

With a Little Help from My Friends? Asymmetrical Social Influence on Adolescent Smoking Initiation and Cessation¹

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Abstract

The current study examines the impact of asymmetrical peer influence on adolescent smoking behavior while accounting for endogenous peer selection. Using data from National Longitudinal Study of Adolescent Health we implement a stochastic actor-based model of the coevolution of smoking and social networks and estimate differential peer influence effects on increases and decreases in smoking. We find that among adolescents the impact of peer influence is much stronger for the initiation of smoking than on cessation. We find that to the extent that adolescents exert a significant influence on the smoking behavior of their peers they do so primarily to increase levels of smoking.

The notion that individual health and well-being is determined, at least in part, by connections to other social actors, and thus their integration within larger social structures, has been a bedrock idea in the social sciences since at least Durkheim (1897). Accordingly, a central line of health research has been concerned with uncovering exactly how the quantity, quality, and content of social network ties impact health and its psychosocial and behavioral antecedents (Berkman and Glass 2000). One key contribution has been the investigation of the complex role of peer social influence on the development of health related behaviors such as smoking, alcohol, and substance use. The current study expands on that work by taking advantage of recent advances in dynamic social network models to develop and test theoretical predictions of asymmetrical peer influence on adolescent smoking behavior.

BACKGROUND

Despite recent declines, smoking remains a leading threat to population health, ranking amongst the single largest sources of premature and preventable morbidity and mortality (CDC 2008). In the years 2000-2004, smoking and passive exposure to cigarette smoke resulted in more than 400,000 deaths and 5 million lost life-years (CDC 2008). Smoking also accounts for nearly \$100 billion in lost productivity annually (CDC 2008). Adolescence continues to be a key point of experimentation and initiation of smoking (Duncan et al. 1995) with more than one in five Americans becoming smokers by ages 18-24 (CDC 2008) and 80% of smokers having their first cigarette before age 18 (Kessler et al. 1997). Therefore, from a policy perspective understanding and preventing adolescent smoking remains a key public health goal.

In addition, uncovering the complex role of peers in adolescent smoking has significant sociological value in that it illuminates a fundamental social process—the dynamic micro-level social systems that structure human behavior.

Social Influence Processes

The past four decades has seen numerous studies documenting and attempting to explicate the role of peers in adolescent smoking (Levitt and Edwards 1970; Huba and Bentler 1980; Flay et al. 1994; Ali and Dwyer 2009; Lakon, Hipp, and Timberlake 2010). Having friends who smoke increases the chances that an adolescent will smoke (Ennett and Bauman 1994; Kobus 2003;) and may reduce odds of quitting (Jones, Schroeder, and Moolchan 2004; Zhu et al. 1999). Social network analysis has further demonstrated that adolescent’s smoking behavior increasingly comes to resemble that of their peers over time (Hall and Valente 2007). The development of dynamic actor-based social network models (Snijders 2001; Steglich, Snijders, and Pearson 2010) has buoyed those efforts, facilitating important new insights into the complex social network processes surrounding adolescent smoking. Greater understanding of social influence processes has also led to the development of anti-smoking interventions grounded in exploiting social network dynamics (Valente et al. 2003; Campbell et al. 2008).

Despite these advances, significant unresolved issues remain. These issues stem both from how peer influence has been conceptualized as well as how it has been operationalized by analytic techniques designed to model social influence. An important limitation of prior research on peer influence is that it has focused almost exclusively on the role of peers on current smoking behavior and the extent to

which it corresponds to those with whom that have social ties. To the extent that prior work has examined the effect of social influence on adolescent smoking longitudinally, it has tended to focus on the initiation and uptake of smoking behavior. This obscures the fact that for many adolescents smoking represents an experimental or transitory behavior, which may or may not become an established habit (Mayhew, Flay and Mott 2000; Wang et al. 1996; USDHHS 1994; Levanthal and Cleary 1980). While understanding why adolescents may initially begin to use cigarettes remains important, less is known about what leads many of the adolescents who experiment with smoking to reduce or quit, and what role peers play in that process. This is due in part to the tendency to conceptualize social influence as unidirectional—in which negative influences entice individuals to engage in unhealthy or antisocial behaviors while positive influences work to prevent their initiation or to promote pro-social behaviors.

Another important issue is that the analytic methods commonly used to model social influence, have until now, assumed symmetric effects of peers. No study has simultaneously examined the impact of social influence on smoking initiation and cessation and how the magnitude of those effects may vary. We are also not aware of any study that has examined the role of peer influence on adolescent smoking cessation from a social network perspective. Thus, a conceptualization that views social influence as unidirectional and symmetrical and analytic strategies that operationalize these assumptions into models obscures the complex social milieu that exists around adolescent smoking behavior.

Among adults, there is a literature that has examined the effect of social support on smoking cessation attempts (Park, Tudiver, Schultz, and Campbell 2004; Westmaas, Bontemps-Jones, and Bauer 2010). However, scant research has examined smoking cessation explicitly within a social network framework (Cobb, Graham, and Abrams 2010) and we are only aware of one study that has directly looked at social influence processes on cessation. In their widely-cited yet controversial work Christakis and Fowler (2008) argued for a *social contagion* effect by which smoking cessation spreads through a social network. Under contagion theory, peers in one's network are hypothesized to serve as conduits of information about the benefits of quitting and methods to do so and to provide various forms of social support during cessation attempts. Peers are also thought to be influential in cessation by providing a collective normative structure around behavior, either exerting pressure to quit or undermining attempts.

A major limitation of previous work on peer influence on smoking cessation is that few studies have utilized a social network analytic framework or have explicitly addressed the critical issue of endogenous network selection. To the extent that smoking behavior acts as a criterion by which actors form, maintain, and dissolve ties then traditional estimates of the extent of social influence become inflated. Thus, increasing network clustering in smoking behavior that traditional studies would attribute to the effects of social influence may alternatively result from selective attrition of smoking discordant ties. The contagion work of Christakis and Fowler (2008) employed a network analytic framework and attempted to address the issue of selection by comparing the impact of social ties of various types,

and social and geographic distances. However, critics have charged that their Generalized Estimating Equation (GEE) approach did not adequately address the issue of shared environmental context among peers or endogenous friendship selection (Cohen-Cole and Fletcher 2008; Halliday and Kwak 2009). This critique is not unique to contagion theory. Identification of a true causal effect of social influence distinct from larger contextual effects has long been an issue of concern in social network research (Manski 1993). Prior work confirms the importance of endogenous selection processes in generating behavioral homophily on smoking within networks (Mercken, Candel, Willems, and de Vries 2007; Mercken, Snijders, Steglich, and de Vries 2009; Schaefer et al. 2012). Even moderate levels of selective attrition in peer networks can lead to substantially inflated estimates of social influence (Noel and Nyhan 2011).

Additionally, it is unclear if the type of contagion effects on cessation hypothesized by Christakis and Fowler would be expected to exist to the same extent among adolescents. There are reasons to suspect that the impact of peers on smoking cessation may be as large or even larger in adolescence than in adulthood. First, adolescence is the stage of the life course where peers first emerge as an important source of influence (Brown 1990; Beal, Ausiello, and Perrin 2001; Collins and Laursen 2004). The impact of the parental family begins to wane (Larson et al. 1996) and the influence of adult marital family has yet to emerge. Thus, in a number of ways, adolescence is a period of development when peers may be expected to exert their greatest impact. Second, smoking and other health behaviors first emerge in adolescence and are thus less well established (Chassin et al. 1990).

