Simulating Dynamic Network Models and Adolescent Smoking: The Impact of Varying Peer Influence and Peer Selection

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We used a stochastic actor-based approach to examine the effect of peer influence and peer selection—the propensity to choose friends who are similar on smoking among adolescents. Data were collected from 1994 to 1996 from 2 schools involved in the National Longitudinal Study of Adolescent to Adult Health, with respectively 2178 and 976 students, and different levels of smoking. Our experimental manipulations of the peer influence and selection parameters in a simulation strategy indicated that stronger peer influence decreased school-level smoking. In contrast to the assumption that a smoker may induce a nonsmoker to begin smoking, adherence to antismoking norms may result in an adolescent nonsmoker inducing a smoker to stop smoking and reduce school-level smoking. (Am J Public Health. 2015;105:2438–2448. doi:10.2105/ AJPH.2015.302789)

Cigarette smoking trajectories beginning in adolescence often persist into adulthood, 1 as most adult smokers begin in adolescence.² Adolescents are uniquely susceptible to peer influence3,4 making friendship networks a primary socialization context shaping smoking and friendship tie choice. Within an adolescent friendship network, youths are likely to engage in health-promotive behaviors if they affiliate with friends who condone health-promotive peer influences. Conversely, youths may engage in health-compromising behavior if their friends exert deleterious peer influences, as supported by insights from differential association theory and social control theory.5,6 Numerous studies indicate a positive relationship between friends' smoking behavior and smoking among adolescents.^{7,8}

Social contagion models have been applied to examine the diffusion of healthcompromising social influences and behavior through social networks, including smoking.^{4,9} Less research, however, has focused on the possibility that the diffusion of peer influence in a network may result in deleterious or salutary consequences for adolescent smoking, and even fewer studies have considered these effects at a population level. A recent review article on peer influence and adolescent development stressed the need for studies that consider the salutary effects of peer influence.³

Only a few studies have indicated that peer socialization is protective against tobacco use among adolescents.^{10,11}

Although peer influence is a potent socialization force shaping adolescent smoking, it does not act alone. Peer selection, the propensity to choose friends who are similar, is based on the principle of homophily, 12 and is another salient process affecting adolescents' friendship networks. Adolescents select friends who are similar to themselves on multiple dimensions.13 Peer selection is an alternative explanation to peer influence for the similarity in behavior among adolescent friends on many dimensions including smoking.¹⁴

Stochastic actor-based models have yielded keen insights into adolescent smoking by disentangling the endogenous processes of peer influence and selection, 15 while considering structural, triadic, and degree-based adolescent network characteristics. $16-19$ Studies to date, however, have not examined how changes in peer influence and selection affect the individual and aggregate smoking level of adolescent populations. Adolescents' schools are a policyrelevant contained social system in which to investigate both individual and school-level smoking. We focused on these effects at both levels, as the former may give insight into the diffusion of smoking-related peer influences at an individual level within friendship networks, and the latter may be a proxy for school-level smoking norms. Understanding how smoking at both levels is affected by perturbations in these processes likely yields information about the relationship between the transmission of peer influences and selection and the simultaneous formation of school smoking-level norms, which is key for building school-based network interventions harnessing peer influence. Moreover, the approach of examining individual- and school-level smoking within a simulation framework gives insight into the sensitivity of a network system, at each level of smoking, to alterations in peer influence and selection, which is key for informing interventions.

We examined how youths' behavioral decisions regarding smoking and friendship tie choices coevolve under varying levels of peer influence and selection in school-based social network systems, which affect the overall smoking levels of these schools. We used the 2 largest schools from the special oversample from the National Longitudinal Study of Adolescent to Adult Health (Add Health) containing network information, termed the "saturation sample" of 3145 youths in grades 9 through 12, surveyed 3 times during 1994 to 1996. We employed a stochastic actor-based model, which captures the coevolving processes of friendship tie choice and smoking.²⁰

As networks can behave as a system that exhibits nonlinearities and threshold effects, we experimentally manipulated the size of the peer influence and selection parameters over a wide range of values to explore the behavior of the network at the extreme bounds of influence and selection effects. We then simulated the network forward 1000 times for each experimental condition and assessed the level of smoking behavior at the individual and school levels. We examined the distribution of smokers and the level of smoking in the population following these manipulations. We

also assessed the extent of clustering, or the occurrence of highly connected groupings of adolescents in the network, to understand how altering peer influence and selection may parse youths into densely connected clusters in the school network with youths displaying similar smoking behaviors within cluster.

Few studies have used such a simulation technique to explore the consequences of changes in network processes for adolescent substance use. One exception was a study exploring the consequences for adolescent smoking when manipulating peer influence and popularity in one school from the Add Health study. 21 This previous research provides key insights regarding the effects of popularity on smoking behavior; the current study complements and diverges from this study by explicitly focusing on the consequences of manipulating the size of both peer influence and selection effects over a wider range of values in 2 schools with differing levels of smoking.

