



# DYNAMIC NETWORKS AND BEHAVIOR: SEPARATING SELECTION FROM INFLUENCE

*Christian Steglich\**  
*Tom A. B. Snijders\*,†*  
*Michael Pearson‡*

*A recurrent problem in the analysis of behavioral dynamics, given a simultaneously evolving social network, is the difficulty of separating the effects of partner selection from the effects of social influence. Because misattribution of selection effects to social influence, or vice versa, suggests wrong conclusions about the social mechanisms underlying the observed dynamics, special diligence in data analysis is advisable. While a dependable and valid method would benefit several research areas, according to the best of our knowledge, it has been lacking in the extant literature. In this paper, we present a recently developed family of statistical models*

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\*University of Groningen, The Netherlands

†Nuffield College, University of Oxford

‡Edinburgh Napier University

*that enables researchers to separate the two effects in a statistically adequate manner. To illustrate our method, we investigate the roles of homophile selection and peer influence mechanisms in the joint dynamics of friendship formation and substance use among adolescents. Making use of a three-wave panel measured in the years 1995–1997 at a school in Scotland, we are able to assess the strength of selection and influence mechanisms and quantify the relative contributions of homophile selection, assimilation to peers, and control mechanisms to observed similarity of substance use among friends.*

## 1. INTRODUCTION

In social groups, there generally is interdependence between the group members' individual behavior and attitudes, and the network structure of social ties between them. The study of such interdependence is a recurring theme in theory formation as well as empirical research in the social sciences. Sociologists have long known that structural cohesion among group members is important for compliance with group norms (Durkheim 1893; Homans 1974). Research on social identity theory identified within-group similarity and between-group dissimilarity as principles by which populations are subdivided into cohesive smaller social units (Taylor and Crocker 1981; Abrams and Hogg 1990). Detailed network studies (e.g., Padgett and Ansell 1993) as well as discussion essays (Emirbayer and Goodwin 1994; Stokman and Doreian 1997) made clear that to obtain a deeper understanding of social action and social structure, it is necessary to study the dynamics of individual outcomes and network structure, and how these mutually impinge upon one another. In methodological terms, this means that complete network structure as well as relevant actor attributes—indicators of performance and success, attitudes and cognitions, behavioral tendencies—must be studied as joint dependent variables in a longitudinal framework where the network structure and the individual attributes mutually influence one another. We argue that previous empirical studies of such dynamics have failed to address fundamental statistical and methodological issues. As demonstrated by Mouw (2006) for the case of social capital effects on individual outcomes, this failure can lead to severe bias in reported results. As an alternative, we present a new, statistically rigorous method for this type of investigation, which we illustrate in an elaborate empirical application.

The example concerns the joint dynamics of friendship and substance use in adolescent peer networks (Hollingshead 1949; Newcomb 1962). It is by now well-established that the smoking, alcohol, and drug use patterns of two adolescents tend to be more similar when these adolescents are friends than when they are not (Cohen 1977; Kandel 1978; Brook, Whiteman, and Gordon 1983). Formulated more generally, people who are closely related to each other tend to be similar on salient individual behavior and attitude dimensions—a phenomenon for which Fararo and Sunshine (1964) coined the term *homogeneity bias*. In statistical terminology, this kind of association is known by the name of *network autocorrelation*, a notion originating from the spatial statistics literature (Doreian 1989). Up until now, however, the dynamic processes that give rise to network autocorrelation have not been sufficiently understood. Some theorists evoke *influence mechanisms* and *contagion* as possible explanations (Friedkin 1998, 2001; Oetting and Donnermeyer 1998)—a perspective largely in line with classical sociological theory on socialization and coercion. Others invoke *selection mechanisms*, more specifically *homophily* (Lazarsfeld and Merton 1954; Byrne 1971; McPherson and Smith-Lovin 1987; McPherson, Smith-Lovin, and Cook 2001), while still others emphasize the unresolved tension between these alternative perspectives (Ennett and Bauman 1994; Leenders 1995; Pearson and Michell 2000; Haynie 2001; Pearson and West 2003; Kirke 2004). Attempts to overcome this tension on the theoretical level are rare and in general not geared to statistical analysis, but they employ simulation (e.g., Carley 1991) or derive properties of long-term equilibria (Friedkin and Johnsen 2003). For the empirical researcher, these attempts therefore may not be very helpful.

In order to explain network autocorrelation phenomena, a dynamic perspective is indeed necessary. Considering the case of network-autocorrelated tobacco use, a smoker may tend to have smoking friends because, once somebody is a smoker, he or she is likely to meet other smokers in smoking areas and thus has more opportunities to form friendship ties with them (“*selection*”). At the same time, it may have been the friendship with a smoker that made him or her start smoking in the first place (“*influence*”). Which of the two patterns plays the stronger role can be decisive for success or failure of possible intervention programs—moreover, a policy that is successful for one type of substance use (say, smoking) may fail for another (say, drinking) if the generating processes are different in nature. Modeling this as a

dynamic process using longitudinal network data is necessary to address the problem adequately (Valente 2003).

The most common format of such data in sociological studies is the panel design—which introduces some analytical complications, because the processes of influence and selection must reasonably be assumed to operate unobservedly in continuous time between the panel waves. Finally, *complete network* studies (i.e., measurements of the whole network structure in a given group) are clearly preferable to personal (ego-centered) network studies, because selection patterns can best be studied when the properties of nonchosen potential partners are also known, and because of the possible importance of indirect ties (two persons having common friends, etc.) that are difficult to assess in personal network studies. Complete network data, on the other hand, are characterized by highly interdependent observations. This rules out the application of statistical methods that rely on independent observations—i.e., most standard techniques. To our knowledge, no previous study succeeded in a statistically and methodologically credible assessment and the separation of selection and influence mechanisms.

In this paper, we show how previous approaches failed to adequately respond to these statistical-methodological challenges, and we present a new, flexible method that enables researchers to statistically separate the effects of selection from those of influence. A fundamental difference with respect to earlier approaches is that our method formalizes the simultaneous, joint evolution of social networks and behavioral characteristics of the network actors in an explicit, stochastic model. Proposed and described mathematically by Snijders, Steglich, and Schweinberger (2007), it can be fitted to data collected in a panel design, where complete networks as well as changeable attributes are measured. We will call this data type *network-behavior panel data*, understanding that “behavior” here stands for changeable attributes in a wide sense, including attitudes, performance, etc. Model fitting yields parameter estimates that can be used for making inferences about the mechanisms driving the evolution process. The new method extends earlier methodology for the analysis of “pure” network dynamics (Snijders 2001, 2005) by adding components that allow for the inclusion of coevolving behavioral variables.

In addition to the already mentioned network autocorrelation phenomena, other aspects of dynamic network-behavior interdependence can in principle be investigated with our models, when

network-behavior panel data are available. For instance, Granovetter's (1982) theory about weak ties as providers of opportunities for changing individual properties, or Burt's (1987, 1992) theory about brokerage and structural competition, can be tested for their validity on the actor level in a dynamic context where the network and individual actor characteristics are subject to mutually dependent endogenous change. More generally, the focus of the research to which our models can be applied need not include the aspect of partner selection, but it can also be on the level of individual outcomes. Whenever endogeneity of partner choice is regarded as an obstacle to reliable inferences, the study of the co-dependent network as a second dependent variable is opportune, and the modeling proposed here is a potentially useful avenue. A case in point is the study of causal relations between social capital and labor market outcomes, as critically reviewed by Mouw (2006). We think that the modeling presented here has the potential to open new paths for theory development and testing in many network-related research areas. In the present paper, however, we restrict attention to a general sketch of the modeling framework and one illustrative application, the analysis of selection and influence mechanisms with respect to substance use behavior (tobacco and alcohol consumption), based on network-behavior panel data measured in 1995–1997 at a secondary school in Scotland.

### 1.1. *Overview*

We begin with an illustration of the problem of assessing simultaneously operating selection and influence processes by identifying three major methodological obstacles that need to be addressed, and giving a summary review and critique of prior research methods. The example of friendship and substance use in adolescent peer groups will play only a tangential role at this stage. Then, the actor-based model family for network and behavior coevolution is presented. We illustrate the new method by applying it to a three-wave data set about the coevolution of smoking and drinking behavior with friendship networks (Pearson and West 2003). In these analyses, selection and influence effects are assessed simultaneously, hence controlling one effect for the other. A simulation study based on the obtained parameter estimates allows us to draw quantitative conclusions about the sources of observed network autocorrelation. In addition to selection and influence on the two substance

use dimensions, three other possible sources are distinguished: (1) the carryover of network autocorrelation that already existed at the beginning of the investigated period, (2) selection based on other individual variables (sex, age, money, dating), and (3) the effects of endogenous network formation (reciprocity, balance, and network closure). In the concluding section, the main results of the article are summarized, and prospective further applications of the new method are sketched.

## 2. NETWORK AUTOCORRELATION AS AN EMPIRICAL PUZZLE

The genesis of network autocorrelation is not yet sufficiently understood in many research areas (Manski 1993), and multiple explanatory propositions and theories coexist. This also holds for the literature on the effects of peer groups in adolescence, on which we focus here. We can find several quite specific hypotheses about how friendship networks coevolve with behavioral dimensions in general, and with tobacco use and alcohol consumption in particular. The underlying theories posit conceptually distinct mechanisms which, nonetheless, lead to similar cross-sectional patterns of network autocorrelation. We confine our review to a short sketch of the three most prominent mechanisms covered in extant literature: (1) assimilation, (2) homophily, and (3) social context.

According to socialization theory, peer groups are responsible for creating behavioral homogeneity in a group (Olson 1965; Homans 1974). The claim is that adolescents, being influenced by their peers, will *assimilate* to the peers' behavior, hence the finding of similarity among friends. Applications can be found—for example, in a series of papers on substance use by Oetting and colleagues (Oetting and Beauvais 1987; Oetting and Donnermeyer 1998), or in Haynie's (2001) analysis of adolescents' delinquency. In this perspective, change primarily takes place in the individual's behavior, while the friendship network is treated as rather static (Friedkin 1998, 2001).

A diametrically opposite approach, in which the network is treated as dynamic but its determinants as rather static, is taken by research on friendship formation (Byrne 1971; Lazarsfeld and Merton 1954; Moody 2002). Here, similarity among friends is explained by selection—that is, by similar adolescents seeking out each other as

friends. There can be different reasons why this happens. The most widely known is *homophily*, the principle by which similarity in behavior acts as a direct cause of interpersonal attraction (Lazarsfeld and Merton 1954; McPherson and Smith-Lovin 1987; McPherson et al. 2001). Applications of theories on homophily to adolescents' substance use can be found, for example, in Fisher and Bauman (1988), Ennett and Bauman (1994), Elliott, Huizinga, and Ageton (1985), and Thornberry and Krohn (1997).

As stressed by Feld (1981, 1982; Kalmijn and Flap 2001), however, what on the surface looks like homophily may in reality result from other network formation processes. Different *social contexts* (settings, foci) can be responsible for the observed similarity of friends, because when people select themselves to the same social setting, this will usually indicate prior similarity on a host of individual properties, while actual friendship formation simply reflects the opportunity of meeting in this social setting and does not allow us to infer a causal role of the prior similarity in friendship creation. The argument can be extended to also cover informal forms of social settings, such as the opportunity structure of meeting others that is implied by the social network itself (Pattison and Robins 2002). Endogenous mechanisms of friendship dynamics, such as network closure or social balance (Feld and Elmore 1982; van de Bunt, van Duijn, and Snijders 1999), may this way also lead to similarity of friends and must not be misdiagnosed as homophilous selection patterns (Berndt and Keefe 1995).

In the terminology of Borgatti and Foster (2003), the main dimensions on which these three accounts of network autocorrelation differ are whether behavior is treated as a consequence of the network (assimilation) or as its antecedent (homophily, context), and whether temporal antecedence is also causal (homophily) or only correlational (context). In the case of adolescents' substance use in friendship networks, it is obvious that all three accounts potentially play a role, as adolescents typically change their networks frequently (Degirmencioglu, Richards, Tolson, and Urberg 1998) and many of them start experimenting with risk behavior such as substance use. As can already be seen from the brief discussion above, multiple theoretical accounts have been advanced for explaining network autocorrelation, and a test of these theories against each other is expedient. Considering that previous research on substance use found evidence for both selection and influence processes, this holds all the more in the case of our empirical

application. In our analyses, we will achieve a separation of assimilation, homophily, and other effects (some related to social context, but also others), on two substance use dimensions. This separation consists of hypothesis tests of the different autocorrelation-generating mechanisms in one model, and calculation of model-based effect sizes for these mechanisms.

Our method is certainly not the first attempt to simultaneously assess selection and influence and to determine the relative strength of each process. However, it differs from previous approaches by employing a model that explicitly represents the mutual dependence between network and behavior, coevolving in continuous time. Such a model permits a more rigorous statistical treatment than previous methods, which left the model implicit. Restrictions inherent to our method will be addressed in the discussion. In the following sections, we will provide reasons why previous, similar attempts cannot be considered trustworthy. For this aim, a set of criteria (“key issues”) is derived which an explanatory model for network autocorrelation should fulfill. The section ends with an evaluation of previous attempts at disentangling selection and influence, making use of these criteria.