Adolescence is a time of frequent experimentation with tastes, preferences, and behaviors. In this early milieu of experimentation, smoking behavior may therefore be more malleable in the face of peer influence. Conversely, in adulthood, patterns of smoking behavior may be thoroughly entrenched over the course of years or decades and may therefore be much more resilient to change. Finally, ties to peers themselves are less secure in adolescence as there is relatively high turnover in network membership (Cairns et al. 1995). This greater fragility in peer ties and the risk of social isolation may induce greater conformity with group norms and behaviors. Thus, adolescents may be more inclined to shift their behavior to coincide with that of their peers rather than attempt to shift peers to coincide with behavior, thus magnifying the role of social influence.

Alternatively, there is reason to suspect peers may be less influential in adolescence than they are later on in adulthood. The greater fluidity in social network ties also may cut the other way as adolescents may find it either easier or preferable to shift their limited social resources towards establishing and strengthening those friendship ties that are supportive of their behavioral choices and away from those that are not. In adulthood, peer networks have already been subject to years if not decades of sifting and winnowing such that only the strongest and arguably most influential ties may remain.

Asymmetrical Peer Influence

Given that we expect peer influence on cessation to exist among adolescents, our driving question is how does it differ in magnitude from peer influence on smoking initiation? Prior network research examining peer effects on smoking and

other behaviors has implicitly assumed symmetrical peer influence effects (Mundt, Mercken and Zakletskaia, 2012; Mercken et al. 2009; Schaefer et al. 2012). We are not aware of any research that has investigated the differential role of peer influence on smoking initiation and cessation. Yet there are reasons to expect asymmetrical effects of peer influence on the initiation and cessation of smoking behavior.

One potential source asymmetry involves peers ability to differentially provide resources towards smoking initiation and cessation. There are numerous barriers to smoking initiation that must be overcome. Non-smokers must learn how and where to obtain cigarettes, where they can smoke without being discovered by authorities, how to conceal evidence of their smoking behavior, and most importantly, how to smoke. These barriers to smoking initiation can be overcome when adolescents have friends who smoke and can share their experiences. By contrast, the types of information and other resources required for successful smoking cessation (knowledge of effective methods; access to smoking cessation programs or to nicotine replacement products) are very different. An adolescent's peers are not likely to be good sources for such resources. Research on adolescent quitting motivations and cessation methods has shown that the majority of adolescents smokers indicate desire and intention to quit, have attempted to quit at least once and report social pressure from peers as an important motivator (Burt and Peterson 1998). However, the most commonly used methods in cessation attempts are quitting "cold turkey" and gradual reduction with only a very small minority employing nicotine replacement or a formal cessation program (Myers and

MacPherson 2004). As a result success rates for smoking cessation among adolescents are quite low (Burt and Peterson 1998).

The psychophysiological effects of nicotine addiction may be a third source of asymmetry. The psychological and neurophysiologic effects of smoking are well characterized (Mansvelder and McGehee 2002; Benowitz 2010). There is evidence that the initial psychological and somatic symptoms of nicotine addiction appear within days or weeks of occasional use and often predate the onset of regular daily smoking (DiFranza et al. 2000). Thus, attempts to push friends to quit smoking may run head long into the psychophysiological effects of nicotine addiction, limiting the impact of peer influence on cessation (Chassin et al. 1984). There are no such psychophysiological constraints on smoking initiation.

Finally, social influence to conform to peer behavior and norms occurs within the context of broader societal norms that dictate appropriate behavior. Therefore, asymmetry may arise from the interaction between peer social influence and the larger school, neighborhood, and family normative context (Elickson et al. 2003; Simons-Morton and Farhat 2010). Injunctive norms—dictating what one “ought to do”—proscribe that adolescents should not smoke. To smoke, an adolescent must violate this norm. Such violation becomes more likely when there are group or peer norms in support of smoking. Thus, peer influence becomes a route to smoking—smoking that is unlikely to occur if both peer and societal norms discourage it (i.e., peer smoking acts as a necessary, but not sufficient, condition for initiation). By contrast, when adolescents smoke, they are already in violation of societal norms. Quitting smoking, even when friends smoke, represents a shift to violating peer

norms but conforming to societal norms. Thus, change in smoking behavior may occur without peer influence. Still, when peers do not smoke, then adolescent smokers are in violation of both peer and societal norms, which should push them to cease smoking. (In this way, peer non-smoking acts as a sufficient, but not necessary, condition for cessation).

The Present Study

The degree to which there is asymmetrical peer influence on smoking among adolescents thus represents an important and unexplored question, which has both significant sociological and public policy implications. To explore this issue we utilize a stochastic actor-based model (SAB) (Snijders 2001; Steglich, Snijders, and Pearson 2010) to model the co-evolution of peer social networks and smoking behavior. The SAB framework allows for the simultaneous modeling of friend selection and behavioral assimilation (social influence) and the decomposition of the autocorrelation in behavior among friends due to those two processes. Therefore this approach allows us to test for the asymmetrical impact of peer influence on the uptake of smoking (increases over time) and cessation (declines over time) among adolescents, while also accounting for endogenous friend selection.

METHODS

Data

The analysis below utilizes the wave 1 in-school, wave 2 in-home, and wave 3 in-home interviews from the National Longitudinal Study of Adolescent Health (Add Health). Add Health used a stratified sampling design to identify 132 middle and

high schools in the US. All students in these schools were targeted for an in-school survey at wave 1. Subsequently, 16 “saturated” schools were chosen in which all students were targeted for more in-depth, in-home surveys (8 and 11 month intervals between waves). At all three waves, students provided self-report measures of their smoking behavior and friendships with other students (i.e., networks). Our sample includes high school students from two of the saturated schools.¹ The two schools we focus on are school 58 which frequently takes the pseudonym “Jefferson High School” and school 77 commonly referred to as “Sunshine High School”. Jefferson and Sunshine are the largest schools with longitudinal network data, and thus, have the greatest statistical power to detect peer influence on smoking initiation distinct from smoking cessation. Because of their size these two schools are frequently singled out in social network analyses using Add Health. These schools also represent very different social contexts. Jefferson (N=757) is almost all white and is located in a mid-sized town in the Midwest while Sunshine (N=1,673) is racially and ethnically diverse and is located in suburban community in the West.

Our analysis includes all students in the two schools who provided data during at least one survey wave. Many students necessarily left the school after wave 1 (e.g., graduating seniors). Students who left the school are coded as such, which omitted them from the analysis of subsequent time points. Students who remained in the school but did not complete parts of the survey are retained and coded as missing. Standard SAB model missing data imputation techniques are used in those cases (Ripley, Snijders and Preciado 2012; Huisman and Steglich, 2008).