METHODS

Data are from 3 waves of the AddHealth survey, 22 including the wave-1 interviews occurring in school (i.e., in-school survey), collected from September 1994 to April 1995; the wave-2 interviews occurring at home 6 months later (i.e., wave-1 in-home survey) from April to December 1995; and the wave-3 interviews also occurring at home 1 year later (i.e., wave-2 in-home survey). Over the 3 waves, information describing the demographic characteristics of adolescents, their parents, and adolescents' risk behaviors including smoking was collected. We used network data from the 2 largest schools—both public, one in a rural Midwestern community and one in a suburban Western community $(n=2178$ and $n=976$)-of the 16 schools in the saturation sample. 22 These are the only 2 high schools in the sample. More importantly, they represent relative extremes in school levels of smoking behavior, allowing comparison of the effect of the smoking context in the school. Whereas the largest school has relatively low levels of smoking (i.e., it is nearly 1 standard deviation below the mean smoking level of all 133 schools in the wave-1 nationally representative Add Health school sample), the second school has the highest level of smoking in the saturated sample and is almost 2 standard deviations above the mean school smoking level of the wave-1 Add Health school sample.

To address missing network data at wave 1, we used the latent missing data approach,^{23,24} which uses an exponential random graph model to estimate the probability that a tie exists among the missing network data in the first wave, and then imputes these probabilities accordingly. Recent research shows that this more principled approach is more justified than other approaches to handling missing data (e.g., excluding cases), yielding results that can be noticeably different.²⁵ Data missing in the later waves are treated as noninformative by RSiena (version 1.1-278, University of Oxford, Department of Statistics, Nuffield College at Oxford, Oxford, England).²⁶ Indeed, the total outgoing ties by wave 3 in the networks declined because of graduation, moving, dropping out, attrition, nonresponse, or missing network data.

Dependent Variables

At wave 1, the item measuring smoking was "During the past 12 months, how often did you smoke cigarettes?" $(0 =$ never; 1 = once or twice; $2 =$ once a month or less; $3 = 2$ or 3 days a month; $4 =$ once or twice a week; $5 = 3-5$ days a week; $6 =$ nearly every day). At waves 2 and 3, the question was "During the past 30 days, on how many days did you smoke cigarettes?" measured continuously $(0 = no$ days to $30 = 30$ days). The variable we used across the 3 waves recategorizes the response categories across the 2 items such that they match the category framing over waves: 0 = never; $1 = 1$ to 3 days; $2 = 4$ to 21 days; $3 = 22$ or more days.

To create the adolescent friendship networks, each adolescent nominated up to 5 female and 5 male best friends in his or her school from a roster. We used this information to create the school-specific friendship network. The variable friendship tie choice is the presence or absence of a tie.

Independent Variables

We included several network measures in the friendship tie choice equation to account for key degree and structural network effects

leading to network self-organization and to minimize bias in our estimated model parameters. These network effects have also been examined in previous studies of adolescent smoking that used a stochastic actor-based model approach.²⁷ Rate functions for each of the 2 periods between waves capture the average number of times a person considers changing friendship ties between waves (friendship rate). Out-degree captures the propensity to send a tie to another adolescent, and reciprocity indicates the extent to which ties are reciprocated. Higher values of the reciprocity parameter indicate that an adolescent is more likely to name another adolescent as a friend if that person had already named him or her as a friend. Transitive triplets indicate the tendency for an adolescent to nominate a friend of a friend, and 3 cycles reflect the tendency for a friend's friend to nominate the respondent as a friend; these 2 effects indicate the presence of triadic closure within a friendship network (i.e., the tendency for groups of 3 persons to form friendship triads). In-degree popularity is the propensity to choose popular youths as friends. In-degree assortativity (square root) is the propensity to choose friends who are similarly popular as oneself.²⁶

We assessed various homophily effects (i.e., the tendency to form ties to others who are similar on key dimensions). We used the term "similarity" to capture these homophilic tendencies as this term is commonly used in the stochastic actor-based modeling framework. The measure of similarity in smoking behavior between respondent and alter (i.e., friend) assesses whether adolescents are more likely to form friendship ties with others who are similar in smoking behavior (i.e., a selection effect). Because friendship ties are more likely to form among adolescents in the same grade, we included a grade similarity effect. Likewise, adolescents may be more likely to form friendship ties with others from a similar socioeconomic background and we assessed this with a measure of parental education similarity. In the largest school, we included a measure of race similarity to capture race homophily; the smaller school was too racially homogeneous to include such a measure.

We also assessed whether certain characteristics of respondents affect their friendship

tie choices. A measure of the smoking behavior of the ego (i.e., respondent) assesses the tendency of adolescents who smoke to form more friendship ties than those who do not smoke.^{28,29} The measure capturing the smoking behavior of the alter assesses whether higher-level smokers are more likely to receive friendship ties (i.e., are more popular).³⁰

Because of an administrative error, some participants (fewer than 5%) were only able to choose 1 male friend and 1 female friend during the wave-1 in-home and wave-2 inhome surveys. We accounted for this error with a limited nomination variable (measured as -1 = changed from full to limited nominations; $0 = no$ change; and $+1 =$ changed from limited to full nominations).