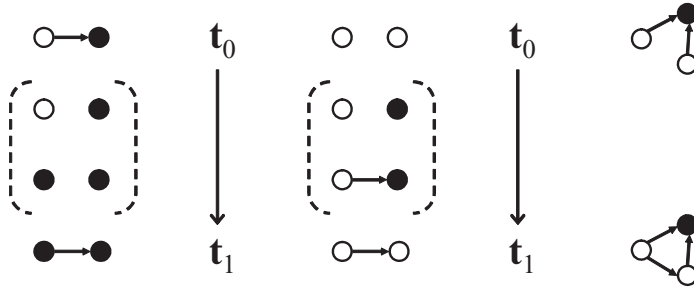
### 2.1. *Key Issues and a Typology of Previous Approaches*

A few earlier studies tested the competing explanations of network autocorrelation against each other in longitudinal studies of complete networks. The earliest publications on this topic seem to be the articles by Cohen (1977) and Kandel (1978), which represent two of the three major previous approaches to the study of network autocorrelation that we propose to distinguish here. These are the *contingency table approach* (Kandel 1978; Billy and Udry 1985; Fisher and Bauman 1988), the *aggregated personal network approach* (Cohen 1977; Ennett and Bauman 1994; Pearson and West 2003; Kirke 2004), and *structural equation modeling* (Krohn et al. 1996; Iannotti, Bush, and Weinfurt 1996; Simons-Morton and Chen 2005; de Vries et al. 2006). We will shortly characterize and evaluate these approaches against three key issues that are fundamental for the separation of selection and influence effects. These are (1) *network dependence* of the actors, which is essential in modeling dynamics of network ties, and which precludes the application of statistical techniques that rely on independent observations;



(2) the necessity to control for *alternative mechanisms* of network evolution and behavioral change in order to avoid misinterpretation in terms of homophily and assimilation; and (3) *incomplete observations* implied by the use of panel data while the underlying evolution processes operate in continuous time.

These key issues are interrelated, as will become clear in our discussion of the limitations of how they are handled by earlier proposed methods. Especially in view of the third key issue, a consideration of data formats is opportune. Panel data, measured at only a few discrete time points, are the most common longitudinal format in sociological studies, and social network research is no exception. The temporal incompleteness of panel data makes it impossible to unequivocally identify which process is responsible for an observed change, even if only the network or only the behavior changes from one observation to the next—simply because a change on the respective other dimension may also have happened, but there has been a change back to the original value afterward during the same period. The two columns on the left in Figure 1 illustrate such situations. Suppose that the pair of pupils in the middle column is observed at time points  $t_0$  and  $t_1$ . At both moments, they are nondrinkers, but while they are unconnected at  $t_0$ , there is a unilateral friendship tie between them at  $t_1$ . At first sight, we might diagnose a pattern of homophilous selection. However, the unobserved process that generated these data may have looked fundamentally different, as illustrated in the brackets. A while after the observation at  $t_0$ , the actor on the right may have started drinking, say, because he did not have any friends. The actor on the left may have noticed that and started a therapeutic friendship with the new drinker. Under these circumstances, the drinker quit drinking again. Only now, the network is observed again at  $t_1$ . In this scenario, the processes actually happening have nothing to do with homophilous selection, and to diagnose the observations as unequivocal evidence for selection is plainly wrong. Nonetheless, literally *all* quantitative studies on the topic that we are aware of commit this error. As the example illustrates, alternative mechanisms of network formation as well as behavior change need to be controlled for in order to preclude such misinterpretation. A similar scenario, sketched in the left of Figure 1, illustrates how homophilous selection (taking place shortly before observation moment  $t_1$ ) can be misdiagnosed as social influence (the default interpretation of the observed data when the happenings in the brackets are neglected). The longer the time intervals



**FIGURE 1.** Illustrations for two of the three “key issues” mentioned in the text: incomplete observation of changes (left and middle column) and alternative mechanisms (all columns).

are between observations, the higher the chances that such alternative trajectories happen. Calculations based on our empirical study suggest that on top of the observed changes, another 7–10% of changes remain unobserved because they cancel each other out before observation. In studies on adolescent behavior, time intervals of one year are the rule, while scenarios as sketched in Figure 1 can reasonably be assumed to take place within a few months. The use of retrospective questions for assessing the particular relationship’s history (Kirke 2004) in principle could remedy this predicament. However, retrospective social network information is rare and prone to unreliability (Bernard et al. 1985), so this remedy leads to other problems.

The problem of alternative generating mechanisms is not limited to situations where the data are incompletely observed. In the column on the right of Figure 1, the newly created tie *could* result from homophilous selection (and indeed would be unequivocally diagnosed as such by previous approaches in the literature). However, it could also result from a purely structural mechanism known to play a strong role in friendship formation—namely, triadic closure. Having a common friend at  $t_0$  may be the reason why at  $t_1$ , a tie is established between the two previously unrelated actors. The message is that even if we can assume that no unobserved changes have taken place, there still is interpretative leeway concerning the mechanisms responsible for a given observed change. Controlling for such mechanisms as far as possible is a criterion that previous research largely has failed to address.

Next to the temporal aspect of data collection, the cross-sectional design is also important in distinguishing selection from influence

effects. There are two ideal types of social network studies: (1) *ego-centered network studies*, in which for a random sample of individuals (the egos), the network neighbors and their properties are assessed; and (2) *complete network studies*, in which for a given set of actors, all relational links in this set are assessed, and the assumption is made that for the research question being addressed, links outside of this set are less consequential and by approximation can be neglected. For the present purpose, an ego-centered network design is inadequate because even in a panel study, such data do not provide information about other, potential relational partners that were not selected. This precludes a meaningful assessment of selection processes. For adequately measuring selection effects, a meaningful approximation of the set of potential relational partners must be made, whose individual properties must be known irrespective of whether they actually become partners or not. In studies of complete networks, these data are available for all actors in the network. Thus, a necessary condition for conducting a complete network study is that the delineation of the network gives a reasonable approximation to the set of the actual and potential relational partners. However, this information comes at the price of dependence of observations, which rules out the application of the common statistical procedures, as these rely on independence of residuals. Depending on the exact nature of the data-generating process, such procedures can be biased toward conservative as well as liberal testing (Kenny and Judd 1986; Bliese and Hanges 2004), and therefore lack reliability.

## 2.2. *An Assessment of Previously Used Analytical Methods*

There have been earlier attempts to separate selection effects from influence effects, which we categorize in three main groups: (1) modeling frequencies in a contingency table, (2) analyzing characteristics aggregated from the personal network, and (3) structural equation modeling. Here, we will briefly characterize these methods, in this order, and highlight the degree to which they meet the requirements on the three key issues introduced.

In the contingency table approach, dyads of mutually chosen best friends are typically cross-tabulated according to whether or not the two pupils' friendship remains stable between first and second measurement, and whether or not their behavior falls in the same, binary

category. Influence is typically assessed by studying the subsample of respondents who named the same best friend in both waves, while selection is assessed on the subsample of changing friendship ties. For both mechanisms, probabilities of change toward a behaviorally homogeneous friendship are calculated, and based on these probabilities, predictions are generated for the whole sample of dyads. Significance tests for the two mechanisms are then derived by comparing these predictions to the observed data, under the assumption of dyad independence. Prominent research that employs variants of this approach are Kandell's (1978) seminal study of marijuana use, educational aspirations, political orientation, and delinquency at New York State high schools, Billy and Udry's (1985) study of adolescents' sexual behavior, and Fisher and Bauman's (1988) investigation of adolescents' smoking and alcohol consumption. Our key issue of network dependence, here in the shape of inadequately assuming dyad independence, is usually acknowledged as an acceptable weakness in this research. The two other key issues, though, remain fully unaddressed. The use of incompletely observed data in particular puts a question mark over the results. Because the categorization of dyads in the contingency table is based only on the observed states at the beginning and at the end of the observation period, the possibility of multiple explanatory trajectories linking these observations (as shown in Figure 1) is ruled out. Alternative generating mechanisms, finally, can to some degree be controlled for in this approach; see Fisher and Bauman (1988) for an example. However, triadic effects such as network closure or balance cannot be controlled because of the restriction to dyads.

The studies we summarize as taking an aggregated personal network approach follow an explicit two-stage strategy. First, the network data are collapsed into summary statistics on the actor level, indicating the actor's network position and properties of his personal network. Second, these reduced data are analyzed under independence assumptions. Implicit in this approach is the assumption that all change is observed and sufficiently reflected in the chosen statistics. The independence assumptions used in the second stage are clearly erroneous, which means that the studies cannot establish a firm statistical conclusion concerning processes of influence and selection. Next to leaving all our key issues unresolved, however, there are additional problems with this approach. One is related to the arbitrariness in the choice

of the particular preprocessing algorithm. While Ennett and Bauman (1994), Pearson and Michell (2000), and Pearson and West (2003) make use of the *negopy* software (Richards 1995), which categorizes respondents into the four sociometric positions—group member, peripheral, liaison, and isolate—Cohen (1977) relies on a definition of sociometric groups proposed by Coleman (1961), while Kirke (2004) relies on the identification of weak components provided by the *GRADAP* software (Sprenger and Stokman 1995). The different options available at this preprocessing stage are manifold, and their consequences are not well-understood. More importantly, though, the reliance on the output of such algorithms implies that network positions and neighborhood properties are used as if they were exogenously determined actor attributes. Further mutual interdependencies in the network structure, or the specific identity of the peers, are not taken into account. A “group member” at one observation may still be identified as a “group member” at the subsequent observation, yet the groups referred to may consist of completely different peers. In such a situation, even the assumption that “the group” exerted social influence in the period in-between seems dubious. Also, the selection of specific peers cannot be modeled in an actor-based analysis, and accordingly, the study of influence processes is not controlled for selection. As partial solution to the problem of peer identity, the actor-level statistics obtained in the first stage can be combined with an analysis on the dyad level under a contingency table approach; see Ennett and Bauman (1994) for an example.

The third type of studies that we distinguish here employ structural equation models, specified as a cross-lagged panel or a latent trajectory model, to assess selection and influence (Krohn et al. 1996; Iannotti et al. 1996; Simons-Morton and Chen 2005; De Vries et al. 2006). This approach has been used for data collected using complete network as well as personal network designs. An advantage of this approach over the other two is the relative ease with which selection and influence effects can be simultaneously assessed in the same analysis, controlling one for the occurrence of the other. The dependent variable for selection is defined here not at the dyadic level but by using a variable expressing peer behavior, which is an aggregate of the personal network, usually defined as the average behavior of the current peers. In cross-lagged model specifications, there are paths from previous-wave respondent behavior to current-wave peer behavior, and from

previous-wave peer behavior to current-wave respondent behavior. In this setup, the estimated path coefficient from lagged peer behavior to current respondent behavior is taken as a measure of peer influence, while the coefficient for the path from lagged respondent behavior to current peer behavior is taken as an indicator for selection effects. By estimating separate effects for old friends and new friends, the interpretation of path coefficients in terms of selection and influence is possible, at least conceptually. However, the method still neglects the three key issues of incomplete observations (estimated path coefficients directly link the observed variables to each other), alternative generating mechanisms (selection being modeled at an aggregate level precludes expressing alternative mechanisms at the dyadic or triadic level), and—here especially important—the interdependence of observations. When this approach is applied to complete network data, the individual respondent, whose behavior figures centrally in one observation, will also appear among the peers for other observations. This clearly violates independence of observations, which is one of the crucial assumptions of structural equation modeling. While the use of ego-centered data would solve this problem, it is not an option because it effectively precludes the study of selection processes, as outlined earlier. Mouw (2006) identifies a number of other limitations of the cross-lagged panel specification for the case of ego-centered data, in particular that this specification controls insufficiently for stable unobserved differences between respondents.

To summarize: the three analytical strategies covered here all follow a two-stage procedure. In the first stage, the network data are collapsed into individual-level variables (e.g., local density, centrality, indicators of group position, peer behavior) or dyad-level variables (behavioral homogeneity), which in the second stage figure as variables in more conventional analyses—as dependent variables for assessing selection effects, and as independent variables for assessing effects of social influence. The shortcomings of such approaches are related to the key issues introduced earlier. The stage of collapsing networks into individual- or dyad-level data remains arbitrary and does not do full justice to the structural aspect of evolving networks. In particular, the use of such collapsed variables artificially freezes their values at the last preceding observation, which negates their endogenously changing nature and inhibits the study of potentially important feedback mechanisms between network and behavior that are unobserved.

Alternative mechanisms that may be responsible for observed changes are generally difficult to control for in such models, especially when they express triadic or higher-order network effects such as closure or structural balance. Finally, due to the problem of nonindependence of actors and dyads, the first step of data reduction does not deliver data that meet the requirements of the statistical procedures applied in the second-stage analyses.

### 3. A NEW APPROACH: MODELING THE COEVOLUTION OF NETWORKS AND BEHAVIOR

A more principled statistical approach is possible by basing the data analysis on an explicit probability model for network-behavior coevolution that deals in a satisfactory way with the three key issues of network dependence, control for alternative mechanisms, and unobserved changes in between observation moments. A computational model, which can be analyzed by means of computer simulations, is likely to be the only satisfactory way to proceed. There is a rich tradition in computational models for networks, and a smaller literature in which network ties as well as individual attributes change endogenously. Some examples are Carley (1991), Macy et al. (2003), and Ehrhardt, Marsili, and Vega-Redondo (2007). In part of this literature the changing attributes refer to strategies played in games (e.g., Eguíluz et al. 2005). Research in the physics literature on models of mutually dependent networks and attributes is reviewed by Gross and Blasius (2008). This literature is mainly oriented toward deriving macrolevel properties of data sets implied by models defined by relatively simple behavioral rules—but it is not very useful for purposes of statistical inference. Statistical models for cross-sectional data with mutually dependent network ties were proposed by Robins, Elliott, and Pattison (2001) and Robins, Pattison, and Elliott (2001), and are further discussed by Robins and Pattison (2005). For our purpose of longitudinal statistical analysis, we make use of the model proposed by Snijders and colleagues (2007), which is an extension of earlier modeling work by Snijders (2001, 2005) for networks without coevolving behavioral dimensions. This model has a transparent definition as a stochastic process, and it is sufficiently flexible to incorporate a wide variety of alternative mechanisms. The process of network-behavioral coevolution is modeled here as an emergent

group-level result of interdependent behavioral changes occurring for single actors, and network changes occurring for pairs of actors. One major characteristic of this model is its assumption that changes may occur continuously between the observation moments. Models for panel data that assume unobserved changes at arbitrary moments between panel waves are particularly attractive for modeling feedback between multiple variables. Such models are called continuous-time models, and they were proposed for discrete variables by Coleman (1964) and discussed for continuous variables, for example, by Arminger (1986) and Singer (1998). Holland and Leinhardt (1977) proposed to use continuous-time models specifically for network dynamics.