Measurement

Smoking at wave 1 was measured by asking students how often they had smoked in the past 12 months, which was recoded into: 0="never"; 1="twice a week or less"; 2="3-5 days a week or more". At waves 2 and 3, *smoking* was measured as the number of days students had smoked in the past 30 days. To create as much comparability across waves as possible these items were recoded into:

0=none/never; 1=1-11; 3=12-30. The ordinal scaling of the smoking measure is a requirement of the SAB model (Ripley et al. 2012).

Friendship networks were created by asking students to nominate up to 5 male and 5 female friends. Nominations to friends outside of the school were omitted because those friends were not included in the Add Health data and therefore do not have information on smoking or other attributes. Thus, the data represent student's school-based friendship networks. For a small number of respondents (less than 5% in each school) a programming error restricted their friend nominations to 1 female and 1 male friend. We control for this by creating a variable representing which questionnaire version the respondent received: 0=correct questionnaire; 1=truncated questionnaire.

We include a number of controls for other factors that are related to smoking and salient to the friend selection process. *Gender* is coded is 1=male; 0=female. *Age* is measured in years at the wave 1. The model includes a parameter of race-ethnic similarity using the following mutually exclusive categories: non-Hispanic white; non-Hispanic black; Hispanic; Asian; other. *Grade point average* was calculated at each wave based on students reported grades in English/Language Arts,

Mathematics, History/Social Studies, and Science. *Parental education* captures the level of education of the most highly educated parent. *Alcohol use* was measured at all three waves points by asking students how often they used alcohol in the past 12 months. Responses were coded: 0=never; 1=twice a week or less; 2= 3-5 days a week or more. We also include *Self-control* scale constructed from 14 items asked during the first in-home interview ($\alpha=.64$; Beaver et al. 2009). For scaling purposes, we divided self-control by 100. In predicting adolescent's smoking we also control for *parental smoking*: 1=any parent smokes; 0=no parent smokes. Finally, we account for the effects of propinquity by controlling for whether adolescents shared mutual contexts within the school that can promote friendships and may be related to smoking. For each pair of students we calculated how many (out of 30) *extracurricular activities* they reported in common during the in-school survey. We also control for *course overlap* for each dyad. This is calculated as the number of courses in common during the school-year corresponding to the survey wave.

Statistical Analysis

The co-evolution of adolescent social networks and smoking behavior was assessed with a Stochastic Actor-Based (SAB) model using the Simulation Investigation for Empirical Network Analysis (SIENA) package in the R statistical program (Ripley and Snijders 2010). This dynamic social network approach is particularly well-suited to the questions raised here because it simultaneously models change in smoking, such as due to peer influence, and change in the friendship network. Thus, our model addresses the issue of endogeneity in peer influence by explicitly modeling its form. The SAB approach has been used

previously to study peer influence on obesity (Shoham et al. 2012), alcohol use (Mundt, Mercken and Zakletskaia, 2012), and smoking (Mercken et al. 2009; Schaefer et al. 2012).

The SAB approach has at its core the assumption that individuals have control over their outgoing ties and their behavior. The model is constructed such that over time, actors are given multiple iterative opportunities to evaluate their current ties and behavior as well as prospective changes in relations (by adding or dropping a tie) and behavior (either increasing or decreasing its level), which serve as the basis for ensuing changes. The SAB model thus simultaneously optimizes two objective functions, a behavior (e.g. smoking) function and a friend selection function. The behavior function predicts changes in smoking over time. Predictors can include one's own attributes as well as the attributes of one's friends. We are most interested in the effect of friends' smoking on ego's own smoking. To capture the tendency for adolescents to adopt a level of smoking that brings them closer to the smoking level of their friends we use a measure of total similarity. *Total similarity* is defined by the sum of the similarity scores sim_{ij}^z between individual i and the peers j to whom they are tied (sim_{ij}^z), which is centered using the average similarity across all possible dyads ($\overline{sim^z}$).

$$s_i^{beh}(x, z) = \sum_j x_{ij} (sim_{ij}^z - \overline{sim^z})$$

Similarity itself is measured as the absolute difference in scores between i and j divided by the observed range of scores, and then subtracted from the observed

range (ensuring that similarity ranges from 0 to 1, with larger values indicating i and j have more similar scores).

Unless otherwise specified, the SAB model assumes symmetrical effects (i.e. processes operate the same for increases vs. decreases in smoking behavior). Indeed, prior studies of smoking and other behaviors have implicitly assumed that peer influence operates the same for increases and decreases in the level of the behavior. However, SAB models can distinguish the effect of friends on smoking increases from their effect on smoking decreases by relaxing this constraint.

To answer our question about the differential effect of peer influence on smoking increases versus decreases, we add an extra “endowment” effect to the model—*total similarity (decrease)*. Whereas the main “evaluation” effect of total similarity is evaluated for every choice of behavior (increase, decrease, or remaining the same), the added term is only evaluated when considering decreases in smoking. Specifically, the added term estimates how much the effect of peers on smoking decreases *deviates* from the effect of peers on smoking increases. If friends exert the same influence on smoking escalation versus cessation then the added influence parameter will equal zero; however, if friends are more important for behavior change in one direction than the added parameter will depart from zero. Our hypothesis is that friends will be more influential for smoking increases than decrease, which would be indicated by a positive main effect of total similarity and a negative effect for total similarity on decreases.

Change in the friendship network is modeled using a network function. The network function models the likelihood of one adolescent (ego) nominating another

adolescent (alter) as a friend. Thus, the outcome for each dyad is whether a tie is present or absent (coded 1 and 0 respectively). Predictors include individual attributes, such as the smoking level of ego or the smoking level of alter. Predictors may also be dyadic, such as the level of similarity in the smoking behavior of ego and alter. We include ego, alter, and similarity controls for smoking and all other individual attributes.

Beyond individual attributes, friendships are shaped by the current structure of the network. Adolescents are more likely to form ties with friends of their current friends (transitivity) or peers who are higher social status (popularity). Importantly, these endogenous network processes must be accounted for when modeling friend selection in order to avoid bias in other estimated effects (Mouw and Entwisle 2006). Accordingly, we include a number of network effects to control for reciprocity, transitivity, and selection related to popularity and sociability.

Estimation of the SAB model precedes iteratively following a continuous-time Markov-chain process where the trajectories between observation points are imputed and changes to the network are assumed to be dependent only on the current state of the network. A series of micro-steps provide each actor opportunities to make changes to their outgoing ties or behavior within the context of constraints and opportunities determined by the structural aspects of the social network and the individual's personal attributes. Snijders and colleagues (2010) give a detailed explanation of the SAB modeling framework and estimation.

We estimate one model for the two schools; however, we treat the schools as distinct networks, thereby prohibiting ties between them. Parameters are

constrained to be equal across schools with the following exceptions. Each school has separate rate and out-degree parameters in the network function, which allows the rate of change and average number of friends to differ between the schools. In the smoking function, rates of smoking change and baseline tendencies toward smoking are free to differ between schools. As a final step, we test for heterogeneity in effects across schools and add appropriate controls when needed.²

Drawing causal inferences from the SAB model requires two additional assumptions. The first is that the peer selection and behavior change models are properly specified. That is, there are no unobserved factors that drive change in both networks and behavior conditional on the initial pattern of smoking and friendship ties. Second, the model assumes temporal separability in which the total observed change in smoking and networks can be broken down into a series of smaller unobserved changes. Under these assumptions it is possible to establish Granger-type causality (Granger 1969).