In the equation predicting change in smoking behavior over time, we included rate functions to estimate the average number of possible changes in smoking behavior (smoking behavior rate) between waves. The smoking linear shape effect and the smoking quadratic shape effect capture the general tendency to change smoking behavior over time. Whereas a negative quadratic effect would indicate that persons who smoke at the first time point are less likely to increase their smoking behavior, a positive quadratic effect indicates that those who smoke are more likely to increase their level of smoking rather than decrease it. We also included key degree and structural network effects on smoking behavior to minimize the possibility of biased parameter estimates. A measure of in-degree assesses whether adolescents who are named as friends by others more frequently (i.e., are more popular) smoke more over time. We assessed peer influence effects with a measure of similarity of behaviors between ego and all his or her alters averaged by ego's out-degree. This assesses whether adolescents are more likely to change their smoking behavior to match that of their peers over time (i.e., peer influence effect).

We accounted for gender with a measure of female. In the larger school we coded variables for (1) Black and (2) other race/ethnicity (mixed race, Asian, Latino, Native American, and other). The smaller school was racially homogenous. We constructed a measure of depressive symptoms as a factor score of 19 items modified from the Center for Epidemiologic

Studies Depression Scale assessing past-week mood (Cronbach α = 0.83).³¹

Because of the relevance of parental influences for adolescent smoking, we included indicators of 3 key parental influences: parental support, $32-35$ parental monitoring, $36,37$ and the parental home smoking environment. We measured parental support as a factor score (Cronbach α = 0.80) and it is premised upon items including whether adolescents have talked about a personal problem with their parents $(0 = no; 1 = yes)$ and 5 other items. Respondents separately rated each parent as

- 1. warm and loving,
- 2. a good communicator,
- 3. part of an overall good relationship (response categories for all 3 items were $1 =$ strongly disagree; $2 =$ disagree; 3 = neither agree nor disagree; 4 = agree; $5 =$ strongly agree);
- 4. close, and
- 5. caring (the response categories for the latter 2 items were $1 = not$ at all; $2 = very$ little; $3 =$ somewhat; $4 =$ quite a bit; $5 =$ very much).

We measured parental monitoring as a factor score (Cronbach α = 0.60), which combines 10 questions, with the first 7 related to whether the adolescents were allowed to decide

- 1. their weekend curfew,
- 2. with whom they hang around,
- 3. what they wore,
- 4. what they ate,
- 5. what they watched on TV,
- 6. how much TV they watched, and
- 7. their weekday bedtime (with the response categories of $0 = yes$ and $1 = no$).

The remaining 3 questions measuring parental monitoring indicated whether the parent was present when an adolescent came home from school $(0 = never; 1 = almost$ never; $2 =$ some of the time; $3 =$ most of the time; $4 =$ always; $5 =$ they brought the student home from school), went to bed $(0 =$ never; $1 =$ almost never; $2 =$ some of the time; $3 =$ most of the time; $4 =$ always), and ate dinner (0 to 7 days per week). We measured parental home smoking environment by summing dichotomous measures of parent smoking behavior^{38,39} and cigarette availability in the home.

Statistical Analyses

We used a stochastic actor-based model, which attempts to account for the dynamic nature of friendship tie choice and smoking behavior.²⁶ Although the longitudinal data were collected at 3 discrete time points, the model tries to capture these processes in approximately continuous time through an agent-based simulation. At each microstep, an actor is randomly chosen and he or she then chooses to increase, decrease, or leave unchanged his or her level of smoking behavior, or else chooses whether to dissolve a current tie or form a new one. The actor makes these decisions based on his or her current state of network-behavioral configuration, which is referred to as the objective function. The objective function is defined as

$$
(1) f(b,x) = \sum_{k} b_k s_{ik}(x),
$$

where b_k is the kth estimated parameter for the actor-specific effect, $s_{ik}(x)$, and x is the joint network-behavioral state. Positive parameter values in the network equation of the objective function indicate that a person is more likely to dissolve or form a tie if it increases that state for a person: for example, a positive value of the reciprocity parameter (described earlier) would indicate that a person is more likely to form a tie with someone if that person already named him or her as a friend (i.e., reciprocating the friendship).

In the next microstep, another actor is chosen randomly, who makes a decision based on his or her own objective function, and the process reiterates over subsequent discrete time periods. The number of such decisions regarding changing network ties or changing smoking behavior between 2 waves of data is determined by the rate functions (as described in the preceding section). In the statistical software program RSiena, the objective function of network changes and behavior changes are estimated simultaneously to generate both a network and a behavioral equation. We estimated the models with the RSiena software program by simulating the networks and behavior forward in time with a method of moments estimator. Our analytic strategy can be found as supplement to the online version of this article at [http://www.ajph.org.](http://www.ajph.org)

RESULTS

The summary statistics for the 2 schools are presented in Tables A1 to A3 (available as a supplement to the online version of this article at [http://www.ajph.org\)](http://www.ajph.org). The most notable difference is that school 077 (n = 2178) had much less smoking behavior than school 058 (n = 976): between 7% and 10% of the adolescents smoked 22 or more days per month across the 3 waves in school 077 compared with 26% to 32% of those in school 058. There was somewhat less reciprocity in the network of school 077 than that of school 058, as 30% of ties were reciprocated compared with 38% of ties, on average.