Handling the dynamic mutual dependence of the network ties and the individual behavior requires a process model that specifies these dependencies in a plausible way. The way to achieve this is based on the second major characteristic of this model, which is its actor-based architecture. An explicit actor-based approach is in line with extant theories of individuals who act in the context of a social network. For example, for the study of friendship networks, taking the network actors as the foci of modeling seems natural, as commonly invoked mechanisms of friendship formation (like homophily, reciprocity, or transitive closure) are traditionally formulated and understood as forces operating at the actor level, within the context of the network; the same holds for mechanisms of behavioral change (like social influence). Modeling these changes in an actor-based framework implies that actors are assumed to “make” the change, by altering either their outgoing network ties or their behavior. The central model components will be the actors’ behavioral rules determining these changes.

### 3.1. *Action Rules and Occasions to Act*

Some assumptions need to be made for the model to be tractable. Changes in network ties and behavior are assumed to happen in continuous time, at stochastically determined moments. This allows us to tackle the key issue of unobserved changes. We follow the principle, proposed by Holland and Leinhardt (1977) for network dynamics, to decompose the change process into its smallest steps. This is a way to obtain a relatively parsimonious model, and its implementation combines in a natural way with the continuous time parameter. Distinguishing



between the network changes of an actor and his behavior changes, this principle rules out the possibility that changes in network ties and in actor behavior, or changes by two different actors, occur at the same time point. An example for such forbidden simultaneous changes would be binding contracts of the type “when you start smoking, I’ll become your friend.” While such bargaining is clearly possible in reality, we will here model it as two subsequent changes, the connection of which cannot be enforced. Given the present application of the model to the evolution of substance use and friendship ties, such an assumption seems reasonable—in other applications, it could be relaxed. The compound change that is observed between two observations thus is interpreted as resulting from many small, unobserved changes that occurred between the observation moments. The assumption that at any given moment, not more than one tie variable or one behavior variable can change, enables us to keep the rules that govern actors’ behavior relatively simple, relieving us from the burden of explicitly modeling the totality of changes between two measurements all at once, an advantage of continuous-time modeling put forward already by Coleman (1964). Here, this assumption provides an elegant and simple way of expressing the feedback processes inherent in the dynamic process, where the currently reached state is also the initial state for further developments, and where the probabilities for specific changes can depend, in perhaps complicated ways, on the entire current network-behavior configuration. The continuous-time nature of the model this way allows to pragmatically express the total dynamics according to a simple set of behavioral principles, with the observed, complex patterning accruing over time. In a framework without unobserved changes between observation moments, the same complex patterning could be expressed only by a larger number of more complicated effects, which would obscure the potentially much more simple mechanisms that may govern the change process. For example, the model then would need a separate component to represent the probability that three previously disconnected actors form a clique at the next observation—whereas in our model this can be represented as the resultant of the more basic processes of single tie formation, reciprocation, and transitive closure. The actor-based model allows us to represent network dependencies and also to represent alternative generating mechanisms that are endogenous to the network or dependent on observed actor variables—two of our key issues. There is a cost to this approach, however. Because

TABLE 1  
Schematic Overview of the Model Components

	Occurrence	Rule of Change
Network changes	Network rate function	Network objective function
Behavioral changes	Behavioral rate function	Behavioral objective function

we cannot know which precise trajectory of small changes happened from one observation to the next, we have to rely on data augmentation procedures and simulation-based inference for estimating our models. In the simulations of the model that are employed in the estimation procedure, the first observation in the panel data set is not modeled but conditioned upon, and used only as the starting values in the simulation of network ties and behavior. This implies that the evolution process is modeled without contamination by the contingencies leading to the initial state, and that no assumption of a dynamic equilibrium needs to be invoked. For changes of network as well as behavior, we distinguish the temporal occurrence of opportunities for the different types of changes, and the rules of change followed by the actors, once they face such an opportunity.

These model components, summarized in Table 1, will be sketched below in a formal probabilistic operationalization, using the application to substance use in high school as an illustration. Formally, the model is a continuous time Markov process, where the totality of possible combinations of network ties and actor behavior figures as the state space. While the model in principle is equipped for analyzing the coevolution of multiple dimensions of networks and behavior, we consider—for ease of presentation—the case of one network variable  $\mathbf{X}$  and one dependent actor variable  $\mathbf{Z}$  only (in the empirical section, we will give an example with two behavioral dimensions). In the following, first some notational conventions are introduced, and then the formal model is sketched. A detailed mathematical account of our model is given in Snijders and colleagues (2007).

### 3.2. *Notation and Data Requirements*

The outcome variables for which the model is defined are the changing network and the changing actor behavior—which may be called endogenous, since they evolve as a function of each other. Independent (exogenous) variables can be individual or dyadic, changing or

unchanging. We make use of the following notation. The network is assumed to be based in a group of  $N$  actors—for example, a cohort of pupils at the same school, or business firms active in the same period in the same industry. The network is denoted by  $\mathbf{x}$ , where  $\mathbf{x}_{ij}(\mathbf{t})$  stands for the value of the directed relationship between actors  $i$  and  $j$  at time point  $\mathbf{t}$ . Examples for such relational variables are *friendship* between the pupils of a year group, *advice* among employees in an organization, or *share ownership* between business firms. We assume that  $\mathbf{x}$  is *dichotomous*—that is,  $\mathbf{x}_{ij} = 1$  stands for presence of a tie and  $\mathbf{x}_{ij} = 0$  for absence. Next,  $\mathbf{z}$  denotes the behavioral variable, with  $\mathbf{z}_i(\mathbf{t})$  standing for the score of actor  $i$  at time point  $\mathbf{t}$ . Examples here are the *smoking behavior* of pupils, the *performance* of employees, or the *activity in a given market segment* of a business firm. We assume that behavioral dimensions are measured on a discrete, ordinal scale represented by integer values (e.g., dichotomous scales). Finally,  $\mathbf{v}$  and  $\mathbf{w}$  denote actor-level and dyad-level exogenous covariates, respectively (for ease of presentation here assumed to be constant over time), with  $\mathbf{v}_i^{(k)}$  standing for the score of actor  $i$  on actor covariate  $k$ , and  $\mathbf{w}_{ij}^{(k)}$  standing for the dyadic covariate  $k$  measured for the pair  $(ij)$ . Typical actor covariates are *sex*, *age*, or *education* of a pupil or an employee, or *number of employees* of a business firm. Examples for dyadic covariates could be a *classmate relation* between pupils in a year group, an *organizational hierarchy* between employees, or the *geographical distance* of business firms.

We consider the case of *network-behavior panel data*, where the network and behavioral data are collected for a finite set of time points only (say,  $\mathbf{t}_1 < \mathbf{t}_2 < \dots < \mathbf{t}_M$ ), with at least  $M = 2$  waves. In the following section, the data are indicated by lowercase letters (networks  $\mathbf{x}(\mathbf{t}_1), \dots, \mathbf{x}(\mathbf{t}_M)$ , behavior  $\mathbf{z}(\mathbf{t}_1), \dots, \mathbf{z}(\mathbf{t}_M)$ , etc.), while the stochastic model components (of which these data are assumed to be realizations) are indicated by uppercase letters (network model  $\mathbf{X}(\mathbf{t})$  and behavioral model  $\mathbf{Z}(\mathbf{t})$ ). The formal model is obtained by spelling out the submodels indicated in Table 1 and integrating them into the overall model. Although the objective functions are the most important model components, for ease of presentation we first explain the model for occurrence of changes.

### 3.3. Modeling Opportunities for Change

The assumption was already mentioned that at any single moment, only one tie variable or one behavioral variable may change. More

specifically, it is assumed that at stochastically determined moments, one actor gets the opportunity to change one of his or her outgoing tie variables, or to change his or her behavior. Such opportunities for change are called *micro steps*. It is also allowed that the actor does not make a change but leaves things as they are. The frequency by which actors have the opportunity to make a change is modeled by *rate functions*, one for each type of change. The main reason for having separate rate functions for the behavioral and the network changes is that practically always, one type of decision will indeed be made more frequently than the other. In information flow networks, for example, we can expect that the actors' individual properties (here: knowledge states) change more quickly than their network ties. In group formation processes, where the behavioral dimensions may represent attitudes, the opposite may be true. In the application to substance use and friendship at high school, we would expect more frequent changes in the network than in substance use, caused by (1) the addictive nature of substance use and (2) the students' social orientation phase in adolescence.

The first observations of network ties  $\mathbf{x}(t_1)$  and behavior  $\mathbf{z}(t_1)$  serve as starting values of the evolution process — that is, they are not modeled themselves but conditioned upon, and only the subsequent changes of network ties and behavior are modeled. In order to obtain a Markov process, waiting times between micro steps must have exponential distributions, as this is the only distribution with the lack of memory property (Norris 1997). For each actor  $i$  and for network and behavioral changes alike, we model the waiting time until actor  $i$  takes a micro step by exponentially distributed variables  $T_i^{\text{net}}$  and  $T_i^{\text{beh}}$  with parameters  $\lambda_i^{\text{net}} > 0$  and  $\lambda_i^{\text{beh}} > 0$ , i.e., the waiting times are distributed such that  $\Pr(T > t) = \exp(-\lambda t)$  for all  $t > 0$ . The parameters of these distributions indicate the rate (or speed) at which the respective change is likely to occur; the expected waiting time is  $1/\lambda$ . Since actual waiting times between changes are not observed, more complicated modeling is unwarranted. It is further assumed that all waiting times are *independent*, given the current state of network and behavior. Properties of the exponential distribution imply that, starting from any given moment in time (e.g., the moment of the preceding micro step), the waiting time until occurrence of the next micro step of either kind by any actor is exponentially distributed with parameter

$$\lambda_{\text{total}} = \sum_i (\lambda_i^{\text{net}} + \lambda_i^{\text{beh}}). \quad (1)$$

The probability that this is a network micro step taken by a particular actor  $\mathbf{i}$  is given by

$$\lambda_{\mathbf{i}}^{\text{net}} / \lambda_{\text{total}}, \quad (2)$$

and the probability that this is a behavior micro step taken by a particular actor  $\mathbf{i}$  is given by

$$\lambda_{\mathbf{i}}^{\text{beh}} / \lambda_{\text{total}}. \quad (3)$$

There may be heterogeneity in the activity of actors — some actors may change their network ties, or their behavior, more quickly than others. Such activity differences may be caused by individual properties (e.g., by sex differences) or by an existing network structure (e.g., by the number of ties an actor already has). We can incorporate such activity differences between actors by letting the parameters  $\lambda$  that govern the rate functions depend on actor attributes and network positions, as in Snijders (2001, 2005). In this paper, however, we limit the discussion to model specifications where both types of rate functions are constant across actors, and depend only on the periods between panel waves. Accordingly, from here onward, the lower index  $\mathbf{m}$  of a rate  $\lambda_{\mathbf{m}}^{\text{type}}$  will denote a time period,  $1 \leq \mathbf{m} \leq \mathbf{M}$ , not an individual anymore.

### 3.4. Modeling Mechanisms of Change

What happens in a micro step is modeled as the outcome of changes made by the actors. Micro steps can be of two kinds, corresponding to network changes or behavioral changes. For network changes, the micro step consists of the change of *one tie variable* by a given actor. Say  $\mathbf{x}$  is the current network and actor  $\mathbf{i}$  has the opportunity to make a network change. The next network state  $\mathbf{x}'$  then must be either equal to  $\mathbf{x}$  (i.e., keep the current situation) or deviate from  $\mathbf{x}$  in exactly one element in row  $\mathbf{i}$  (i.e., change the tie variable linking actor  $\mathbf{i}$  to one other actor). From these  $\mathbf{N}$  possible outcomes, it is assumed that  $\mathbf{i}$  chooses that value  $\mathbf{x}'$  for which  $\mathbf{f}_{\mathbf{i}}^{\text{net}}(\mathbf{x}, \mathbf{x}', \mathbf{z}) + \mathbf{e}_{\mathbf{i}}^{\text{net}}(\mathbf{x}, \mathbf{x}', \mathbf{z})$  is maximal. Here  $\mathbf{z}$  is the current vector of behavior scores,  $\mathbf{f}^{\text{net}}$  is a deterministic *objective function* that can be interpreted loosely as a measure of the actor's satisfaction with the result of the network decision (“what the

actor strives for when changing network ties”), and  $\mathbf{e}^{\text{net}}$  is a random disturbance term representing unexplained change. By making some convenient standard assumptions about the distribution of the random component (McFadden 1974; Pudney 1989), the choice probabilities can be expressed in multinomial logit shape,

$$\exp(\mathbf{f}_i^{\text{net}}(\mathbf{x}', \mathbf{z})) / \sum_{\mathbf{x}''} \exp(\mathbf{f}_i^{\text{net}}(\mathbf{x}'', \mathbf{z})), \quad (4)$$

where the sum in the denominator extends over all possible next network states  $\mathbf{x}''$ . This model for tie probabilities was used also by Snijders (2001) and Powell and colleagues (2005).