RESULTS

Table 1 presents the characteristics of the respondents in each school. Jefferson had higher levels of smoking on average both among students themselves as well as their parents. Jefferson also has a somewhat higher level of network autocorrelation (e.g., Moran's I), indicating that friends are more similar to one another than chance in their level of smoking. Sunshine is more racially heterogeneous and has lower prevalence of alcohol use and extra-curricular activity participation.

The results of the SAB model reveal several interesting aspects of friend selection dynamics. While full estimates for the network function are presented in Appendix table A1, Fig. 1 summarizes the parameter estimates for effects related to individual attributes. Students were more likely to select friends who were similar to themselves in terms of sex, race, age, GPA, alcohol use, and self-control.³ Beyond that, students were more likely to be selected as a friend (i.e. more popular) if they used alcohol more often. In addition, students who were younger or had higher self-control named more friends on average.

Turning to smoking in particular, Fig. 1 reveals that higher levels of smoking led to naming more friends (smoking ego) and receiving more friendship nominations (smoking alter). School-level interactions indicate that both of these effects were stronger in Sunshine than in Jefferson. Adolescents were also more likely to befriend peers whose level of smoking was similar to their own (the positive similarity effect). This suggests that friend selection is part of the reason the network is clustered based on smoking.

Beyond individual attributes, features of the school environment promoted some friendship ties over others. Students were more likely to be friends if they participated in the same extracurricular activities or took the same courses. Additionally, several common endogenous network processes helped structure the network. Students tended to select friends who selected them (reciprocity) and friends of their friends (transitivity). More popular students tended to name more friends (in-degree – activity) and receive more friendship nominations in the future

(in-degree – popularity). Finally, students who previously named more friends were more likely to name additional friends in the future (out-degree – activity).

Turning to the smoking function (full estimates provided in Appendix table A2), we find that several background factors affect smoking (Fig. 2). Smoking was greater for students with more frequent alcohol use and for students with a parent who smoked while smoking levels were lower on average for older students. Participating in extracurricular activities did not affect smoking behavior. The linear and quadratic effects represent the distribution of smoking likelihood across the three levels. The significant quadratic term indicates that tendencies to adopt each level of smoking are non-linear. Interpretation of these effects is facilitated by evaluating the smoking function for each prospective smoking level. As shown in Figure 3, the distribution of smoking is U-shaped. Adolescents have a higher likelihood of adopting smoking levels 0 and 2 relative to level 1. These predicted likelihoods differ by school, with students at Jefferson displaying a greater likelihood of smoking at either level than students at Sunshine.

Finally, we turn to the effects of friends on smoking behavior. The smoking similarity evaluation effect (*total similarity*) represents the effects of friends' smoking on one's own smoking. The significant positive parameter indicates that students are likely to change their level of smoking if it brings them closer to their friends' level of smoking. The endowment effect for smoking similarity (*total similarity decrease*) tests how the effect of friends differs for decreases in smoking versus increases (i.e., deviation from the effect of friends on smoking increase). The endowment parameter is negative and statistically significant suggesting that peers

have less of an influence on smoking decreases than they do on smoking increases. The magnitude of friends' influence on decreases is calculated by summing the evaluation and endowment parameters. This summation reveals that the endowment effect more than balances out the evaluation effect ($-.261 = 1.191 - 1.452$). Thus, friends do not influence one another to decrease their smoking to the same extent that they influence one another to increase smoking.

Although the endowment effect offsets the evaluation effect, this does not imply that peer influence on cessation is absent (only that the effect is weaker for cessation than for initiation). To evaluate the net peer influence effects for initiation and cessation, we must account for the distribution of smoking and similarity in the sample as reflected in the centering of scores. Comparing cessation to initiation is accomplished by evaluating the smoking function under multiple scenarios that manipulate 1) ego's current smoking level, 2) friends' current smoking level, and 3) ego's prospective smoking level. For these calculations, we hold the number of friends constant at their observed means in each school and assume that all other predictors are at their observed means. Fig. 4 details how the total similarity effects representing peer influence affect the likelihood of adopting a prospective smoking level across the 9 combinations of ego's current smoking and friends' current smoking.

To begin, consider the case of a non-smoking adolescent (Panels A, D, and G). For non-smokers, the level of smoking with the greatest likelihood is the level that matches one's friends smoking. Non-smokers with non-smoking friends remain non-smokers (panel A). When one's friends smoke (panels D and G), then non-

smoking adolescents are likely to increase their smoking level to match their friends' level. This pattern is present in both schools, but stronger in Jefferson than in Sunshine.

When adolescents are intermittent smokers (level 1), there is still a tendency to match friends' smoking behavior, but it is not as strong as the tendency for non-smokers. Adolescents are most likely to match their friends' level of intermittent smoking (Panel E) and increase their smoking to match heavier smoking peers (Panel H), but do not necessarily decrease their smoking behavior to match non-smoking friends (Panel B). Intermittent smokers are largely indifferent between remaining intermittent smokers and ceasing their smoking behavior.

Adolescents who are presently at the highest smoking level show weaker signs of susceptibility to peer influence. When friends are non-smokers (Panel C) they are likely to reduce smoking, but not necessarily cease altogether. When friends smoke intermittently, then adolescents at the highest smoking level are indifferent between all levels of smoking (Panel F)—they are just as likely to remain the same, decrease smoking, or completely stop. And, when friends also smoke at the highest level (panel I), adolescents are either indifferent between smoking levels (in Sunshine), or only slightly more likely to remain heavier smokers than to decrease or cease smoking.

The asymmetry of peer influence for smoking initiation versus cessation is evident in several aspects of these findings. First, compare the likelihoods for non-smokers (Panels A, D, and G) to the heaviest smokers (Panels C, F, and I). When the decision is whether to initiate smoking or not, non-smokers tend to follow their

friends. However, when the decision is to decrease or cease smoking, smokers are either indifferent about matching their friends (Panel F) or may only partially decrease their smoking (Panel C). Further, when an adolescent and his or her friends are both smoking at the highest level, the likelihood of decreasing smoking is only slightly lower than the likelihood of remaining the same (Panel I), indicating that some smokers cease smoking even when their friends smoke. We do not see such an effect for non-smokers—non-smokers are not inclined to initiate smoking when their friends are non-smokers (Panel A).

As a final step, we present the likelihoods of smoking that take into account the tendencies toward smoking at each level represented in Fig. 3. The likelihoods shown in Fig. 5 are the sum of the likelihoods presented in Figs. 3 and 4. This figure better conveys the probability of adopting each level of behavior. Results are largely the same as discussed earlier with a few notable exceptions. First, there is less indifference between intermittent smoking and the other levels. This is because of the U-shaped distribution of smoking, where adolescents either don't smoke or smoke regularly. Thus, when peer influence leads adolescents to be indifferent between two behaviors, they will tend toward one of the extremes of smoking instead of adopting intermittent smoking. Second, and related, there is a lower likelihood of students in Sunshine becoming intermittent smokers versus non-smokers. Compare Panels C, D, E, and G and note that peer influence alone suggests that students at Sunshine have a greater likelihood of being intermittent smokers versus being non-smokers (Fig. 4). However, when considering the base tendencies to adopt an extreme level of smoking behavior accounted for in Fig. 5 we see that

Sunshine students are more likely to adopt non-smoking in these cases. This suggests that in Sunshine, there is a larger hurdle between non-smoking and intermittent smoking that peer influence alone does not overcome.