We first estimated stochastic actor-based models to obtain parameter estimates for our simulations, and the results across the 2 schools are displayed in Table A4 (available as a supplement to the online version of this article at [http://www.ajph.org\)](http://www.ajph.org). Of key interest is the evidence in both schools of selection effects for friendship tie choice and a peer influence effect for smoking. We observed the selection effect in the smoking similarity variable in the friendship tie choice model, which shows effects of $b = 0.18$ (P \leq 01; 95% confidence interval $[CI] = 0.07, 0.30$ and $b = 0.26$ $(P<.001; 95\% CI = 0.21, 0.31)$ in schools 077 and 058, respectively. We observed the peer influence effect in the smoking behavior similarity measure in the smoking behavior equation, which showed positive effects of $b = 0.54$ (P < .001; 95% CI = 0.11, 0.97) and $b = 0.80$ (P < .001; 95% CI = 0.62, 0.98) for each school, respectively.

Regarding model fit, we assessed and found that our model adequately reproduced the network and the behavior of individuals in the schools (Figures A1 and A2, available as a supplement to the online version of this article at [http://www.ajph.org\)](http://www.ajph.org).

When we experimentally manipulated the selection parameter, Figure 1 shows that altering the level of selection had minimal impact on the composition of smokers and nonsmokers in the school. In the lower-smoking school (077), there was almost no change in the number at each level of the smoking variable. In the higher-smoking school (058), there was a modest decline in the number of nonsmokers when selection was not present: this is the left side of the figure, where nonsmokers have fallen from 49% in the model with the selection parameter at its estimated values to 45% with no selection. The percentage of those smoking 1 to 3 or 4 to 21 days per month each increased 2 percentage points when we turned off selection. As we increased selection to very high positive values, the composition of smokers did not change very much. Even in a situation of very strong selection effects, the percentage of nonsmokers had increased to just 53%. It was interesting that the percentage of those smoking 22 or more days per month also increased slightly—1.7 percentage points—in this hypothetical setting with very strong selection effects, suggesting a slight degree of polarization in the network. The impact of tuning up selection was quite modest in the lower-smoking school (077), as the percentage of those smoking 22 or more days per month increased just 2 percentage points in a scenario of very high selection effects.

Although stronger selection effects did not appear to change the composition of smokers in a school, they did change the clustering of smoking behavior in a school. Whereas the Moran I was 0.20 and 0.32 in schools 077 and 058, respectively, in the observed data, this increased to 0.41 and 0.90, respectively, in the simulation with the strongest selection effect. Thus, the higher-smoking school (058) had almost complete clustering by smoking behavior in the network.

Turning to the experimental manipulations of the peer influence parameter, we observed much stronger effects (Figure 2). Notably, increasing peer influence appeared to have a strong diminishing effect on smoking behavior. In the experimental manipulation with no influence effects, the percentage of nonsmokers decreased 4.1 and 11.6 percentage points in schools 077 and 058, respectively, compared with the observed sample, whereas the number of those smoking 22 or more days increased 2.3 and 9 percentage points, respectively. When we increased the influence parameter, we found that it had an even stronger diminishing effect on smoking behavior. In the most extreme case with very strong peer influence effects there were 21 and 17.9 percentage points, respectively, more nonsmokers in the 2 schools. There was

a consequent decrease in smoking at all levels, particularly the heaviest smokers who declined 12 and 8.3 percentage points in this hypothetical scenario of very heavy influence effects.

When we increased the influence parameter, we found a change in the amount of clustering in smoking behavior in the school; however, this differed on the basis of the level of smoking in the school. In the lower-smoking school (077) the Moran I decreased from 0.20 in the observed data to 0.15 in the manipulation with very strong influence effects. However, in the higher-smoking school (058) the Moran I increased from 0.32 in the observed sample to 0.67 in a situation of very strong influence effects, suggesting considerable clustering in the network on the basis of smoking behavior.

We next assessed the effects of peer influence and selection by manipulating their parameter values simultaneously. When we assessed the percentage of smokers at the end of each simulation (regardless of their level of smoking), Figure 3 demonstrates that, for both schools, the effect of stronger influence dominated the effect of stronger selection. The lines in the figure show the range of manipulated values for the selection parameter, whereas the x-axis shows the range of manipulated values for the influence parameter. The y-axis shows the average percentage of smokers at the end of the simulations for each of the manipulations. We see that with stronger influence effects (moving from left to right) the percentage of smokers at the end of the simulation runs steadily fell. For any given parameter value of the peer influence effect, the value of the selection parameter made minimal difference. With weaker influence, there was some evidence that stronger selection effects somewhat reduced the percentage of smokers. Nonetheless, the predominant pattern in these graphs is one in which increasing levels of influence most strongly reduce the percentage of smokers.

We next assessed how simultaneously manipulating the influence and selection parameters affected the percentage of youths who start smoking at any level by the end of the simulation. In Figure 4, we see that for the lower-smoking school (077), a larger influence parameter had the strongest inhibitory effect on smoking initiation. As we manipulated the

influence parameter to larger values in school 077, the percentage of adolescents who begin smoking steadily decreased, regardless of the size of the selection parameter. It appears that stronger selection effects did not increase the number of new smokers in this school with lower levels of smoking behavior in general. However, in the higher-smoking school (058) we detected strong reinforcing effects: whereas stronger influence effects resulted in a smaller proportion of smoking initiators, this inhibitory effect was strongest when it was accompanied by a strong selection effect. It appears

that selecting friends on the basis of their smoking behavior along with a strong influence effect (in the context of relatively many smokers available) led to the greatest reduction in smoking initiation.

