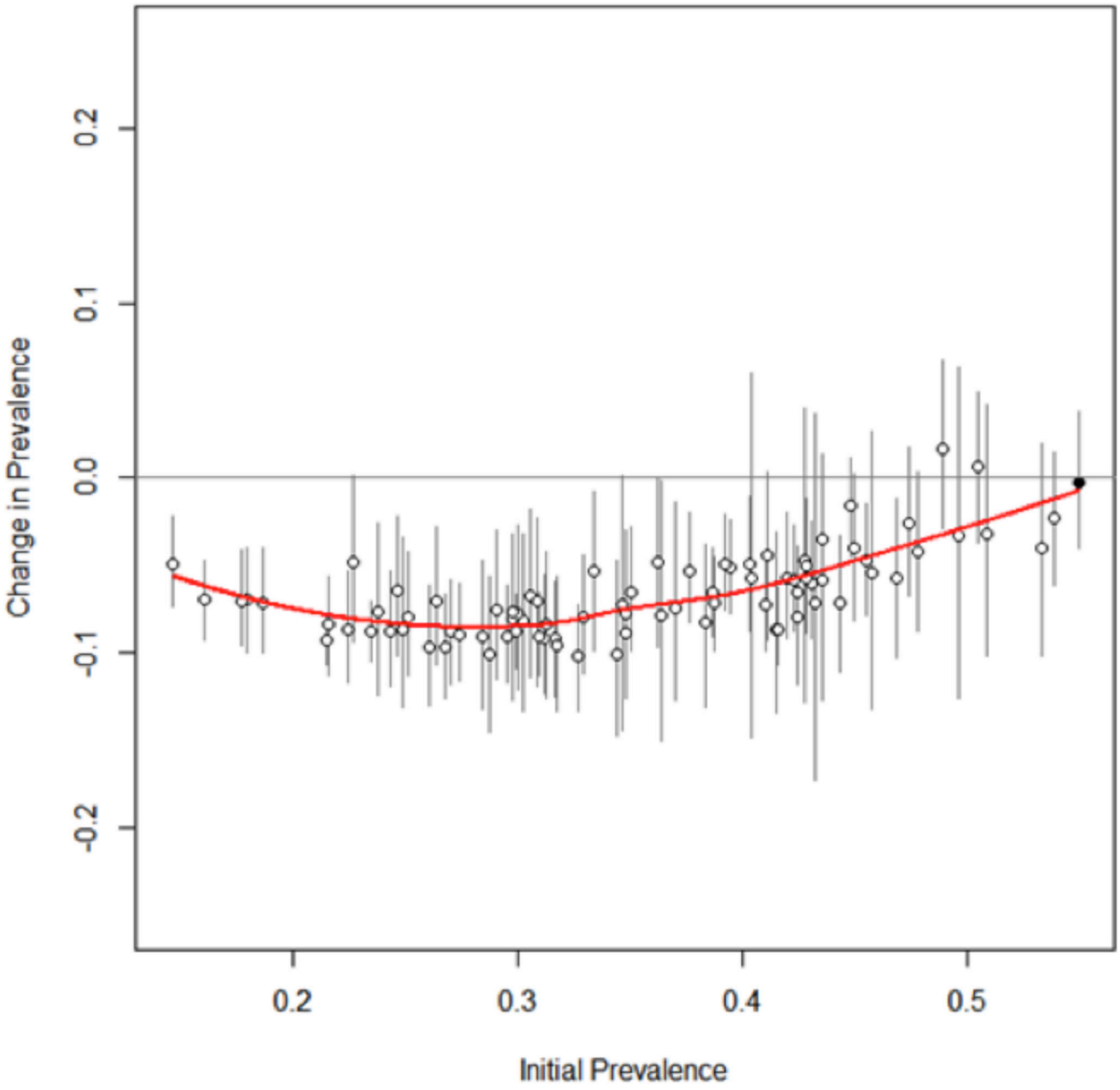

Figure 1. Smoking Changes from Applying Observed Peer Influence and Popularity Effects to 85 Schools

NOTE: The school upon which the model parameters are based (from SAH) is highlighted with a filled circle. The red line is a fitted LOESS curve. Error bars present 95% confidence intervals.