In a behavioral micro step, following again the parsimonious modeling principle that changes occur in the smallest possible steps, it is assumed that a given actor either *increments* or *decrements* his score on the behavioral variable by one unit, provided that this change does not step outside the range of this variable; it is also assumed that the score is not changed. The modeling is completely analogous to that of the network micro steps. If  $\mathbf{z}$  is the current vector of behavior scores for all actors, and  $\mathbf{i}$  is the actor allowed to change his behavior, let  $\mathbf{z}'$  denote the vector resulting from an allowed micro step. It is allowed that  $\mathbf{i}$  chooses that value  $\mathbf{z}'$  for which  $\mathbf{f}_i^{\text{beh}}(\mathbf{x}, \mathbf{z}, \mathbf{z}') + \mathbf{e}_i^{\text{beh}}(\mathbf{x}, \mathbf{z}, \mathbf{z}')$  is maximal, where now  $\mathbf{f}^{\text{beh}}$  is a different deterministic objective function that again can be interpreted as the actor’s satisfaction with the result of the behavioral decision, and  $\mathbf{e}^{\text{beh}}$  again is a random disturbance term representing unexplained change. By making appropriate assumptions about the distribution of the random component, choice probabilities can also be expressed in multinomial logit shape,

$$\exp(\mathbf{f}_i^{\text{beh}}(\mathbf{x}, \mathbf{z}')) / \sum_{\mathbf{z}''} \exp(\mathbf{f}_i^{\text{beh}}(\mathbf{x}, \mathbf{z}')), \quad (5)$$

where now the sum in the denominator extends over all possible next behavior states  $\mathbf{z}''$ . This type of model, for changes in an ordinal variable not combined with network ties, was used also by Chan and Munoz-Hernandez (2003).







It may be noted here that the model formulation in terms of choice models does in no way imply that we assume actors to be fully strategically rational, let alone that estimated parameters for the

objective functions express respondents' actual preferences. Because the states' evaluations in terms of the objective function are being compared immediately after a contemplated micro step, the actors' behavior lacks the strategic element of considering reactions from other actors. The model therefore can best be viewed as an instantiation of myopic rationality, following Luce's (1959) rationality axioms.

The focus of modeling is on the deterministic parts, defined by the objective functions  $f$ . A high degree of flexibility is achieved by modeling these as linear combinations of effects that express the dependence of network and behavior on each other as well as on externally given variables. The term *exogenous* will be used for effects depending on such external variables, while *endogenous* effects depend on the current values of the dependent variables (networks and behavior). For network changes, the objective function has the general shape  $f_i^{\text{net}}(\mathbf{x}, \mathbf{x}', \mathbf{z}) = \sum_h \beta_h^{\text{net}} s_h^{\text{net}}(\mathbf{i}, \mathbf{x}, \mathbf{x}', \mathbf{z})$ , where statistics  $s_h^{\text{net}}$  stand for the effects, weighted by parameters  $\beta_h^{\text{net}}$  whose size is determined by fitting the model to the data. Analogously, the objective function for behavioral changes has the form  $f_i^{\text{beh}}(\mathbf{x}, \mathbf{z}, \mathbf{z}') = \sum_h \beta_h^{\text{beh}} s_h^{\text{beh}}(\mathbf{i}, \mathbf{x}, \mathbf{z}, \mathbf{z}')$ . The statistics, or effects, must be defined on substantive grounds based on theory and field knowledge. They are arbitrary from the point of view of mathematical modeling, although in practice it is an advantage that they are not too complicated computationally. The most important network and behavior effects depend only on the new states  $\mathbf{x}'$  and  $\mathbf{z}'$ , not on the previous states  $\mathbf{x}$  and  $\mathbf{z}$ , and their weights  $\beta$  can be interpreted as the degree to which the actors have a tendency to change into a direction where the network-behavioral state has high values for these effects. A selection of possible endogenous network effects  $s_h^{\text{net}}$  is given in Table 2, while a similar selection of effects  $s_h^{\text{beh}}$  for behavioral changes is given in Table 3. These components are based on indicators of structural positions in networks that are of fundamental importance in social network analysis (Wasserman and Faust 1994). The second column of these tables contains the formulas of the statistics that express the respective effects.

In these tables, similarity of the behavioral scores of two actors  $i$  and  $j$  is defined as  $\text{sim}_{ij} = 1 - |z_i - z_j|/\text{range}_z$ , where the range of behavioral scores is defined as the maximum minus the minimum of observed values. By this definition, similarity is standardized to the unit interval,  $\text{sim} = 0$  indicating maximally dissimilar scores and  $\text{sim} = 1$  indicating identical (i.e., maximally similar) scores. For the calculations







TABLE 2  
Selection of Possible Effects for Modeling Network Evolution

Effect	Network Statistic	Effective Transitions in Network <sup>a</sup>	Verbal Description
1. Outdegree	$\sum_j x_{ij}$		Overall tendency to have ties
2. Reciprocity	$\sum_j x_{ij} x_{ji}$		Tendency to have reciprocated ties
3. Preferential attachment	$\sum_j x_{ij} \sqrt{\sum_h x_{ih} x_{hj}}$		Tendency to attach to popular others (with decreasing marginal sensitivity to alter's popularity)
4. Transitive triplets	$\sum_j x_{ij} \sum_h x_{ih} x_{hj}$		Tendency toward triadic closure of the neighborhood (linear effect of the number of indirect ties)
5. Transitive ties	$\sum_j x_{ij} \max_h (x_{ih} x_{hj})$	 <i>(number of intermediaries is irrelevant)</i>	Tendency toward triadic closure of the neighborhood (binary effect of indirect ties)
6. Actors at distance 2	$\sum_j (1 - x_{ij}) \max_h (x_{ih} x_{hj})$	 <i>(number of intermediaries is irrelevant)</i>	Tendency to keep others at social distance 2 (negative measure of triadic closure)

(Continued)



TABLE 2  
(Continued)

Effect	Network Statistic	Effective Transitions in Network <sup>a</sup>	Verbal Description
7. Balance	$\sum_j \mathbf{x}_{ij} \text{strsim}_{ij}$		Tendency to have ties to structurally similar others (structural balance)
8. 3-cycles	$\sum_j \mathbf{x}_{ij} \sum_h \mathbf{x}_{jh} \mathbf{x}_{hi}$		Tendency to form relationship cycles (negative measure of hierarchy)
9. Betweenness	$\sum_j \mathbf{x}_{ij} \sum_h \mathbf{x}_{hi} (1 - \mathbf{x}_{hj})$	 (no direct link from left to right actor)	Tendency to occupy an intermediary position between unrelated others (broker position)
10. Covariate alter	$\sum_j \mathbf{x}_{ij} (\mathbf{z}_j - \bar{\mathbf{z}})$		Main effect of alter's behavior (covariate determines popularity in network)
11. Covariate ego	$\sum_j \mathbf{x}_{ij} (\mathbf{z}_i - \bar{\mathbf{z}})$		Main effect of ego's behavior on tie preference (covariate determines activity in network)
12. Covariate similarity	$\sum_j \mathbf{x}_{ij} \text{sim}_{ij}$		Tendency to have ties to similar others (homophile selection on covariate, linear in score differences)

<sup>a</sup>In the *effective transitions* illustrations, it is assumed that the covariate is dichotomous and centered at zero; the coding is  $\circ$  = low score (negative),  $\bullet$  = high score (positive),  $\oplus$  = arbitrary score. The tie  $\mathbf{x}_{ij}$  from actor  $i$  to actor  $j$  is the one that changes in the transition indicated by the double arrow. Illustrations are not exhaustive.

TABLE 3  
Selection of Possible Network Effects for Modeling Behavioral Evolution

Effect	Network Statistic	Effective Transitions in Behavior <sup>a</sup>	Verbal Description
1. Shape: linear and quadratic	$(z_i - \bar{z})$ and $(z_i - \bar{z})^2$		<i>The two parameters together define a parabola shape of the objective function, allowing it to capture the basic shape of the observed distribution of the behavioral variable.</i>
2. Average similarity	$(\sum_j x_{ij} \text{sim}_{ij}) / (\sum_j x_{ij})$		Assimilation to neighbors' average behavior (small neighborhoods pull as much as big ones)
3. Sum of similarity	$\sum_j x_{ij} \text{sim}_{ij}$		Assimilation to neighbors' average behavior (size of neighborhood determines size of effect)
4. Average alters	$(\sum_j x_{ij} (z_j - \bar{z})) / (\sum_j x_{ij})$		Main effect of neighbors' average behavior (contagion/influence, but not necessarily assimilation)
5. Indegree × behavior	$(z_i - \bar{z}) \sum_j x_{ij}$		Effect of own popularity in the network on behavior
6. Outdegree × behavior	$(z_i - \bar{z}) \sum_j x_{ji}$		Effect of own activity in the network on behavior
7. Isolation × behavior	$(z_i - \bar{z})(1 - \max_j(x_{ij}))$		Effect of being isolated in the network on behavior

<sup>a</sup>In the effective transitions illustrations, it is assumed that the behavioral dependent variable is dichotomous and centered at zero; the color coding is ○ = low score (negative), ● = high score (positive), ⊕ = arbitrary score. Actor i is the actor who changes color z<sub>i</sub> in the transition indicated by the double arrows. Illustrations are not exhaustive.

of the statistics in the tables, similarity is further centered around the empirical average over all measurement points. Such centering reduces estimation difficulties caused by collinearity. Therefore, the covariates and behavioral variables are also centered (which, in this case, can be seen back in the formulas). The balance effect for network evolution (Davis 1963; Mizruchi 1993) contains a measure of structural similarity  $\text{strsim}_{ij} = \sum_{\mathbf{h}} (\mathbf{b} - |\mathbf{x}_{i\mathbf{h}} - \mathbf{x}_{j\mathbf{h}}|)$  that is analogous to similarity, where  $\mathbf{b}$  is a parameter used for standardization. In network terms, this may be regarded as a measure for structural equivalence regarding outgoing ties (Lorrain and White 1971). The tables can naturally give only a glimpse of the complexity and richness of modeling that becomes possible within the proposed framework and may be elaborated in future research.

### 3.5. *Integration of Model Components and Model Estimation*

The total model specification for network-behavioral coevolution consists of the first wave observations  $\mathbf{x}(\mathbf{t}_1)$  and  $\mathbf{z}(\mathbf{t}_1)$  as the initial state of the stochastic process, the rate functions defining the rates of occurrence of network or behavioral micro steps by specific actors as sketched above, and the choice probabilities for each possible micro step. As a whole, the model belongs to the class of *continuous time Markov chains* (e.g., Norris 1997). The description given above allows us to construct a computer simulation of this process and also to specify the so-called intensity matrix which is the mathematical characterization of the Markov chain process (cf. see Snijders et al. 2007).

For given sets of parameter values, the evolution model can be implemented as a stochastic simulation algorithm that can be used to generate network and behavioral data according to the postulated dynamic process. Since the model is a Markov chain, the simulation algorithm is defined by giving the step of a single change in the process. We start out at some network-behavior configuration  $(\mathbf{x}(\mathbf{t}), \mathbf{z}(\mathbf{t}))$ . First, a waiting time is drawn from the exponential distribution with parameter **(1)**, and the time parameter  $\mathbf{t}$  is incremented by this waiting time. The process stops when time exceeds the end time of the period. If it continues, using probabilities **(2)** and **(3)**, it is determined whether the next event is a network change or a behavior change, and who is the actor making the change. The change to be made is chosen according

to probabilities (4) or (5), respectively. The process repeats itself until the end of the period is reached. At that point, the resulting simulated network-behavior configuration can be evaluated.

The model is too complex to allow for closed-form calculations of probabilities, expected values, etc., which is why direct ways of parameter estimation such as maximum likelihood are not easily implemented. However, the simulation algorithm can be employed to determine parameter estimates as those values under which simulated and observed data resemble each other most closely. This idea is instantiated in the *method of moments estimation routine*, which is used in the empirical section of this paper. To define it, each model parameter  $\theta$  must be matched with a statistic  $S$  that can be evaluated on a given data set (be it simulated or observed), and which defines an estimation equation for this parameter by demanding that the expected value over simulations is equal to the observed value of the statistic. The vector of parameter estimates is obtained as the joint solution to the corresponding system of equations. For network-behavior coevolution models, Snijders and colleagues (2007) proposed the following four types of statistics for the four types of model parameters according to Table 1:

<i>Network rate, period <math>m</math></i>	$\theta = \lambda_m^{\text{net}}$	$S = \sum_{ij}  X_{ij}(t_{m+1}) - x_{ij}(t_m) $
<i>Behavior rate, period <math>m</math></i>	$\theta = \lambda_m^{\text{beh}}$	$S = \sum_i  Z_i(t_{m+1}) - z_i(t_m) $
<i>Network objective function effects</i>	$\theta = \beta_h^{\text{net}}$	$S = \sum_m \sum_i s_h^{\text{net}}(i, X(t_{m+1}), z(t_m))$
<i>Behavior objective function effects</i>	$\theta = \beta_h^{\text{beh}}$	$S = \sum_m \sum_i s_h^{\text{beh}}(i, x(t_m), Z(t_{m+1}))$

The expected value over simulations of each of these estimation statistics  $S$  can be understood as a function of the vector  $\theta$  of all model parameters, and it is typically monotone increasing in its “own” parameter. Under mild regularity conditions (nondegeneracy of the matrix  $D$  of partial derivatives of statistics  $S$  by parameters  $\theta$ ), parameters can be uniquely identified. Notice the “cross-lagged” combination of simulated data at the end of an observation period with observed data

at the beginning of the period, on which the estimation statistics for objective function parameters are based. Here it is ensured that selection and influence processes that result in the same cross-sectional patterns can be empirically distinguished, provided that the lagged statistic is reasonably stable over time.