We next computed the proportion of youths who no longer engaged in any smoking at the end of the simulation run. In Figure 5, we see for the lower-smoking school (077) that stronger influence effects monotonically increased the number who stopped smoking. In the higher-smoking school (058), we again see a synergistic effect: whereas altering the influence parameter to larger values resulted in

greater levels of smoking cessation, this effect was diminished when the selection parameter was tuned to larger values. Thus, in a school environment with relatively high levels of smoking, higher levels of influence and lower levels of selection resulted in greater levels of youths ceasing to smoke.

DISCUSSION

A substantial body of literature has demonstrated the relevance of stochastic actorbased models for understanding how peer influence and selection shape adolescent

friendship tie choice and smoking. $17,40-42$ Whereas the present study focused on disentangling the effects of peer influence and selection in affecting smoking behavior, it also focused on a relatively unexamined question, which is how adolescent smoking behavior and clustering in a network are affected when levels of peer influence and selection are varied. Although our stochastic actor-based models indicated both peer influence and selection effects at the individual level, our simulation experiments indicated that higher levels of peer influence or higher levels of

selection either alone or simultaneously did not lead to increases in school levels of smoking. In fact, higher levels of peer influence actually reduced school smoking levels. Our study suggests the relevance of peer influence for school- and individual-level smoking in these networks.

The effects of peer influence on adolescent smoking are nuanced, as the consequences of peer influence for adolescent smoking should be examined at both the individual and school levels. The sometimes implicit assumption that a smoker may induce a nonsmoker to begin

smoking places less emphasis on the possibility that an adolescent nonsmoker may induce a smoker to become a nonsmoker. The latter scenario is likely a consequence of antismoking norms held by the majority in a school, and when peer influences aligned with these antismoking norms are strengthened, smoking behavior decreases at the school level.

Our experimental manipulations indicated that setting the peer influence parameter to larger values resulted in fewer adolescents in the networks smoking overall, less smoking initiation, and more adolescents ceasing to

FIGURE 3—Jointly altering selection and influence parameters for percentage of smokers in (a) high school 077 and (b) high school 058: National Longitudinal Study of Adolescent to Adult Health, United States, 1994–1995.

smoke. Moreover, eliminating the peer influence effect in these network experiments actually resulted in more smokers, adding further evidence that peer influence might lessen overall levels of adolescent smoking in a school. It is possible that this effect is attributable to the relative level of smoking in the schools, implying that there may be

many students who hold and transmit antismoking peer influences, which may in turn decrease smoking at the school level. This finding is consistent with previous research suggesting that peer influence is a salutary socialization force for adolescent development that protects against deleterious health outcomes including smoking.10,11 It is

unclear, however, how the school-level smoking distribution would be affected over a longer observation time period: would school-level smoking continue to decelerate over time, or would nonlinearity be observed in this effect?

Our findings also indicate that selection had little effect on the overall level of smoking in a network with a relatively low level of smoking behavior. This may have been attributable to less clustering of youths by smoking status in light of the lower level of smoking behavior to begin with. However, in the school network with higher levels of smoking, there was a synergistic effect between peer influence and selection, such that the lowest level of smoking initiation occurred in the manipulation of both strong peer influence and selection. It may be that the high level of selection among smokers and nonsmokers resulted in clustering into groups by smoking status and, therefore, the higher levels of peer influence within clusters kept youths from beginning smoking because of the homogeneity of influences and norms that can occur within clustered areas of networks. A scenario of strong peer influence and weak selection resulted in high proportions of youths ceasing to smoke. Thus, strong peer influence and weak selection results in lower levels of overall smoking in a context that starts with higher levels of smoking, likely because of the possibility of selecting friends with different smoking behavior and the reinforcement of antismoking peer influences within antismoking clusters. Whether such an effect would be even stronger in a context with even higher levels of smoking behavior—for example, in continuation high schools for youths who have left mainstream schools because of truancy or drug use 43 —is an avenue for future research.

Implications

Our study suggests merit in the strategy of examining the sensitivity of a network system to alterations in peer influence and selection over time for adolescent networks and smoking as a precursor to building interventions. Understanding first how a network system responds to such fluctuations yields information for how to begin building peer influence-based network interventions. This approach may also facilitate our understanding of how

FIGURE 4—Jointly altering selection and influence parameters for new smokers in (a) high school 077 and (b) high school 058: National Longitudinal Study of Adolescent to Adult Health, United States, 1994–1995.

individual-level peer-influence processes affect the formation of school-level smoking norms, information that may be used in creating intervention strategies simultaneously targeting the diffusion of individual-level smokingrelevant peer influences in friendship networks and school-level smoking norms.

Because networks are dynamic systems that can demonstrate nonlinear and threshold effects, we altered the peer influence and selection parameters over a wide range of values. This strategy allowed us to assess whether the network exhibits possible nonlinearities when selection and influence are set to extremely large values (likely beyond values feasible in actual interventions) or extremely low values (when influence and selection are not even present). We know of no existing studies that measure and quantify how much peer influence strength changes in a network because of an intervention, nor any research that would allow the translation of real-world peerinfluence network-based intervention strategies to the level of change in peer influence or selection effects. It is therefore not possible to translate the consequences of the influence and selection values we explored here to a hypothetical intervention. Our findings do provide insight into the sensitivity of the system to changes in peer influence and selection, and moreover how peer influence functioned in these networks to affect the network and smoking.