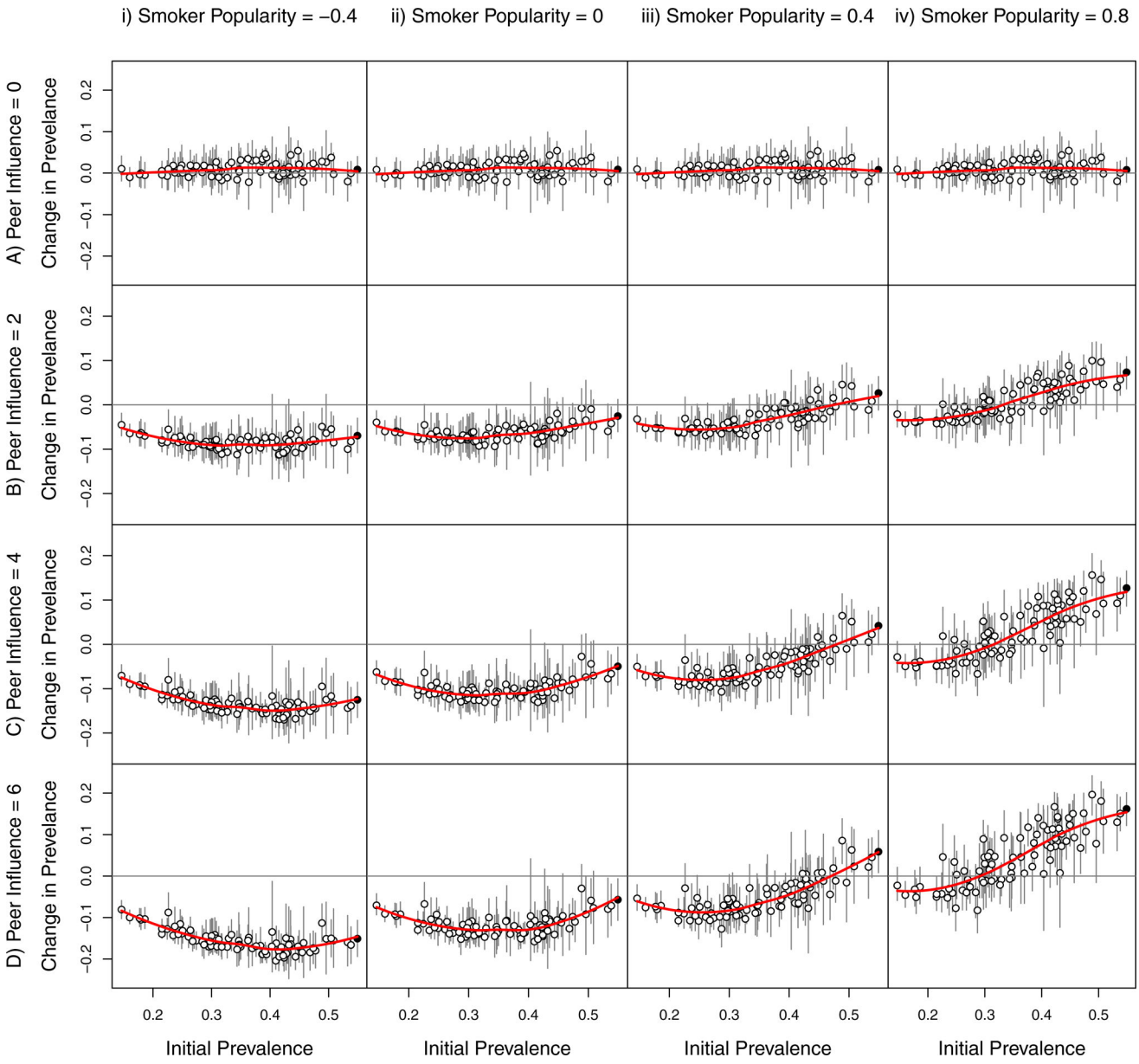

Figure 2. Simulated Smoking Prevalence Changes from Intervention Scenarios Combining Peer-Influence and Population Effects

NOTE: The school upon which the model parameters are based (from SAH) is highlighted with a filled circle. The red line is a fitted LOESS curve. Error bars present 95% confidence intervals. The peer influence effects range from 0 (no peer influence) to 6 (approximately twice the strength observed in SAH) and the popularity effects range from moderately negative (indicating smokers are less popular as friends) to strongly positive (friend preference for smokers). Both effects are centered on their observed values. For interpretation of parameter sizes, see the SABM Estimation section.