DISCUSSION

Research on the social influences to smoking has found strong evidence of peer effects. However, such research has not distinguished peer influence on smoking initiation from smoking cessation. There are several reasons to suspect that among adolescents, peers are more influential for smoking uptake versus cessation. We investigate this using data from two US high-schools and a longitudinal network model that controls for the effects of smoking on friend selection. The results support the hypothesis that peers exert social influence that leads their friends both to increase and decrease their smoking. However, the effects of peer influence are asymmetrical as the tendency for adolescents to follow their friends into smoking is stronger than the tendency to follow friends out of smoking. Thus, for many adolescents the decision to quit smoking often occurs in the absence of strong peer support of their choice.

We articulated several reasons why peer influence on smoking increase should be stronger than for smoking decreases. First, for adolescents, smoking initiation requires information on the logistics of acquiring and using cigarettes. By contrast, the types of information and resources that maximize smoking cessation are not likely to be provided by adolescent's peers. Thus, adolescents are unlikely to initiate smoking without peer assistance, but are likely to attempt to quit smoking without necessarily following their peers. Second, nicotine dependence can restrict

peer influence on cessation but has no effect on initiation via peer influence. Third peer group social norms that support smoking are likely necessary for smoking initiation, but not for smoking cessation, which conforms to larger societal norms prohibiting adolescent smoking. While we found evidence of stronger peer influence on smoking initiation than for cessation, our research design does not allow us to test these factors, nor quantify the relative strength of each. Thus, we can't estimate how much the lack of information is a barrier to initiation, how much nicotine dependence prevents smokers from following the lead of their non-smoking friends, or the alignment between group and societal norms.

A strength of our approach is that we tested for asymmetry in peer influence while simultaneously modeling the effect of smoking on friend selection. This approach provides estimates of peer influence that are unconfounded by homophilous selection processes. Our results confirm prior work that smoking is an important source of network tie creation and maintenance among adolescents and that, on average, smokers are more popular than their non-smoking peers. However, we observe substantial impacts of social influence net of these selection effects.

We observed some differences in smoking-related social influence between the two high-schools. In Jefferson, we observed stronger peer influence, whereby adolescents were more likely to follow their friends into smoking. In Sunshine, however, there was a larger hurdle to overcome to initiate smoking. This raises the question of how contextual effects, such as smoking prevalence within a school, may affect the dynamics we observed (Alexander 2001). Prior work suggests that behaviors are more likely to spread when they are exhibited by popular individuals,

who have more connected through which they can influence others (Osgood et al. 2013; Schaefer, Adams, and Haas 2013). In a context where smokers are more popular and more influential, we may see stronger pressures to initiate smoking versus cease smoking. Moreover, attempts to gain popularity may serve as another impetus to follow peers into smoking, but not into non-smoking. With only two school contexts, it is impossible for us to test for school effects. Further research could use an approach similar to ours to test whether asymmetry in peer influence varies across schools.

While our results are consistent with prior research by Christakis and Fowler (2008) in that we find a significant effect of social influence on cessation, we generally find weaker effects for smoking cessation than initiation among adolescents. While two analyses are not directly comparable, our results suggest that at the very least the role of peers in smoking cessation among adolescence may be less salient than among older adults. Several factors may explain these differences. First, for reasons discussed above, peers may exert differential social influence at different points in the life course. It may be the case that adults are just more important and influential in their friend's lives than high school friends are for theirs. Alternatively, the context around smoking may be qualitatively different in adolescence, which is primarily a time of smoking initiation, than in adulthood, which is primarily a time of cessation. For example, smoking cessation in adulthood is more likely to be motivated by concerns over health (Hymowitz et al. 1997) and even be precipitated by the onset of a serious health condition. Successful cessation in adulthood may also require stronger and more consistent social support than

similar attempts in adolescence when the behavior is less entrenched. There is also the possibility that Christakis and Fowler have overstated the impact of social influence on cessation due to incomplete adjustment for endogenous peer selection. Finally, the Framingham network examined by Christakis and Fowler comprised a wider range of social ties including siblings, friends, spouses, neighbors, and coworkers with heterogeneous degrees of social influence by relationship type. In the Framingham study social ties were also solicited specifically on the criteria that they were likely to exist into the future. A limitation of our analysis was that it was constrained to school-based friendship networks. While prior work on adolescents has investigated the relative importance of parents and peers (Flay et al. 1994), future research on adolescence would be wise to examine heterogeneous social influence across a wider variety of social ties and settings.

From a theoretical perspective, the results suggest that the effects of social influence on health behaviors such as smoking are more complex and multifaceted than previously acknowledged. Future work investigating the impact of social influence on social and behavioral outcomes would be wise to explicitly theorize and model that complexity including the types of asymmetries explored here. In regards to smoking, more work is needed to better understand why peers appear to be much more influential on initiation than on cessation. For example, do the underlying mechanisms through which social influence operates vary depending upon whether adolescents are moving into smoking or away from it? Toward that end the dynamic SAB approach used here significantly increases the ability to model such complexity.

From a policy standpoint the results are consistent with the prescription to focus adolescent intervention efforts on preventing smoking initiation rather than encouraging smoking cessation (Valente et al. 2003; Campbell et al. 2008). However, as our results demonstrate, peers can exert significant influence on cessation as well. This suggests that successful interventions may also target existing adolescent smokers particularly if those interventions can be designed so as to effectively boost the influence of non-smoking adolescents on their smoking network peers.

NOTES

1. Only the 16 saturated schools targeted a large enough proportion of students over time to make longitudinal network analysis possible. Of these, we exclude 6 middle schools, which suffered a high rate of attrition as students transitioned to high school. We also exclude 8 high schools for either low response rate ($n=1$) or high rate of missing network data ($n=7$). The omitted schools are relatively small, averaging 84 students, compared to 757 and 1673 in the two schools we analyzed.
2. We estimate our model using the multigroup option in RSiena (Ripley, Snijders, and Preciado, 2012). To check for heterogeneity across schools, we estimated separate models for each school and used a t test to determine whether parameter estimates differed between schools (Paternoster et al. 1998). Based on this test, we free several effects in the network and smoking functions to vary across schools (see *Appendix A*). Freeing effects to vary across schools is accomplished by including an interaction between the effect and a dummy variable representing the school a student attended.