It is notable that observed changes in the network were not reliant on extreme values of the strength of peer influence. For instance, in the higher-smoking school, there was an effectively linear inverse relationship between increasing the strength of peer influence and school-level smoking, suggesting that interventions that can increase peer influence to any degree will have an effect on smoking that likely will be aligned with the predominant aggregate school-level smoking norms. Moreover, in the lower-smoking school, the effect was stronger at relatively modest increases in the peer-influence effect. Thus, whereas we observed a sharp change in the number of nonsmokers or heavy smokers in this school when we increased the influence effect 400%, more than half of this change was actually achieved with the first 100% increase of the parameter, implying that even modest

FIGURE 5—Jointly altering selection and influence parameters for ceasing to smoke in (a) high school 077 and (b) high school 058: National Longitudinal Study of Adolescent to Adult Health, United States, 1994–1995.

increases in the strength of peer influence would have notable effects on smoking in such schools.

Past studies provide insight into how to increase peer influence in adolescent peer networks through interventions. One study

delivered an anti-substance use intervention through naturally occurring peer networks to amplify peer influences.⁴⁴ The results were consistent with increases in peer influences, with the intervention increasing risk-protective peer influences among youths with a peer group that did not support substance use, and increased riskpromotive peer influences among youths in peer groups supporting substance use. Another study also delivered an intervention though naturally occurring peer groups, concluding that delinquent peer influences were increased through this peer aggregation.45 Each of these intervention studies used the general network intervention strategy of induction or behavioral diffusion noted by Valente, 46 which in these cases is the diffusion of peer influence amplified by delivery through peer networks.

If one builds upon the notion of behavioral diffusion through peer networks, it is plausible that the strength of peer influences can be increased in other ways. Peer influence is transacted through multiple mechanisms, including verbal persuasion and vicarious learning. The transmission of peer influence is also likely affected by cognitive 47 and affective constructs that affect observational learning, including whether a relationship is perceived as being emotionally supportive. An emotionally supportive bond may heighten the propensity toward similarity in a friendship and therefore provide a strong conduit for influence to occur. One study found that adolescents were more likely to adopt the generalized expectancies of friends when ties were strong.⁴⁸ Thus, future interventions might leverage emotionally supportive, strong adolescent ties to amplify the diffusion of peer influences within adolescent dyads, in addition to bridging ties⁴⁹ that span networks, to simultaneously effect change in the strength and transmission of peer influences at both an individual and network level.

Our findings also suggest that peer influence and selection may work differently in schools on the basis of the school smoking levels, which may reflect school smoking norms. Altering peer influence and selection across schools with different smoking norms may have varied consequences for peer influence-based antismoking interventions targeting peer networks. In

schools where smoking is not normative, interventions might leverage the salutary socialization effects of peer influences likely exerted by the nonsmoking majority by promoting diffusion of antismoking peer influences through adolescent friendship networks. In schools where smoking and other reinforcing drug use is relatively normative, peer influences would likely favor smoking and would need to be dampened while antismoking peer influences would need to be strengthened and diffused through peer networks. Future studies should examine the effects of altering peer influence and selection across a more heterogeneous and larger sample of schools representing a wide range of smoking levels, to understand changes in the network and smoking.

Our findings indicate that interventions targeting adolescent smoking via peer networks should consider the degree of clustering in a school-based network. Specifically, studies should examine whether schools have networks clustered by smoking status. This information can be taken into account to inform which messages to transmit in prosmoking versus antismoking clusters, and moreover how to strengthen existing antismoking norms in antismoking clusters, and dampen prosmoking norms in prosmoking clusters. Moreover, if the networks are clustered as such, the critical connectors or "bridge" adolescents⁴⁹ who occupy a position that links otherwise disconnected cliques in a network may be identified as key targets for diffusing antismoking peer influences to cliques they connect.

In general, a simulation strategy such as the one used in this study can explore the implications of altering network processes for adolescent smoking and other behaviors in a contained social system such as a high school, to inform interventions. For example, future work might explore whether the level of clustering in a network based on homophily along characteristics such as gender or race moderates the patterns observed in the current study. Simulation studies can also examine whether the smoking behavior of adolescents' parents moderates these patterns for peer influence and selection. It may be that the structural position in the network of children of smokers may have important implications that can be explored. Simulation studies

are a low-cost way to assess the possible consequences of an intervention before implementation.

Limitations

There are limitations in this study. First, we performed our simulation manipulations on 2 schools. It is unclear whether our results would generalize to schools of different sizes, with different levels of smoking behavior, or with different levels of clustering in the networks. Second, there could be heterogeneity in the peer influence and selection effects across adolescents. That is, the effect of peer influence may be systematically related to certain characteristics of adolescents. If so, this would need to be accounted for in the model. Also of note, the name generator item was truncated at naming up to 5 female and 5 male friends. Therefore, it is not clear how our findings would have been different had the adolescents been able to nominate an unlimited number of friends.