The iterative procedure by which parameter estimates and standard errors are obtained is described in detail by Snijders (2001) and by Snijders and colleagues (2007), and it can be sketched as follows. The iterations start at a parameter value  $\hat{\theta}_0$ , and at step  $k$  they have reached the current trial parameter value  $\hat{\theta}_k$ . The process is then simulated according to this parameter value, yielding simulated statistics  $S_k^{\text{sim}}$ . After this simulation, the parameter is updated according to the equation  $\hat{\theta}_{k+1} = \hat{\theta}_k - \mathbf{a}_{k+1} \mathbf{D}_0^{-1} (S_k^{\text{sim}} - S^{\text{obs}})$ , where  $\mathbf{D}_0$  is an approximation of the matrix of partial derivatives of statistics  $S$  by parameters  $\theta$  evaluated at  $\theta_0$ , where  $S^{\text{obs}}$  is the observed vector of estimation statistics, and where  $\mathbf{a}_k$  is a sequence of numbers that approach zero at rate  $k^{-c}$ ;  $c$  is chosen from the interval  $0.5 < c < 1$  so as to obtain good convergence properties. The final estimate is defined as a tail average of the trial values,  $\hat{\theta} = \frac{1}{R} \sum_{r=1}^R \hat{\theta}_{r_0+r}$ , where the initial number of trials  $r_0$  is long enough for a suitable burn-in period, and  $R$  is large enough to obtain a stable estimate. Standard errors of the estimate  $\hat{\theta}$  are calculated as the square roots of the diagonal elements of the approximate covariance matrix of the estimator function  $\tilde{\theta}$ , evaluated at the estimate, as given by the equation  $\text{cov}_{\tilde{\theta}}(\tilde{\theta}) = \mathbf{D}_{\tilde{\theta}}^{-1} \Sigma_{\tilde{\theta}}(\mathbf{S}) \mathbf{D}_{\tilde{\theta}}$ . Here  $\Sigma_{\tilde{\theta}}(\mathbf{S})$  stands for the matrix of simulated covariance of the vector of estimation statistics, evaluated at the estimate, and  $\mathbf{D}$  is again the (approximate) derivative matrix, now evaluated at the estimate.

Parameter estimation for this type of model has been broadly categorized as “third generation problems” in applied statistics (Gouriéroux and Monfort 1996) and it is computationally intensive. Depending on the data set and the model, it is possible—but rare—that the algorithm does not converge in a satisfactory way. This happens for models that are complicated in the sense that there are too many parameters relative to the variation in the data, or when effects are highly correlated in the data. Nonconvergence may also be an indicator of model misspecification. In the large majority of cases, however, with data sets ranging between 30 and a few hundred actors, and when the amount of change between successive waves is not too large while the

amount of change between the first and last waves is large enough, our experience is that convergence results are good.

### 3.6. *Interpretation of Model Parameters*

As a consequence of the actor-based nature of modeling and the decomposition of observed transitions from one observation to the next as a sequence of unobserved small changes, special attention needs to be paid to the interpretation of the estimated model parameters. The parameters of the rate functions can be related directly to the speed of the evolution process—keeping in mind that they reflect frequencies of opportunities for change, not frequencies of actual change, and that actors who are near the optimum of their objective functions will have a smaller probability of utilizing their opportunity for change. The parameters of the objective functions, however, relate in a more indirect way to the observed global dynamics of network and behavior. From a perspective of agency, these functions can be regarded as satisfaction measures of the actors with their local network-behavioral neighborhood. At a less construing level, they can be thought of descriptively, as a summary expression of the behavioral rules that are likely to be followed by the actors, given the observed data. These objective functions, together with the current network-behavior configuration, imply specific global dynamics as emergent property of the individual changes, in which network actors are mutually constraining each other and mutually offering opportunities to each other in a feedback process. In order to understand how the estimated parameters of the objective functions relate to the global dynamics observed, the Markov property of the process model needs to be invoked. This property implies that corresponding to the parameters there is a stationary (equilibrium) distribution of probabilities over the state space of all possible network-behavior configurations. Because the data configuration observed in the first wave of the panel will often not be in the center of this equilibrium distribution, the model defines a nonstationary process of network-behavioral dynamics, starting at the first observation and then “drifting” toward those states that have a relatively high probability under the equilibrium distribution; for the mathematical principles, see Norris (1997), for example. The dynamics as well as the stationary distribution of all but the simplest cases of these models are too

complex for analytic calculations, but they can be investigated by computer simulation.

For the “immediate interpretation” of the parameters of the objective functions, it can be useful to consider the odds of somewhat idealized micro steps (this is similar to the interpretation of parameters obtained by logistic regression). Suppose an actor  $i$  is in a situation to make a network micro step, and suppose two alternative courses of action result in networks  $\mathbf{x}_A$  and  $\mathbf{x}_B$ . From the linear shape of the objective function and the multinomial logit probabilities for micro steps, the odds for these outcomes can be derived as  $\Pr(\mathbf{x}_A)/\Pr(\mathbf{x}_B) = \exp(\sum_h \beta_h^{\text{net}} [s_h^{\text{net}}(\mathbf{x}_A) - s_h^{\text{net}}(\mathbf{x}_B)])$ , in simplified notation. As can be seen from this formula, the odds depend on the degree to which the two networks differ on the actor-specific statistics  $s_h^{\text{net}}$  (see again Table 2), these differences being weighted by the model parameters. Likewise, if the actor is in a situation to make a behavioral micro step and is considering two alternative courses of action resulting in behavioral vectors  $\mathbf{z}_A$  and  $\mathbf{z}_B$ , the odds are given by  $\Pr(\mathbf{z}_A)/\Pr(\mathbf{z}_B) = \exp(\sum_h \beta_h^{\text{beh}} [s_h^{\text{beh}}(\mathbf{z}_A) - s_h^{\text{beh}}(\mathbf{z}_B)])$ .

By way of example, let us assume that in a simple model specification, the function  $f_i^{\text{net}}(\mathbf{x}, \mathbf{x}', \mathbf{z}) = -2.0 \sum_j x'_{ij} + 2.5 \sum_j x'_{ij} x_{ji} + 1.0 \sum_j x'_{ij} \text{sim}_{ij}$  was estimated as a typical network objective function, while the behavioral objective function was estimated as  $f_i^{\text{beh}}(\mathbf{x}, \mathbf{z}, \mathbf{z}') = -1.0(\mathbf{z}'_i - \bar{\mathbf{z}}) - 0.5(\mathbf{z}'_i - \bar{\mathbf{z}})^2 + 2.5(\sum_j x_{ij} \text{sim}'_{ij}) / (\sum_j x_{ij})$ , also quite typical. The primes indicate those elements in the formulas the values of which are under the control of actor  $i$  and may be changed in a micro step that is governed by the objective function in which they occur. The network objective function contains three effects: (1) the outdegree effect (with parameter estimate  $\beta_{\text{out}}^{\text{net}} = -2.0$ ), (2) the reciprocity effect (with parameter estimate  $\beta_{\text{rec}}^{\text{net}} = 2.5$ ), and (3) the similarity effect (with parameter estimate  $\beta_{\text{sim}}^{\text{net}} = 1.0$ ). In the behavioral objective function, the model contains three more effects: (1) an effect with two parameters determining the basic shape of the distribution of the variable, (2) one linear (with estimate  $\beta_{\text{lin}}^{\text{beh}} = -1.0$ ) and one quadratic (with estimate  $\beta_{\text{quad}}^{\text{beh}} = -0.5$ ) effect, and (3) an effect of average similarity to neighbors (with parameter estimate  $\beta_{\text{av.sim}}^{\text{beh}} = 2.5$ ). We now address the question of how these parameter values can be interpreted, starting with the network objective function.

The parameter attached to the outdegree effect in the network objective function has a negative sign, which indicates that observed

network densities are low. If the objective function were constant zero (i.e., if the outdegree parameter and all other parameters are zero), the network micro steps would, according to the odds formula above, lead in the long run to a network of density 0.5. In other words, 50% of all possible ties would be present in such equilibrium networks. Empirical densities in most social networks are much lower than 0.5, and therefore, the outdegree parameter (which models the general tendency of the actors to send out ties in the network, hence indirectly also the density of the network) is typically strongly negative. If we for the moment disregard the other parameters, the value of  $-2.0$  for the outdegree parameter means that upon an opportunity for change, the odds for any tie to be present versus absent are  $\exp(-2.0) \approx 0.135$ .

The reciprocity parameter accounts for the observed degree of reciprocity in the networks. If the objective function consisted only of the outdegree effect and if the reciprocity parameter was zero, the micro steps would in the long run lead to a network in which the incoming and outgoing ties are independent. In such a situation, reciprocated ties in sparse networks would be very unlikely. However, many sparse social relations, such as friendship, exhibit a relatively high degree of reciprocation. For modeling these relations, the reciprocity parameter (which models the general tendency of the actors to reciprocate incoming ties by sending back an outgoing tie) is typically strongly positive. The objective function value associated with a reciprocated tie is calculated by adding to the outdegree parameter value (for having the tie) the reciprocity parameter value (for the additional property of reciprocity), which amounts to a net value of  $-2.0 + 2.5 = 0.5$  for a reciprocated tie. Making use of the odds formula above, the odds of reciprocating an incoming tie versus not reciprocating it are  $\exp(0.5) \approx 1.65$ .

The positive similarity effect in our example indicates that actors tend to have ties to similar others rather than to dissimilar ones; the odds are *ceteris paribus* 2.72 for a tie to a maximally similar actor versus a maximally dissimilar actor. By including this effect, homophilous selection based on the behavioral variable can be modeled.

So far, the odds calculations referred only to the existence of a tie versus nonexistence, but they could also be more complex. For instance, the odds of reciprocating an incoming tie to a dissimilar actor versus creating a unilateral, unreciprocated tie to a similar one are  $\exp(0.5 + 2.0 - 1.0) \approx 4.48$ . As in logistic regression, the calculation of odds becomes more complex as the model has more components and



as one is willing to distinguish more variable configurations. We must keep in mind that all odds calculations are model-derived and refer to artificial comparisons, since they hold only under a *ceteris paribus* assumption. Accordingly, for determining their validity, they need to be considered in light of how the actual data look—here reflected in the empirical values of the statistics  $\mathbf{s}$ —and whether a specific odds calculation derived from an estimated model refers to situations that frequently occur in the data (then validity is less problematic) or not (then calculation of the odds in question may be an unwarranted extrapolation). In general, the more effects one is willing to consider (and estimate) simultaneously, the less analytically tractable the model-explained global dynamics get, and the more one needs to consult simulations for learning about what an estimated model means in terms of global network properties.

In the example above, we now consider the behavior objective function, which contains three more parameters. The first two are the linear and quadratic shape parameters, which model the shape of the long-term distribution of the behavior variable. Let us assume that the possible scores on the behavioral variable range from 1 to 5 and that the observed average over all time points is at  $\bar{z} = 3$ . The centered behavior variable then ranges from  $-2$  to  $+2$ . Disregarding the third parameter for the moment, the two shape parameters define a parabolic objective function with maximum at the centered behavior score of  $-1$ , which corresponds to value 2 on the original scale. This means that in the long run, the distribution of respondents' behavior scores will also be unimodal with a maximum at score value 2. Odds calculations can also be done for behavioral micro steps. For instance, comparing a move from the average score of 3 to the optimum score of 2 with the option of staying at score 3, the odds are  $\exp(-1.0[(2 - 3) - (3 - 3)] - 0.5[(2 - 3)^2 - (3 - 3)^2]) \approx 1.65$ , meaning that in direct comparison, the move down to 2 is more likely (62%) than the stay at 3 (38%).

The third parameter, positive average similarity, indicates a propensity of the actors to behave in the same manner as their friends do—that is, to assimilate to them. Consider the same actor as above with score 3 on the behavior, and assume he has five friends, four of which are scoring 3 or higher, and one of which is scoring 2 or lower. Then for the above comparison of staying at score 3 versus moving down to score 2, the actor would get more dissimilar to the four friends

scoring high while getting more similar to the one friend scoring low. Average similarity to friends would decrease by 0.5 (the sum of similarity changes to all actors) divided by 5 (the number of friends), and the new odds can be calculated by multiplying the odds from above with the new contribution  $\exp(6.0 \times (-0.5/5)) \approx 0.549$ , such that we altogether get odds of  $0.549 \times 1.65 \approx 0.90$  for moving down to 2 instead of staying at 3. Due to the additional effect of assimilation to friends, the binary probabilities now change to slightly favoring staying at 3 (52%) over moving down to score 2 (48%).

In the total model, both selection and influence effects were included in the same model specification (though in different parts). Therefore, the effects are controlled for each other—that is, they are statistically separated. In order to assess the empirical evidence for either effect, we need to take a closer look at the standard errors and test the hypothesis that the effect is nil. In the empirical part reported in the following section, we will address these issues in more detail. We will also give examples of simulation-based inference regarding global network dynamics in the shape of an investigation of the determinants of network autocorrelation.