3. Interaction effects suggest that the effects of sex and race similarity are stronger in School B (see Table A1).

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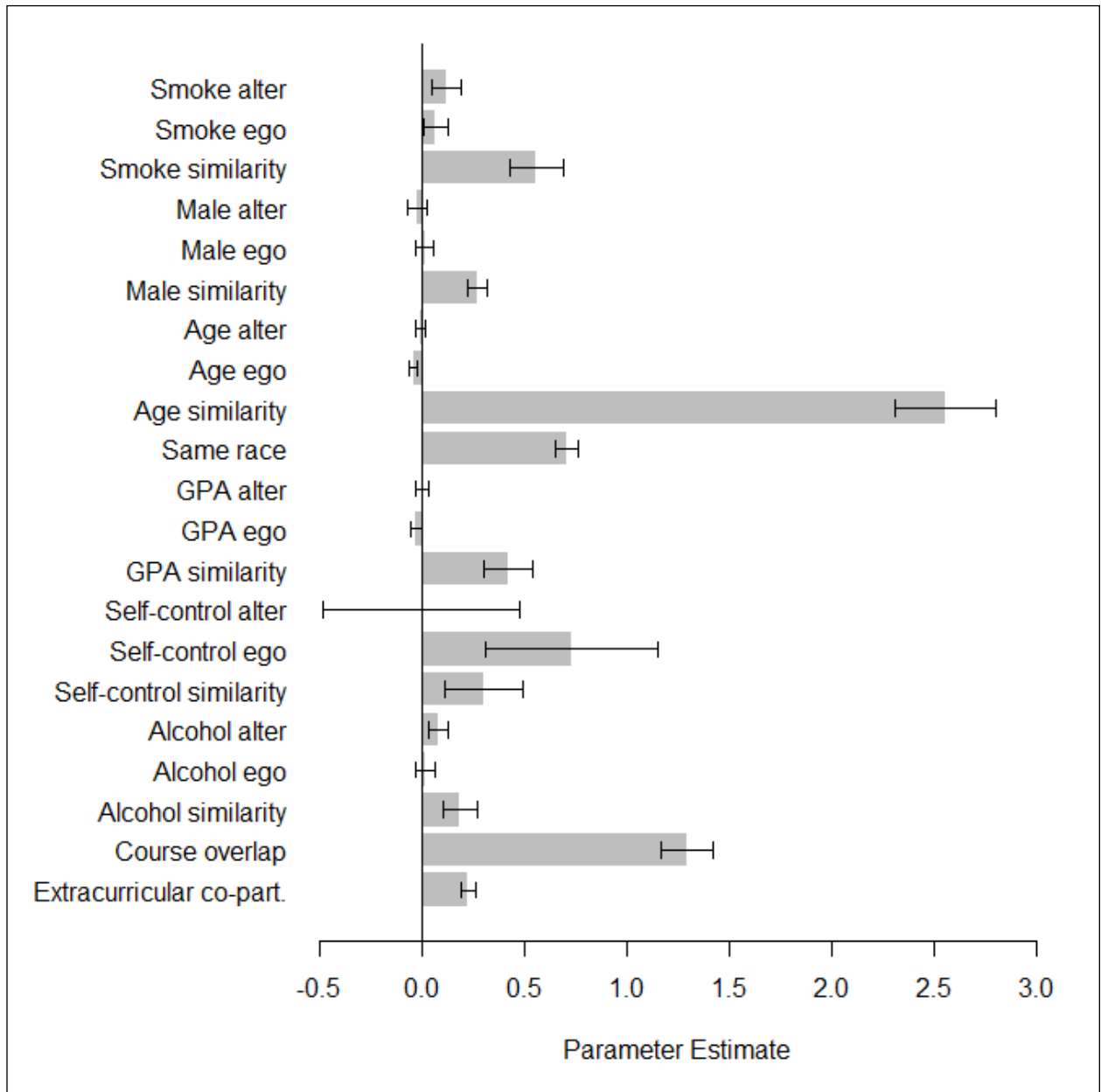
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Table 1. Descriptive statistics by school

School		Jefferson			Sunshine	
Wave	1	2	3	1	2	3
<i>Individual</i>						
Smoking	.81(.82)	.74(.87)	.86(.90)	.38(.62)	.27(.59)	.34(.65)
Increase (N)		61	99		86	117
Decrease (N)		102	59		173	75
Male	51%			52%		
Age	15.73(1.20)			16.09(1.04)		
Race						
White	92%			5%		
Black	4%			22%		
Hispanic	3%			40%		
Asian	1%			32%		
Other	1%			1%		
Alcohol	.73(.52)			.60(.58)		
GPA	2.59(.77)			2.69(.76)		
Self-control	.33(.06)			.33(.05)		
Parent smoke	76%			58%		
Extracurric. Part.	83%			74%		
<i>Network</i>						
M degree	5.24	3.95	3.18	2.74	2.01	1.43
SD outdegree	2.78	2.52	2.46	2.65	1.84	1.60
SD indegree	4.05	2.70	2.34	2.58	1.75	1.43
Jaccard Index		.23	.21		.17	.16
Reciprocity	.27	.24	.23	.24	.17	.19
<i>Network and smoking autocorrelation</i>						
Geary's C	.61	.66	.60	.74	.68	.65
Moran's I	.34	.31	.34	.19	.19	.24
Avg. Similarity	.81	.74	.86	.38	.27	.34
N	757	617	479	1673	1249	850

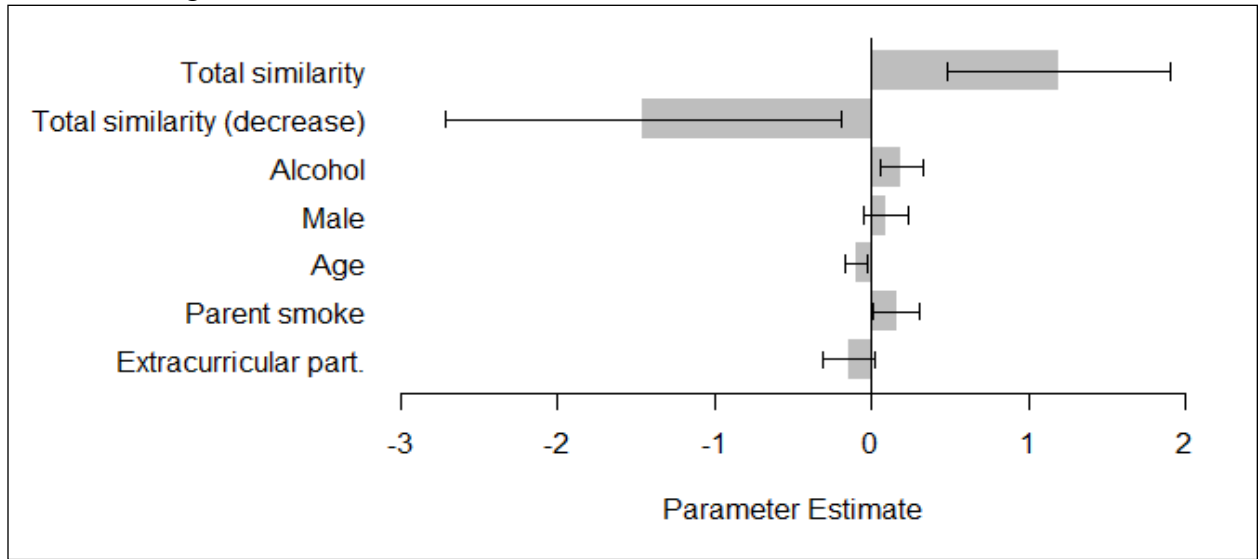
Note: Mean indegree (number of incoming ties) equals mean outdegree (number of outgoing ties) by definition. Geary's C ranges from 0 to 2, with lower values indicating stronger positive correlation between friends' smoking. Moran's I ranges from -1 to +1, with higher values indicating stronger positive correlation between friends' smoking. For both measures, the midpoint of the range indicates no correlation between friends' smoking.