Our self-reported smoking measure is likely subject to self-report biases including social desirability and recall biases. We did not have access to a biological measure of smoking to validate youths' self-reported smoking behavior. Because the Add Health study only follows these adolescents for 3 time points over a 1.5-year period, we were only able to observe the coevolution of the network and adolescent smoking over this time. A longer time window would have provided more evidence of the evolution of the processes under study. In addition, the smoking measure at wave 1 differed from that at waves 2 and 3, which may provide some minor bias in our results. Finally, these data are 20 years old. However, we are not aware of evidence that the dynamic between smoking and peer influence has evolved since then.

Conclusions

Our experimental manipulations and simulations using a stochastic actor-based modeling approach explored the dual effects of peer influence and selection on adolescent smoking. Our simulations indicated that peer influence appears to have a stronger effect on schoollevel smoking than selection effects. Thus, whereas selection effects may be important for bringing about more similarity in smoking among groups of adolescents, we did not find

evidence that it brings about higher overall levels of smoking in the schools under study. We instead found that stronger influence effects actually lead to lower overall levels of smoking among adolescents in these schools. This study is a stepping stone for future research to begin to understand the sensitivity of school network systems to varying peer influence and selection effects, and to further consider how to translate our disparate findings at an individual and aggregate level to inform school-based peer-network interventions. \blacksquare

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Contributors

C. M. Lakon led the conceptualization of the study, measurement, interpretation of results, and the writing of this article. J. R. Hipp directed all data analysis tasks and led the writing of the Methods and Results sections and contributed to the writing of the entire article. C. Wang conducted the analyses, conducted several data management tasks, and aided in writing the Methods and Results sections. C. T. Butts consulted on the statistical analyses and aided in study conceptualization. R. Jose conducted several data management tasks and edited article drafts.

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This study was reviewed and granted approval under exempt review by the institutional review board at the University of California, Irvine.

References

1. Orlando M, Tucker JS, Ellickson PL, Klein DJ. Developmental trajectories of cigarette smoking and their correlates from early adolescence to young adulthood. J Consult Clin Psychol. 2004;72(3):400-410.

2. Giovino GA, Henningfield JE, Tomar SL, Escobedo LG, Slade J. Epidemiology of tobacco use and dependence. Epidemiol Rev. 1995;17(1):48-65.

Brechwald WA, Prinstein M. Beyond homophily: a decade of advances in understanding peer influence processes. *J Res Adolesc.* 2011;21(1):166-179.

4. Dishion TJ, Tipscord JM. Peer contagion in child and adolescent social and emotional development. Annu Rev Psychol. 2011;62:189-214.

5. Hirschi T. Causes of Delinquency. Berkley, CA: University of California Press; 1969.

6. Nye FI. Family Relationships and Delinquent Behavior. New York, NY: John Wiley; 1958.

7. Kobus K. Peers and adolescent smoking. Addiction. 2003;98(suppl 1):37-55.

8. Hoffman BR, Monge PR, Chou C-P, Valente TW. Perceived peer influence and peer selection on adolescent smoking. Addict Behav. 2007;32(8):1546-1554.

9. Christakis NA, Fowler JH. The collective dynamics of smoking in a large social network. N Engl J Med. 2008;358(21):2249-2258.

10. Adamczyk-Robinette SL, Fletcher AC, Wright K. Understanding the authoritative parenting-early adolescent tobacco use link: the mediating role of peer tobacco use. J Youth Adolesc. 2002;31(4): $311 - 318.$

11. Prinstein MJ, Boergers J, Spirito A. Adolescents' and their friends' health-risk behavior: factors that alter or add to peer influence. J Pediatr Psychol. 2001;26(5): 287-298.

12. Lazarsfeld PF, Merton RK. Friendship as a social process: a substantive and methodological analysis. In: Berger M, Abel T, Page CH, eds. Freedom and Control in Modern Society New York. New York, NY: Van Nostrand; 1954:18-66.

13. Kandel DB. Similarity in real-life adolescent friendship pairs. *J Pers Soc Psychol.* $1978;36(3):306-312$.

14. Kirke DM. Chain reactions in adolescents' cigarette, alcohol and drug use: similarity through peer influence or the patterning of ties in peer networks? Soc Networks. 2004;26(1):3-28.

15. Manski CF. Measuring expectations. Econometrica. 2004;72(5):1329-1376.

16. Veenstra R, Dijkstra JK, Steglich CE, Van Zalk MHW. Network-behavior dynamics. J Res Adolesc. 2013; 23(3):399-412.

17. Mercken L, Candel M, Willems P, De Vries H. Disentangling social selection and social influence effects on adolescent smoking: the importance of reciprocity in friendships. Addiction. 2007;102(9):1483-1492.

18. Mercken L, Steglich C, Sinclair P, Holliday J, Moore L. A longitudinal social network analysis of peer influence, peer selection, and smoking behavior among adolescents in British schools. Health Psychol. 2012; $31(4):450-459.$

19. Schaefer DR, Haas SA, Bishop N. A dynamic model of US adolescents' smoking and friendship networks. Am J Public Health. 2012;102(6):e12-e18.

20. Snijders TAB, van de Bunt GG, Steglich CE. Introduction to stochastic actor-based models for network dynamics. Soc Networks. 2010:32:44-60.

21. Schaefer DR, Adams J, Haas SA. Social networks and smoking: exploring the effects of peer influence and smoker popularity through simulations. Health Educ Behav. 2013;40(1 suppl):24S-32S.