#### 4. THE CO-EVOLUTION OF FRIENDSHIP AND SUBSTANCE USE

In this section, the use of the techniques introduced above will be demonstrated. In an example application, we investigate the interplay of friendship dynamics and the dynamics of substance use among adolescents, the substances studied being alcohol and tobacco. On both dimensions, network autocorrelation is a well-documented fact, and on both dimensions, influence as well as selection were advanced as explanatory mechanisms (Napier, Goe, and Bachtel 1984; Fisher and Bauman 1988; Ennett and Bauman 1994; Andrews et al. 2002). The purpose of the present investigation is to decide between the different underlying theories, for the social environment in which our data were collected, by assessing the strength of their corresponding mechanisms. By fitting actor-based models, we are able to overcome the three key issues we identified: the actor-based way of modeling ensures that dependencies in the data can be sufficiently accounted for, the simultaneous modeling of network and behavioral evolution ensures that selection

and influence can be controlled for each other as well as for other mechanisms of network and behavior change, and the continuous-time model controls for invisibility of changes between panel waves.

#### 4.1. *Questions Addressed*

The investigation addresses three main questions:

1. To what degree can influence and selection mechanisms account for the observed coevolution of substance use and friendship ties in our data?
2. Does the answer to this question differ between the use of tobacco and the use of alcohol?

Finally, in order to explicitly address the issue of separating selection and influence and quantify the amount of observed substance use similarity among friends, we ask:

3. Which amount of network autocorrelation on the substance use dimensions can be accounted for by selection mechanisms, by influence mechanisms, or by other ‘control’ mechanisms?

#### 4.2. *Data*

The data were collected in the Teenage Friends and Lifestyle Study (Pearson and Michell 2000; Pearson and West 2003). Tracing a year cohort at a secondary school in Glasgow, Scotland, friendship networks, smoking behavior, alcohol consumption, and other lifestyle variables were measured at three time points in successive academic years, starting in February 1995 when the pupils were 12–13 years old and ending in January 1997. The panel contained a total of 160 pupils, of which 150 were present in the first wave, 146 in the second, and 137 in the third. Social networks were assessed by asking pupils to name up to six friends from their year group. They were also asked about “adolescent issues” such as lifestyle, taste in music, smoking behavior, and alcohol and drug consumption. We focus here on the dynamics of smoking and alcohol consumption only; analyses of other variables in this study can be found in Steglich, Snijders, and West (2006, taste in music) and Pearson, Steglich, and Snijders (2006, cannabis use).

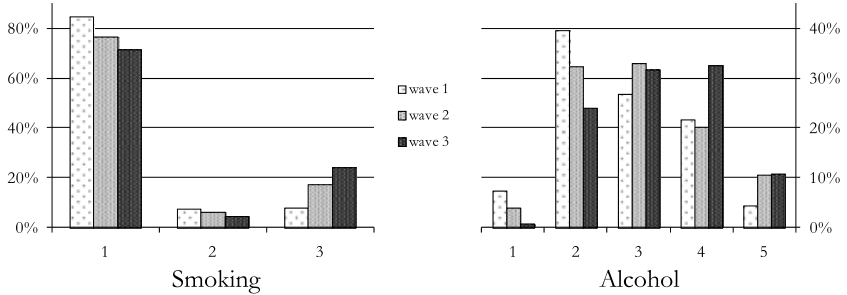


FIGURE 2. Observed distribution of substance use in the three waves.

The network variable of interest is the friendship relation between pupils. If pupil  $i$  reported pupil  $j$  as his friend, this was coded as  $x_{ij} = 1$ , otherwise  $x_{ij} = 0$ . The two dimensions of substance use are smoking  $z^{\text{smoke}}$ , which ranged from 1 (nonsmokers) to 3 (regular smokers, i.e., more than one cigarette per week), and alcohol consumption frequency  $z^{\text{alcohol}}$ , which ranged from 1 (not at all) to 5 (more than once a week). The distribution of these variables at the three measurement points can be seen in Figure 2.

To indicate the magnitude of network autocorrelation that occurs in these data, we consider the two most widely used standardized measures of network autocorrelation, the coefficients proposed by Moran (1948) and Geary (1954). The two coefficients measure slightly different aspects of the association between behavioral homogeneity and presence versus absence of a relational tie, as illustrated by the formulas, which is why it is useful to study them both in parallel to check validity of our results (Cliff and Ord 1981). The  $I$ -coefficient proposed by Moran is based on cross-products of behavioral scores of relational partners. Values close to zero indicate that relational partners are not more similar than one would expect under random pairing, while values close to one indicate a very strong network autocorrelation. Geary's  $c$ -coefficient is based on squared differences on the behavioral variable between relational partners. Values close to one are expected under random pairing, while values close to zero indicate strong behavioral homogeneity—in this sense, Geary's measure is an inverse indicator of network autocorrelation. In formulas, the coefficients are defined as follows:

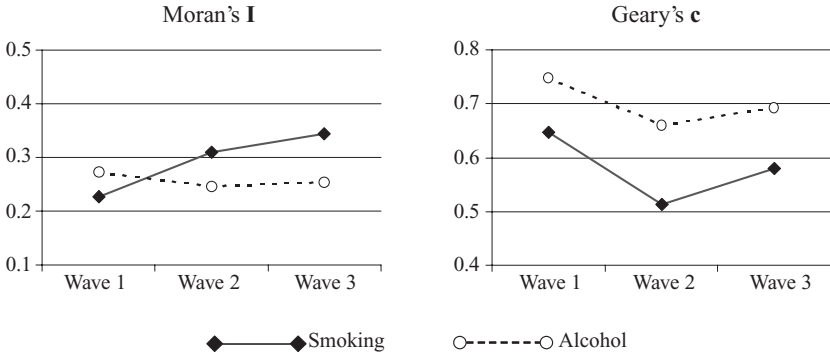


FIGURE 3. Observed network autocorrelation.

$$\mathbf{I} = \frac{n \sum_{ij} x_{ij}(z_i - \bar{z})(z_j - \bar{z})}{\left(\sum_{ij} x_{ij}\right) \left(\sum_i (z_i - \bar{z})^2\right)}, \quad \mathbf{c} = \frac{(n-1) \sum_{ij} x_{ij}(z_i - z_j)^2}{2 \left(\sum_{ij} x_{ij}\right) \left(\sum_i (z_i - \bar{z})^2\right)} \quad (6)$$

The observed values of  $\mathbf{I}$  and  $\mathbf{c}$  are visualized in Figure 3. As expected, on both behavioral dimensions and at all three measurement points, there is considerable network autocorrelation, i.e.,  $\mathbf{I} > 0$  and  $\mathbf{c} < 1$ .

For some initial impressions of selection and influence, consider Figures 4 and 5. In Figure 4, tie change patterns in a period are cross-tabulated with similarity at the beginning of the period (the figure renders results pooled over both periods). We can see that among the pupil pairs connected through a tie at the beginning of the period, tie stability is the more likely the higher their initial similarity (possible deselection of dissimilars). Also among initially unconnected pupil pairs, the more similar the pupils were in the beginning (possibly homophilous selection), the more likely that new ties would be created. Figure 5 depicts how pairs of actors change their behavior relative to each other, setting off pairs with a friendship tie against pairs without a friendship tie at the beginning of the period. Four patterns of behavior change are distinguished: actors can *approach* each other on the behavioral dimension, *distance* themselves, or keep their current similarity. This third group is subdivided by a median split of similarity scores into those pairs of actors who keep their similarity at a high level (*stay close*) or at a low level

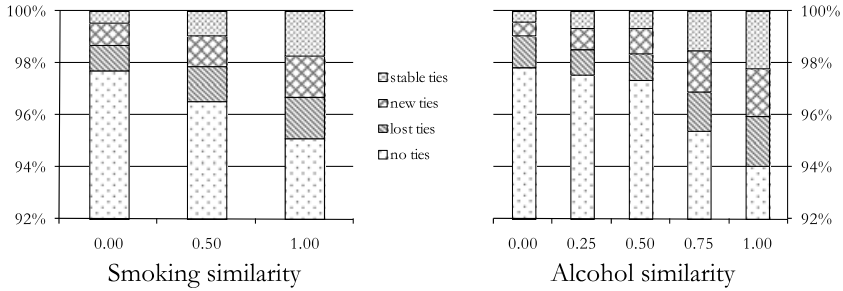


FIGURE 4. Tie change patterns by initial behavior.

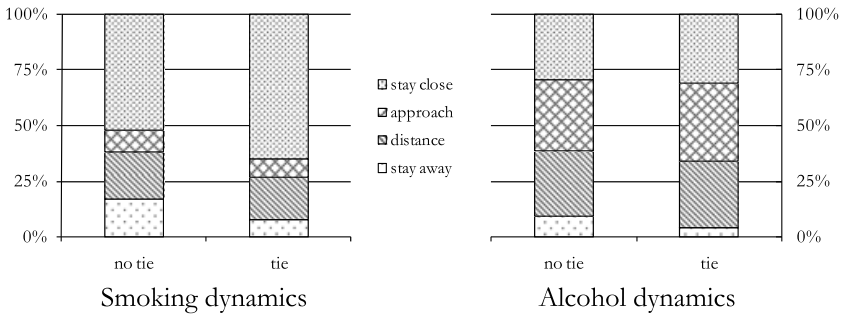


FIGURE 5. Behavior change patterns by initial tie status.

(*stay away*). What can be seen is that for both behaviors alike, presence of a tie at the beginning of a period enhances the odds of staying similar versus distancing themselves, and reduces the odds of staying dissimilar versus approaching each other—possibly caused by social influence. It should be noted, though, that the descriptive patterns of these figures do not allow a conclusion about the underlying mechanisms of network and behavior change, for the reasons outlined earlier.

Next to substance use, some control variables are included in the analyses. The individual control variables included are sex (1 = male, 2 = female), birth year (minus 1900), the amount of money the pupils had at their disposal (measured in units of 10 British £ per week), and whether or not they were currently engaged in a romantic relationship (1 = no, 2 = yes). Furthermore, as potential predictors for substance use, parental smoking and sibling smoking are included (both coded 1 = non-smoking, 2 = smoking). As an example for a dyadic covariate, the classmate-relation (“being in the same class”) was included.

TABLE 4  
Summary of Expectations About Model Parameters

<i>Verbal Description</i>	<i>Model Parameter</i>	<i>Sign</i>
<b>(a) Rules of Network Change (Friendship)</b>		
1. The density of friendship networks is low.	outdegree	–
2. Friendship nominations tend to be reciprocated.	reciprocity	+
3. Popular pupils attract friendship nominations.	preferential attachment	+
4. Friendship networks tend to display triadic closure.	transitive ties	+
	distance-2	–
5. Friends tend to be structurally equivalent.	balance	+
6. Friendship is more likely between classmates.	classmate	+
7. There is homophily according to sex.	sex similarity	+
8. Older students are less invested in school networks.	birth year ego/alter	+
9. Money attracts friends.	money alter	+
10. There is homophily according to money.	money similarity	+
11. Romantic relations reduce investment in the network.	romantic ego/alter	–
12. There is homophily according to romantic relations.	romantic similarity	+
13. There is homophily according to substance use.	behavior similarity	+
<b>(b) Rules of Behavioral Change (Substance Use)</b>		
14. Own substance use is assimilated to friends' use.	average similarity	+
15. Older pupils use more substance.	birth year	–
16. Pupils whose parents/siblings smoke use more substance.	parent/sibling smoking	+
17. Pupils who use one substance also use the other.	other substance use	+
18. Pupils with more money use more substance.	money	+
19. Pupils involved in romantic relations use less substance.	romantic	–

#### 4.3. *Model*

We now fit to the data a series of actor-based models as introduced in the previous section. Here, a description of the most comprehensive of these models is given (the “full model” to be later seen in Tables 5 and 6), of

TABLE 5  
Estimates and Standard Errors for Friendship Evolution

	Full Model			Control Model			Straw Man Model			Trend Model		
	Estimate	Standard Error	p-Value	Estimate	Standard Error	p-Value	Estimate	Standard Error	p-Value	Estimate	Standard Error	p-Value
Network Dynamics												
Rate period 1	14.16	(1.63)		14.05	(1.43)		10.14	(0.70)		6.27	(0.33)	
Rate period 2	11.57	(1.30)		11.48	(1.06)		9.06	(0.69)		5.63	(0.31)	
Outdegree (density)	-2.53	(0.22)	<0.001	-2.64	(0.17)	<0.001	-2.68	(0.06)	<0.001	-1.88	(0.03)	<0.001
Reciprocity	1.55	(0.10)	<0.001	1.63	(0.10)	<0.001	2.44	(0.08)	<0.001	—	—	—
Preferential attachment	0.17	(0.09)	0.041	0.23	(0.08)	0.003	—	—	—	—	—	—
Transitive ties	0.55	(0.12)	<0.001	0.57	(0.12)	<0.001	—	—	—	—	—	—
Number of actors at distance 2	-0.35	(0.09)	<0.001	-0.37	(0.09)	<0.001	—	—	—	—	—	—
Balance	0.13	(0.02)	<0.001	0.13	(0.02)	<0.001	—	—	—	—	—	—
Classmate	0.01	(0.07)	0.905	-0.01	(0.07)	0.934	—	—	—	—	—	—
Sex alter	-0.11	(0.09)	0.189	-0.14	0.08	0.069	—	—	—	—	—	—
Sex ego	0.10	(0.10)	0.327	0.09	0.09	0.304	—	—	—	—	—	—
Sex similarity	0.62	(0.08)	<0.001	0.64	(0.07)	<0.001	0.97	(0.08)	<0.001	—	—	—
Birth year alter	0.10	(0.08)	0.220	0.12	(0.08)	0.144	—	—	—	—	—	—
Birth year ego	0.27	(0.10)	0.009	0.30	(0.10)	0.002	—	—	—	—	—	—
Birth year similarity	0.19	(0.16)	0.226	0.22	(0.18)	0.217	—	—	—	—	—	—
Money alter	0.10	(0.05)	0.050	0.09	(0.05)	0.048	—	—	—	—	—	—
Money ego	-0.05	(0.07)	0.474	-0.02	(0.06)	0.717	—	—	—	—	—	—
Money similarity	0.96	(0.28)	<0.001	0.98	(0.30)	0.001	—	—	—	—	—	—