Fig 1. Parameter estimates and 95% confidence intervals for individual factors in SAB model friend selection function



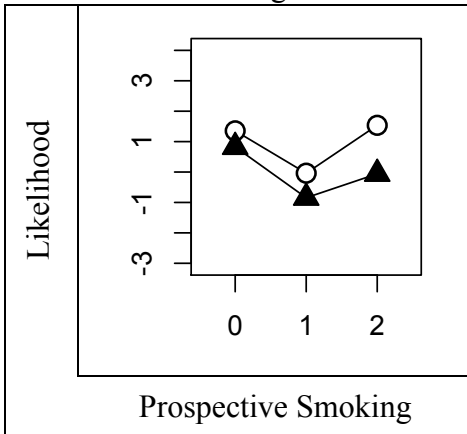
Note: Positive parameter estimates indicate that a friendship tie is more likely at higher levels of the predictor. Alter refers to the recipient of a friendship tie; ego refers to the sender of a friendship tie.

Fig 2. Parameter estimates and 95% confidence intervals for individual factors in SAB model smoking function



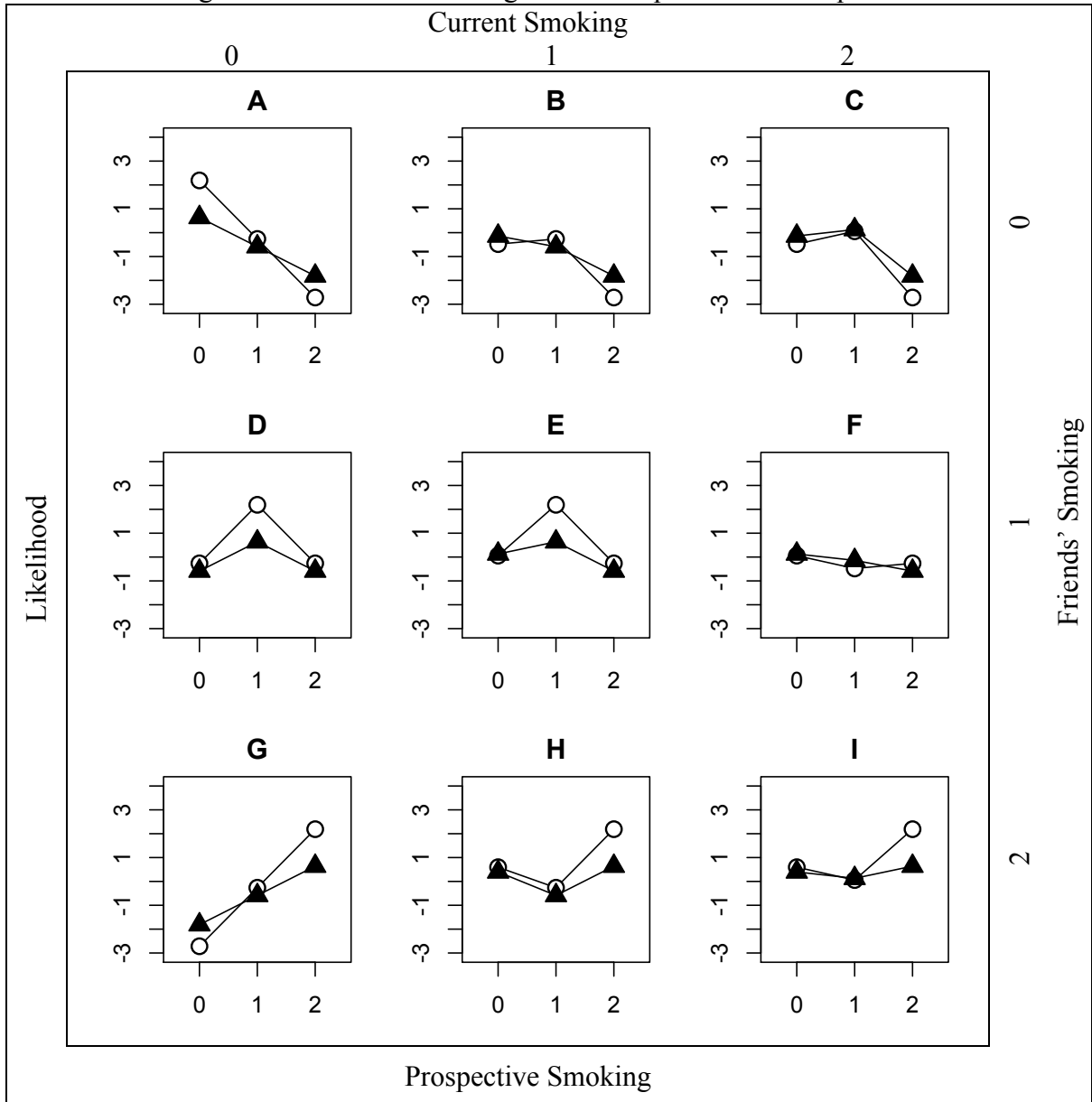
Note: Positive parameter estimates indicate that higher levels of smoking are more likely at higher levels of the predictor. Total similarity reflects the main effect of friends on smoking behavior, and is evaluated for potential increases, decreases, and sustaining the same level of smoking. Total similarity (decrease) indicates how the effect of friend deviates from the main effect for smoking decreases.

Fig 3. Contribution of linear and quadratic effects to predicted likelihood of adopting each level of smoking