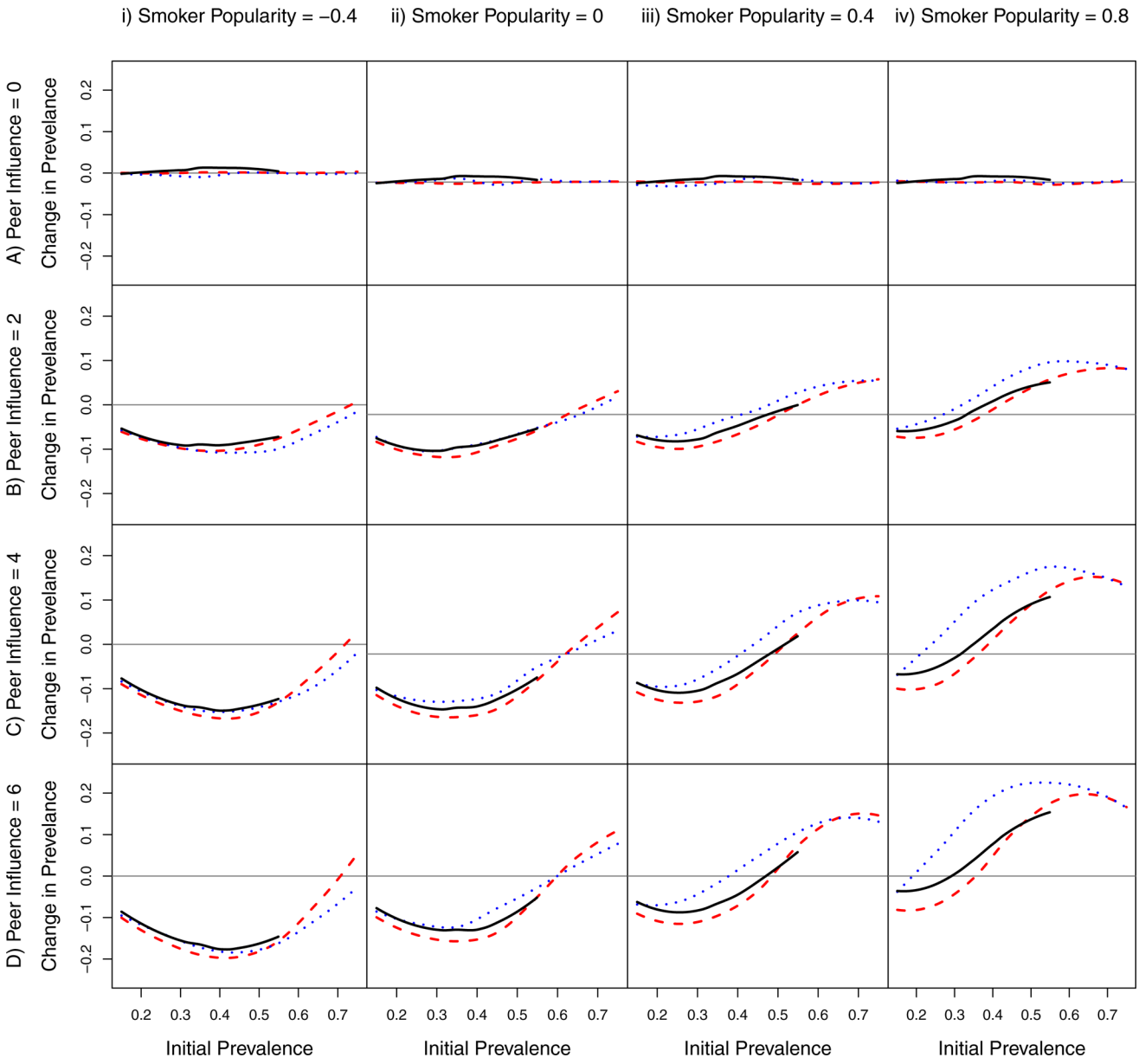

Figure 3. Comparison of Different Baseline Assumptions for Simulated Smoking Prevalence Changes from Intervention Scenarios

NOTE: Line type denotes baseline-condition assignment procedure: (a) Random assignment is presented with the dashed line, (b) Model-based as the dotted line, and (c) Empirically-derived as the solid line. For interpretation of parameter sizes, see the SABM Estimation section.

Table 1.

School level descriptive statistics (N=85)

	Mean	SD	Min	Max
School size	661	464	81	2097
Smoking T1 (proportion)	.230	.086	.032	.472
Smoking T2 (proportion)	.247	.101	.016	.494
Smoking change T1–T2 (proportion)	.017	.068	−.163	.171
Correlation of smoking & indegree	.004	.076	−.187	.173
Smoking autocorrelation (Moran's I)	.218	.092	−.024	.397
Density	.011	.011	.001	.074
Mean Degree	4.429	.915	1.830	5.958
Reciprocity	.241	.037	.143	.329
Transitivity	.201	.050	.113	.389