(Continued)



TABLE 5  
(Continued)

Network Dynamics	Full Model			Control Model			Straw Man Model			Trend Model		
	Estimate	Standard Error	p-Value	Estimate	Standard Error	p-Value	Estimate	Standard Error	p-Value	Estimate	Standard Error	p-Value
Romantic alter	-0.04	(0.08)	0.613	-0.06	(0.08)	0.425	—	—	—	—	—	—
Romantic ego	-0.10	(0.10)	0.337	-0.12	(0.10)	0.241	—	—	—	—	—	—
Romantic similarity	0.13	(0.08)	0.107	0.15	(0.07)	0.033	—	—	—	—	—	—
Alcohol alter	0.03	(0.07)	0.688	—	—	—	—	—	—	—	—	—
Alcohol ego	0.14	(0.09)	0.136	—	—	—	—	—	—	—	—	—
Alcohol similarity	0.73	(0.39)	0.061	—	—	—	1.05	(0.43)	0.014	—	—	—
Smoking alter	-0.05	(0.08)	0.501	—	—	—	—	—	—	—	—	—
Smoking ego	-0.10	(0.11)	0.394	—	—	—	—	—	—	—	—	—
Smoking similarity	0.27	(0.16)	0.086	—	—	—	0.28	(0.13)	0.024	—	—	—

TABLE 6  
Estimates and Standard Errors for the Evolution of Substance Use

Alcohol Dynamics	Full Model			Control Model			Straw Man Model			Trend Model		
	Estimate	Standard Error	p-Value	Estimate	Standard Error	p-Value	Estimate	Standard Error	p-Value	Estimate	Standard Error	p-Value
Rate period 1	1.70	(0.40)		1.39	(0.26)		1.70	(0.35)		1.32	(0.22)	
Rate period 2	2.64	(0.53)		2.52	(0.45)		2.69	(0.53)		2.40	(0.44)	
Shape: linear	0.41	(0.14)	0.004	0.37	(0.11)	0.001	0.45	(0.14)	0.001	0.42	(0.11)	<0.001
Shape: quadratic	0.01	(0.11)	0.926	-0.33	(0.08)	<0.001	0.07	(0.11)	0.511	-0.28	(0.07)	<0.001
Average similarity	6.70	(2.18)	0.002	—			7.32	(2.26)	0.001	—		
Smoking	0.05	(0.20)	0.821	0.17	(0.16)	0.285	—			—		
Sex	0.06	(0.23)	0.801	0.13	(0.19)	0.498	-0.07	(0.20)	0.731	—		
Birth year	0.29	(0.30)	0.342	0.15	(0.24)	0.539	—			—		
Money	0.23	(0.15)	0.134	0.24	(0.12)	0.049	—			—		
Romantic	-0.45	(0.32)	0.151	-0.25	(0.26)	0.344	—			—		

(Continued)

TABLE 6  
(Continued)

	Full Model			Control Model			Straw Man Model			Trend Model		
	Estimate	Standard Error	p-Value	Estimate	Standard Error	p-Value	Estimate	Standard Error	p-Value	Estimate	Standard Error	p-Value
Smoking Dynamics												
Rate period 1	5.09	(2.90)		4.53	(2.66)		5.11	(1.74)		3.82	(0.99)	
Rate period 2	4.26	(1.31)		4.59	(1.81)		4.00	(1.25)		4.08	(1.26)	
Shape: linear	-2.61	(0.42)	<0.001	-3.41	(0.45)	<0.001	-2.57	(0.42)	<0.001	-3.42	(0.37)	<0.001
Shape: quadratic	2.62	(0.31)	<0.001	2.71	(0.33)	<0.001	2.67	(0.29)	<0.001	2.80	(0.32)	<0.001
Average similarity	2.63	(1.06)	0.014	—			2.82	(1.06)	0.008	—		
Alcohol	-0.03	(0.18)	0.868	0.05	(0.16)	0.750	—			—		
Sex	-0.01	(0.28)	0.981	0.24	(0.20)	0.227	-0.03	(0.25)	0.897	—		
Birth year	0.02	(0.29)	0.954	-0.11	(0.23)	0.653	—			—		
Parent smoking	-0.25	(0.26)	0.345	-0.17	(0.21)	0.420	—			—		
Sibling smoking	0.24	(0.37)	0.515	0.15	(0.32)	0.637	—			—		
Money	0.08	(0.17)	0.649	0.11	(0.15)	0.455	—			—		
Romantic	0.23	(0.27)	0.389	0.25	(0.23)	0.274	—			—		

which all the other models are simplifications. The model for friendship dynamics is first discussed by specifying the network objective function and then the behavior objective functions are sketched, one for each type of substance use, modeling behavior dynamics.

An overall tendency to form network ties, expressed by the out-degree effect, forms the basis of the network objective function. It is expected to be negative due to the low density of the observed networks (see the first row in Table 2). Endogenous determinants of network evolution are properties by which the network, in a feedback process, affects its own dynamics. We include several endogenous network effects: the tendency to reciprocate friendship nominations (reciprocity effect, expected positive, second row in Table 2), the tendency to nominate popular (i.e., frequently chosen) others as friends (“preferential attachment,” expected positive, third row), and tendencies toward network closure and structural balance. Network closure means that direct friendship relations are more likely to those others who also indirectly are friends—that is, who are linked via a third pupil. This could be operationalized by three different effects, described in rows 4–6 of Table 2, of which we chose rows 5 and 6. Structural balance is a well-known concept dating back to Heider (1958). In our operationalization (row 7), we measure it by the tendency to be friends with pupils who share the same other friends: the more similar the friendship selection patterns of two pupils (in network jargon: the more structurally equivalent they are), the more likely they are to also choose each other as friends. Other endogenous network effects that could be included in a model, but which we omitted for lack of substantive theory in the present case, are related to hierarchization and brokerage. An aversion to forming three-cycles indicates the presence of an informal hierarchy in the network and could be modeled by including the corresponding effect (row 8). The betweenness effect (row 9) measures a tendency to assume a broker role in the friendship network by establishing indirect links between network regions that are not directly connected. For friendship networks, it can be expected that there is no hierarchy and that brokerage plays little role, hence our omission of these effects.

The dyadic variable  $w_{ij}^{\text{classmate}}$ , indicating whether two pupils belong to the same class in the cohort, is expected to have a main (positive) effect on friendship, which is included in the model as a statistic  $\sum_j x_{ij} w_{ij}^{\text{classmate}}$  (not visualized in Table 2). The individual variables sex, birth year, money, and involvement in romantic relations can be

expected to affect friendship evolution in three ways: as a main effect on alter attractiveness (row 10), as a main effect on network activity of ego (row 11), and as a similarity effect of homophilous selection (row 12). Finally, in order to assess the effects of selection based on substance use, also taking into account the variables smoking and alcohol use, the ego-, alter-, and similarity effects are included in the model specification. In part (a) of Table 4, reasonable expectations about the signs of some effects are given. Because for our purpose most effects play only the auxiliary role of control variables, we do not discuss them further here. Nonetheless, their inclusion is crucial for our aim of explaining network autocorrelation and properly separating selection and influence, as they capture the “alternative generating mechanisms” that we identified as a key issue to take into account.

The basis for modeling the evolution of the substance use dimensions are two parameters expressing the shape of the variables' distributions in the long run. Looking again at Figure 2, the distribution of the smoking variable is U-shaped, while the alcohol consumption variable is distributed according to an inverse U-shape. In the objective function, these different shapes can be expressed by the linear and quadratic effects of substance use (see the first row in Table 3). The sign of the quadratic term here indicates whether the objective function is a parabola opening upward (a positive sign, implying a U-shaped distribution as observed for the smoking variable) or downward (a negative sign, implying an inverse U-shaped distribution as observed for the alcohol variable).

For measuring social influence on substance use, different operationalizations are possible, such as an effect of average similarity to neighbors (second row), an effect of similarity that is not averaged but is added over neighbors (third row), or a main effect of peers' average substance use (row 4). Which of those, if any, is best-suited for modeling social influence remains in principle an empirical question. Based on score-type tests (Schweinberger 2004) that provide a statistical basis for choosing between such alternative operationalizations, we here chose the average similarity effect, as it fit best with our data. It also corresponds most closely to the intuitive idea that each pupil is influenced by his group of friends in about the same way (Cohen 1977).

Individual variables  $v$  (e.g., sibling smoking) are included as main effects in the behavior objective functions, i.e., with statistics  $(z_i - \bar{z})(v_i - \bar{v})$  (not visualized in Table 3), reflecting (if the coefficient

is positive) that higher values of  $\mathbf{v}$  will tend to lead to higher values of  $\mathbf{z}$ . In the same way, the main effect of the substance use dimensions on each other can also be included. Part (b) in Table 4 gives an overview of reasonable expectations concerning the specific model parameters for the substance use dynamics.

#### 4.4. Results

We estimated models of four degrees of complexity for the coevolution of the friendship network with smoking behavior and alcohol consumption. The trend model postulates purely random change of network and behavior, and the control model contains all effects except selection and influence related to the behavioral variables. These models will further serve as benchmarks for explaining observed network autocorrelation in the discussion below. The full model is the one in which the hypotheses from Table 4 are tested. The straw man model, finally, is a deliberately mis-specified model in which selection and influence are modeled in a “naive” way—that is, not controlling for most of the effects in the control model. This straw man will serve for illustrating the dangers of model mis-specification, in terms of failure to control for alternative generating mechanisms. Tables 5 and 6 give parameter estimates for the models discussed below.

First the network evolution part is considered (reported in Table 5) and then the evolution of the two behavioral dimensions smoking and alcohol consumption (reported in Table 6). The parameters of main interest for friendship dynamics are those that operationalize friend selection based on substance use. The ego- and alter effects of this type do not reach significance, suggesting that there is not a main effect of substance use on either friendship nominating activity or on popularity in the network. The homophily-operationalizing parameters have the predicted sign but are only marginally significant (parameters *alcohol similarity* and *smoking similarity* in row 13 of Table 4). This indicates that the descriptive evidence for homophilous selection according to substance use (Figure 4) cannot be fully attributed to a genuine homophilous selection mechanism, but in part also results from a combination of mechanisms expressed by the other parameters. A look at the straw man model’s estimates confirms this impression: when not comprehensively controlling for alternative generating

mechanisms, much stronger test results for homophily based on substance use are found—a result not to be taken seriously. Effects that are more likely responsible for the appearance of significant homophily in the straw man model are those that are significant in the full model but not included in the straw man model.

The inherent tendency of friendship to be reciprocated, a tendency toward seeking popular others as friends, network closure, and structural balance, together constitute a highly significant endogenous explanation of observed friendship dynamics. The corresponding parameters' signs confirm our expectations formulated in Table 4 (parameters *outdegree* through *balance* in Table 5; expectations 1 through 5 in Table 4). Also among the covariate effects, several expectations can be confirmed, such as strong homophily mechanisms based on sex and money (parameters *sex similarity* and *money similarity*; expectations 7 and 10 in Table 4), a marginal homophily effect of having a romantic relation (parameter *romantic similarity*; expectation 12), higher attractiveness of rich versus poor friends (parameter *money alter*; expectation 9), and a lower network presence of older pupils, as expressed in the positive ego effect of birth year (parameter *birth year ego*; expectation 8; the alter effect of birth year fell short of significance). Expectations that cannot be confirmed are those about lower friendship involvement of students with romantic relations (parameters *romantic ego & alter*; expectation 11), and—surprisingly—also the classmate effect (parameter *classmate*; expectation 6). This may be due to the fact that in the school studied, teaching at the time did not take place in a classroom context, but in courses of varying composition. The class was a largely administrative unit and did not necessarily reflect opportunity for social interaction.

The behavioral evolution part of the models consists of the submodels for smoking dynamics and alcohol dynamics, with the results reported in Table 6. Here, the focus of interest is on social influence mechanisms (expectation 14 in Table 4), and these are clearly confirmed by our analyses—the *average similarity* parameter indicates strong assimilation effects on both substance use dimensions, in the full model and in the straw man model alike. The overall consistency between these two models indicates that the mechanisms of behavior change that we control for seem to be less related to social influence processes.

The two *shape* parameters of the smoking submodel confirm the expectation of a U-shaped basis for the objective function, which holds

for all estimated models alike. This suggests that this empirical shape (see again Figure 2) does not result from influence mechanisms alone, but might express a “universal” property of smoking. There hardly seems to be a “middle ground”, and pupils are either nonsmokers or regular smokers, possibly reflecting addiction effects. Social influence exists but is unlikely to override this basic pattern. For alcohol consumption, the expected inverse U-shape is only found back in the simpler models (trend and control) but not in the models containing the assimilation effect. Apparently, the inverse U-shape of the empirical distributions of alcohol use in Figure 2 can be better explained as resulting from assimilation to friends’ alcohol consumption than from a universal, network-independent shape of the distribution. Recall in this context that the modeling conditions on the first observation. The inverse U-shaped distribution of alcohol scores that already exists at this first observation moment is therefore not explained, but its persistence over time is sufficiently explained as resulting from assimilation to network neighbors. Finally, none of the included covariates (expectations 15–19 in Table 4) significantly explains the evolution of either substance use dimension, stressing the point that alternative generating mechanisms seem not to play as strong a role on the substance use dimensions as they did on the network dimension.