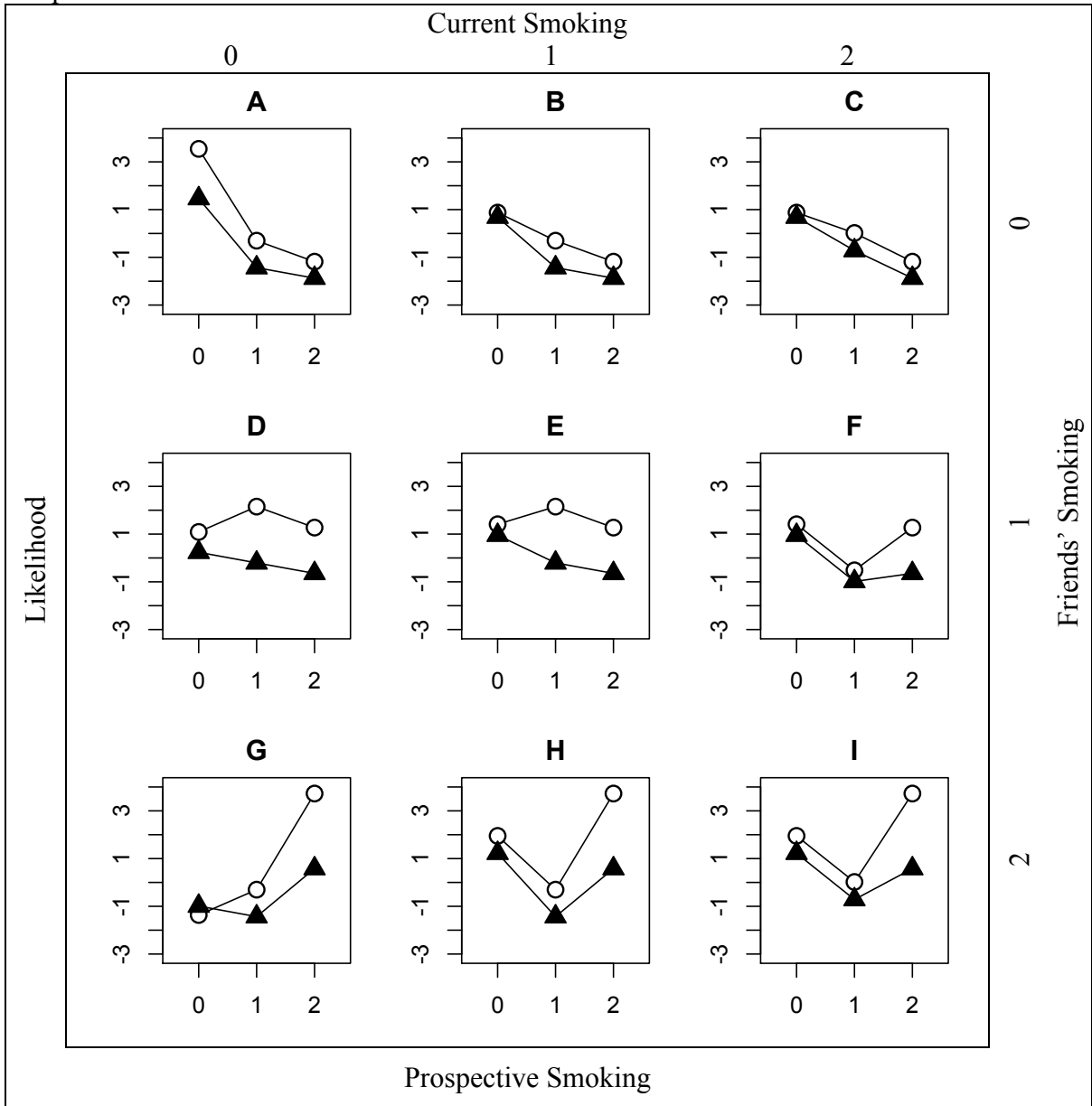
Note: Predicted likelihood of smoking is indicated by the contribution to the smoking function of ego's prospective smoking level. Higher values for the contribution to smoking function equate to a greater tendency to adopt that level of prospective smoking. Contribution to the smoking function is calculated based upon effects for: linear shape, quadratic shape, and school interactions. White circles represent Jefferson; black triangles represent Sunshine.

Fig 4. Contribution of total similarity effects to predicted likelihood of adopting each level of smoking based on current smoking level of respondent and respondents' friends



Note: Predicted likelihood of smoking is indicated by the contribution to the smoking function of ego's prospective smoking level conditional upon egos' current smoking and friends' current smoking levels. Higher values for the contribution to smoking function equate to a greater tendency to adopt that level of prospective smoking. Contribution to the smoking function is calculated based upon effects for: total similarity (increase and decrease) and school interactions. White circles represent Jefferson; black triangles represent Sunshine.

Fig 5. Contribution of linear, quadratic, and total similarity effects to predicted likelihood of adopting each level of smoking based on current smoking level of respondent and respondents' friends



Note: Predicted likelihood of smoking is indicated by the contribution to the smoking function of ego's prospective smoking level conditional upon egos' current smoking and friends' current smoking levels. Higher values for the contribution to smoking function equate to a greater tendency to adopt that level of prospective smoking. Contribution to the smoking function is calculated based upon effects for: linear shape, quadratic shape, total similarity (increase and decrease), and school interactions. White circles represent Jefferson; black triangles represent Sunshine.

Appendix A: Full SAB Model Parameter Estimates

Table A1: Network function parameter estimates from SAB model for friend selection and smoking change

	b		SE
<i>Rate effects</i>			
Jefferson, period 1	13.480	***	.471
Jefferson, period 2	11.102	***	.478
Sunshine, period 1	12.645	***	.420
Sunshine, period 2	6.620	***	.315
Effect of truncated roster on rate	-1.698	***	.149
<i>Endogenous network effects</i>			
Outdegree	-5.480	***	.084
Reciprocity	2.536	***	.066
Transitive triplets	.394	***	.016
Indegree - popularity (sqrt)	.104	***	.022
Indegree - activity (sqrt)	-.485	***	.031
Outdegree - activity (sqrt)	.426	***	.019
<i>Individual factors</i>			
Smoke alter	.124	***	.036
Smoke ego	.070	*	.031
Smoke similarity	.561	***	.066
Male alter	-.021		.023
Male ego	.016		.021
Male similarity	.273	***	.024
Age alter	-.006		.012
Age ego	-.039	***	.011
Age similarity	2.555	***	.124
Same race	.708	***	.028
GPA alter	.001		.017
GPA ego	-.025	#	.015
GPA similarity	.421	***	.060
Self-control alter	.002		.244
Self-control ego	.732	***	.214
Self-control similarity	.304	***	.097
Alcohol alter	.082	***	.024
Alcohol ego	.017		.025
Alcohol similarity	.188	***	.043
Course overlap	1.294	***	.066
Extracurricular activity co-participation	.227	***	.019
Truncated roster ego	-8.234	***	1.089

School ego	-1.964	***	.167
<i>School Interactions</i>			
Reciprocity	.502	***	.085
Outdegree - activity (sqrt)	.259	***	.029
Smoke alter	.102	*	.052
Smoke ego	.103	*	.050
Male alter	-.140	***	.045
Male similarity	.161	***	.044
Same race	1.023	***	.054
GPA alter	.160	***	.033
Self-control alter	-.010	**	.004
Alcohol ego	-.149	***	.044
Course overlap	.714	***	.113
Truncated roster ego	-.312		3.246

* $p < .05$; ** $p < .01$, *** $p < .001$ (two-tailed tests).

Table A2: Smoking function parameter estimates from SAB model for friend selection and smoking change

	b		SE
<i>Rate effects</i>			
Jefferson, period 1	1.371	***	.150
Jefferson, period 2	2.973	***	.392
Sunshine, period 1	1.641	***	.140
Sunshine, period 2	2.814	***	.453
<i>Behavior effects</i>			
Linear shape	-1.587	***	.108
Quadratic shape	1.308	***	.073
Total similarity (increase)	1.191	***	.361
Total similarity (decrease)	-1.452	*	.644
Alcohol	.189	***	.069
Male	.093		.075
Age	-.093	***	.036
Parent smoke	.160	*	.077
Extracurricular activity participation	-.148	#	.084
<i>School interactions</i>			
Linear shape	-1.601	***	.140
Quadratic shape	-.255		.194
Male	.282	*	.143

* $p < .05$; ** $p < .01$, *** $p < .001$ (two-tailed tests).