22. Harris KM, Halpern CT, Whitsel E, et al. The National Longitudinal Study of Adolescent Health: research design. 2009. Available at: [http://www.cpc.unc.](http://www.cpc.unc.edu/projects/addhealth/design) [edu/projects/addhealth/design.](http://www.cpc.unc.edu/projects/addhealth/design) Accessed June 18, 2015.

23. Gile KJ, Handcock MS. Model-based assessment of the impact of missing data on inference for networks. Seattle, WA: Center for Statistics and the Social Sciences; 2006:18. CSSS working paper 66.

24. Handcock MS. Missing data for social networks. Seattle, WA: Center for Statistics and the Social Sciences; 2002.

25. Hipp JR, Wang C, Butts CT, Jose R, Lakon CM. Research note: the consequences of different methods for handling missing network data in Stochastic Actor Models. Soc Networks. 2015;41:56-71.

26. Ripley RM, Snijders TAB, Boda Z, Vörös A, Preciado P. Manual for SIENA Version 4.0. Oxford, England: Department of Statistics, Nuffield College, University of Oxford; 2014.

27. Lakon CM, Wang C, Butts CT, Jose R, Timberlake DS, Hipp JR. A dynamic model of adolescent friendship networks, parental influences, and smoking. J Youth Adolesc. 2014; epub ahead of print September 20, 2014.

28. Steglich C, Snijders TAB, Pearson M. Dynamic networks and behavior: separating selection from influence. Sociol Methodology. 2010;40:329-393.

29. Burk WJ, Steglich CEG, Snijders TAB. Beyond dyadic interdependence: actor-oriented models for co-evolving social networks and individual behaviors. Int J Behav Dev. 2007;31(4):397-404.

30. Alexander C, Piazza M, Mekos D, Valente T. Peers, schools, and adolescent cigarette smoking. J Adolesc Health. 2001;29(1):22-30.

31. Radloff LS. The CES-D scale: a self-report depression scale for research in the general population. Appl Psychol Meas. 1977;1(3):385-401.

32. Wills TA, Cleary SD. How are social support effects mediated: a test with parental support and adolescent substance use. *J Pers Soc Psychol.* 1996;71(5):937-952.

33. Simons RM, Chao W, Conger RD, Glen H, Elder J. Quality of parenting as mediator of the effect of childhood defiance on adolescent friendship choices and delinquency: a growth curve analysis. *J Marriage Fam.* 2001;63(1):63-79.

34. Barber BK. Family, personality, and adolescent problem behaviors. J Marriage Fam. 1992;54(1):69-79.

35. Baumrind D. The influence of parenting style on adolescent competence and substance use. J Early Ado $lesc. 1991:11(1):56-95.$

36. Kerr M, Stattin H. What parents know, how they know it, and several forms of adolescent adjustment: further support for a reinterpretation of monitoring. Dev Psychol. 2000;36(3):366-380.

37. Stattin H, Kerr M. Parental monitoring: a reinterpretation. Child Dev. 2000;71(4):1072-1085.

38. Engels RCME, Vitaro F, Den Exter Blokland E, De Kemp R, Scholte RHJ. Influence and selection processes in friendships and adolescent smoking behaviour: the role of parental smoking. *J Adolesc*. 2004;27(5):531-544.

39. Szabo E, White V, Hayman J. Can home smoking restrictions influence adolescents' smoking behaviors if their parents and friends smoke? Addict Behav. 2006; 31(12):2298-2303.

40. Dishion TJ. Stochastic agent-based modeling of influence and selection in adolescence: current status and future directions in understanding the dynamics of peer contagion. J Res Adolesc. 2013;23(3):596-603.

41. DeLay D, Laursen B, Kiuru N, Salmela-Aro K, Nurmi J-E. Selecting and retaining friends on the basis of cigarette smoking similarity. J Res Adolesc. 2013; 23(3):464-473.

42. Mathys C, Burk WJ, Cillessen AHN. Popularity as a moderator of peer selection and socialization of adolescent alcohol, marijuana, and tobacco use. J Res Adolesc. 2013;23(3):513-523.

43. Sussman S, Dent CW, Stacy AW, Craig S. One-year outcomes of project towards no drug abuse. Prev Med. 1998;27(4):632-642.

44. Valente TW, Ritt-Olson A, Stacy A, Unger JB, Okamoto J, Sussman S. Peer acceleration: effects of a social network tailored substance abuse prevention program among high-risk adolescents. Addiction. 2007:102(11):1804-1815.

45. Dishion TJ, McCord J, Poulin F. When interventions harm: peer groups and problem behavior. Am Psychol. 1999;54(9):755-764.

46. Valente TW. Network interventions. Science. 2012;337(6090):49-53.

47. Chaiken S. The heuristic model of persuasion. In: Zanna MP, Olson JM, Herman CP, eds. Social Influence: The Ontario Symposium. New York, NY: Psychology Press; 1987: 3-35.

48. Lakon CM, Hipp JR. On social and cognitive influences: relating adolescent networks, generalized expectancies, and adolescent smoking. PLoS ONE. 2014; 9(12):e115668.

49. Valente TW, Fujimoto K. Bridging: locating critical connectors in a network. Soc Networks. 2010;32(3): 212-220