#### 4.5. *Conclusions from the Analyses*

Proceeding from these results, we can now draw conclusions about which of the mentioned theories are able to account for the observed coevolution dynamics. The dynamics of smoking behavior and alcohol consumption follow roughly the same patterns: for both substances, the parameters for homophilous selection and for assimilation are significant. Based on significance of effects, the results in the first place can be viewed as a confirmation of social influence theories. However, the evidence for alcohol consumption being a “social phenomenon” in the sense of pupils being influenced by their friends is stronger than for smoking, which can be derived from calculation of odds as well as from parameter significance ( $p = 0.002$  versus  $p = 0.014$ ). A micro step on the alcohol dimension toward the friends’ average alcohol use corresponds to a change of  $1/\text{range}(\text{alcohol}) = 1/4$  in the average similarity statistic, so the relevant odds of the effect are  $\exp(6.7/4) = 5.3$ . For the



corresponding effect in smoking dynamics, the odds are  $\exp(2.63/2) = 3.7$ . Also, the quadratic shape parameter for smoking remains significant and is positive in the full model, unlike for alcohol, which suggests that an important part is played by the addictive nature of smoking, which may override social influence.

Selection of friends may well lead to friendship between pupils of similar substance use, as can be seen from the straw man model ( $p = 0.014$  for alcohol,  $p = 0.024$  for smoking). However, this by and large seems not to result from genuine homophily according to these substance use patterns, but from other, significant selection patterns that are controlled for, as the significance drops to marginal size ( $p = 0.061$  for alcohol,  $p = 0.086$  for smoking) when we control for a broad array of alternative mechanisms of friendship formation, as is done in the full model. So, in the second place, the results can be seen as supporting the argument in Feld (1981, 1982) about similarity resulting from context effects rather than occurring independently from those. In order to show the validity of this argument, however, it needs to be investigated whether these significant alternative mechanisms do indeed imply similarity on the substance use dimensions—that is, do contribute to network autocorrelation. To test this, we now conduct a simulation study.

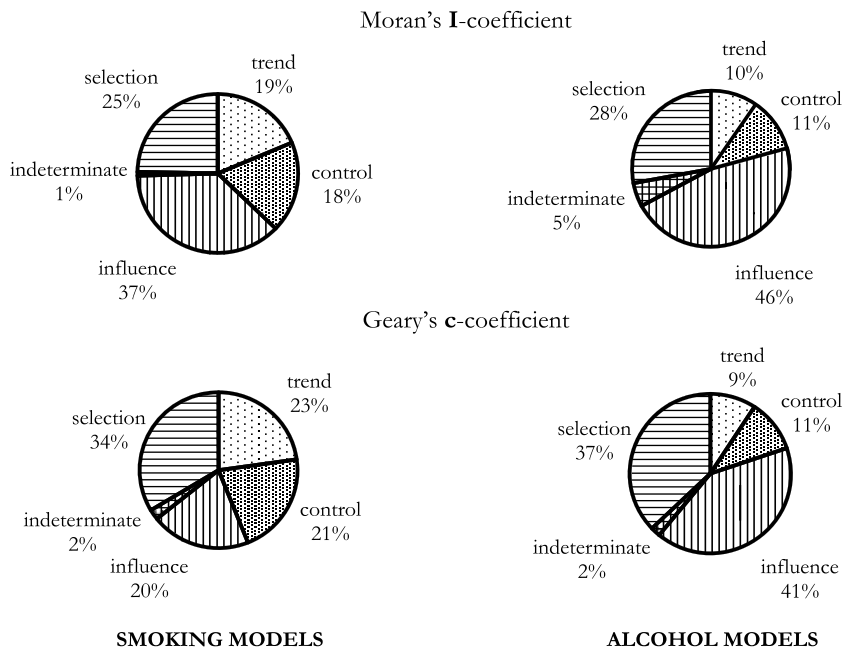
#### 4.6. *A Quantitative Assessment of the Determinants of Network Autocorrelation*

We now address the issue of measuring the “amount” of network autocorrelation allocated by a fitted model to the different generative processes—that is, to selection, influence, and the control processes unrelated to these two main effects. Two alternative measures of network autocorrelation are considered, Moran’s *I*-coefficient and Geary’s *c*-coefficient, as introduced above. Our overall conclusions from these analyses should not depend on the specific measure chosen. We do, however, expect minor quantitative differences since the calculation of Geary’s measure is based on distances on the substance use scale, while for Moran’s coefficient, it is based on cross-products.

The procedure followed is similar in underlying intuition to Kandel’s (1978, p 433) quantification proposal, which in a footnote she attributes to James Coleman. Also Fisher and Bauman’s (1988)

proposal follows the same ideas, so our own proposal in principle is nothing new. Kandel's procedure was as follows: two simple models are first fitted to the data, one operationalizing the selection process "alone," the other the influence process "alone". Both models are then used for calculating expected network autocorrelation, and this calculated value indicates the magnitude of the effect considered. Our implementation differs from those by Kandel and by Fisher and Bauman mainly by the fact that we use a model where the selection and influence effects are mutually controlled for each other and for other mechanisms. We thus evaluate the observed network autocorrelation, the network autocorrelation implied by our fitted model (explained autocorrelation), and the values implied by simpler models, in which either selection, or influence, or both, are "switched off." For this purpose, we combine results from the full and the control model. In addition, in order to assess the amount of network autocorrelation that can be attributed to alternative ("control") mechanisms, we consider implications of the trend model. In this model, both the selection and influence effects as well as the control effects are "switched off", enabling us to improve on a second major criticism we had of the earlier studies—namely, their neglect of assessing the impact of alternative mechanisms. The resulting allocation of explained network autocorrelation according to generating mechanisms can be seen in Figure 6. In these diagrams, it needs to be understood that the pie slices labeled "selection" cover substance-use based selection only—that is, homophily, ego,- and alter-effects based on substance use. Other selection effects, such as closure or covariate-based homophily, are allocated to the "control" slices.

The percentages in this figure were obtained in the following way. The trend model, the control model, and the full model as well as two hybrid models were used for *simulating* in continuous time network-behavioral coevolution trajectories that follow the rules extracted from the data. Each such trajectory starts out at the first observation of network and behavior at  $t_1$ . In the simulations, when time reaches the moment  $t_2$  of the second observation, the autocorrelation measure is evaluated on the simulated data. The same procedure is repeated for all periods until the time reaches the last observation moment  $t_M$  (here  $M = 3$ ). This way, observed autocorrelation measures can be related to a distribution of simulated autocorrelation measures, and different models can be compared on their implied distributions of network autocorrelation measures. For brevity, we report only across-period



**FIGURE 6.** Model-based decomposition of network autocorrelation. Slice sizes correspond to percentages of the coefficient size difference between average predictions of null and full models that through simulations could be allocated to different coevolution mechanisms.

averages on the network autocorrelation measures. Thus, the variables studied in Figure 6 are Moran's I-coefficient and Geary's c-coefficient, averaged over observation moments  $t_2$  and  $t_3$ .

The calculations rely on 1000 independent trajectories of the network-behavioral coevolution process of five models. The main model of interest here is the one in which, next to the control effects, both the main influence effect and the main selection effect were estimated from the data (the full model). The model without influence or selection, in which the network and the two behavioral dimensions evolve independently, is the control model. To assess the impact of the control effects on the network autocorrelation, the trend model is estimated. It contains only the outdegree parameter in the network part and the two shape parameters in the behavioral parts, and yields network autocorrelation values that depend on universal trend together with the initially observed state. These three data-fitted models are complemented by

TABLE 7  
Model-Based Simulated Network Autocorrelation

Model	Smoking		Model	Alcohol	
	Moran	Geary		Moran	Geary
Null	-0.007	1.000	Null	-0.007	1.000
Trend	0.046 (0.049)	0.908 (0.067)	Trend	0.024 (0.055)	0.968 (0.071)
Control	0.097 (0.064)	0.823 (0.084)	Control	0.057 (0.066)	0.930 (0.082)
No influence	0.166 (0.064)	0.680 (0.082)	No influence	0.143 (0.067)	0.799 (0.077)
No selection	0.201 (0.075)	0.733 (0.082)	No selection	0.200 (0.079)	0.785 (0.083)
Full	0.272 (0.076)	0.598 (0.083)	Full	0.301 (0.082)	0.648 (0.077)
Observed	0.310	0.542	Observed	0.247	0.696

two “cross-combined” hybrid models. In one of these, the network part is taken from the full model while the behavioral parts are taken from the control model, and in the other one, the combination is reversed. The idea behind this cross-combination is that, in order to identify the magnitude of network autocorrelation explained by only the main selection effect, we wish to compare network autocorrelation under the full model (where selection is controlled for influence) with network autocorrelation under a model where no selection occurs, while influence resembles that under the full model, and all control effects in the network part fit the data best.

Table 7 renders averages and standard deviations of the network autocorrelation coefficients over the 1000 simulated network trajectories per model, next to the expected value under permutations of the actors (row “null”) and the observed value. The numbers refer to the coefficients’ average over  $t_2$  and  $t_3$ , the time points at the end of the two periods in which unobserved change is simulated. What needs to be noticed first is that the values for the full model do not perfectly fit the observed values. For smoking, the model-predicted values are less extreme than observed, while for alcohol consumption, it is the other way round. This suggests that factors that lie outside the scope of our models probably enhance smoking autocorrelation, while they probably reduce alcohol autocorrelation.

Table 7 clearly shows that the less complex a model is, the less network autocorrelation it implies. Taking the difference between the full model’s predictions and the null expectations under independence as 100% (“all the model explains”), the values of this table were transformed into percentages to arrive at the diagrams in Figure 6. The models trend, control and full, are hierarchically nested, which means that the coefficients can be linearly transformed into percentages. The

other two models, in which either selection or influence are switched off, are nested in-between the control and the full model but not within each other. Therefore, the allocation of percentage values to influence and homophilous selection can be done in two ways: by considering the increase of the predicted coefficient upon including the effect, compared to the control model, or by considering the decrease of the predicted coefficient upon excluding the effect, compared to the full model. The resulting discrepancy of both calculations is rendered as “indeterminate” in the diagrams.

As suspected, trend and control effects are responsible for a considerable part of the observed network autocorrelation. More than one-third of the explained autocorrelation on smoking is epiphenomenal—that is, resulting from processes other than substance use based selection and the social influence of school friends. For alcohol consumption, the role of these effects is much weaker, approximately one-fifth of the total explained autocorrelation. The fractions that are allocated to influence and selection processes show two main results. On the one hand, there is a systematic difference between substance use dimensions. For alcohol use, the contribution of influence effects relative to selection effects is stronger than for tobacco use, where selection plays a more important role. On the other hand, there is a systematic difference between the two autocorrelation coefficients. Moran’s **I** identifies an overall stronger role of influence mechanisms than does Geary’s **c**. For smoking, this discrepancy is even so strong that the two coefficients suggest opposite answers to the question asking which of the two mechanisms contributes most to autocorrelation.

In order to understand this discrepancy, we take a closer look at the mathematical relationship between the two autocorrelation coefficients. Making use of (6), the Geary coefficient can be expressed depending on the Moran coefficient, as follows

$$c = \frac{(1 - n)}{n} I + \frac{(n - 1) \sum_i (x_{i+} + x_{+i})(z_i - \bar{z})^2}{2 \left( \sum_{ij} x_{ij} \right) \left( \sum_i (z_i - \bar{z})^2 \right)} \quad (7)$$

where  $x_{i+}$  and  $x_{+i}$  stand for actor  $i$ ’s outdegree and indegree, respectively, and their sum can be seen as the actor’s overall connectedness. The term by which the two autocorrelation coefficients differ is proportional to

$\sum_i (\mathbf{x}_{i+} + \mathbf{x}_{+i})(\mathbf{z}_i - \bar{\mathbf{z}})^2$ , the connectedness-weighted sum of squared distances from the behavioral average. What (7) implies is on the one hand that for data sets in which the highly connected actors are behaviorally extreme, the two coefficients give qualitatively different results, while for data sets in which they are behaviorally average, the coefficients give identical results. In our data (see again Figure 2), the smoking variable has a U-shaped distribution, so we can expect strong differences. The alcohol variable is unimodally distributed, which means that differences should be smaller. In fact, this pattern is what we observe in Figure 6. On the other hand, (7) implies that the Geary coefficient de-emphasizes the contribution of behaviorally extreme actors compared to the Moran coefficient (recall that Geary's  $\mathbf{c}$  is an inverse measure of network autocorrelation). Any difference between the decomposition results for the two coefficients can therefore be interpreted as resulting from this different emphasis.

Given these considerations, we must come to the following conclusions: While there is significant evidence for assimilation to friends on both substance use dimensions, this influence effect seems to occur more strongly in the behaviorally extreme groups, because Moran's coefficient (which is more sensitive to the contribution of these groups) identifies an overall stronger role of influence. For smoking, these extreme groups are the majority of students (U-shape of the distribution), which explains why the discrepancy of decomposition results between autocorrelation coefficients is particularly strong. For alcohol use, the extreme groups constitute a minority (inverse U-shape), hence the discrepancy of results is smaller.

## 5. DISCUSSION

In this paper, we presented a new method for analyzing the coevolution of social networks and changing characteristics of the actors in the network. The method is based on an explicit probability model for changes in network ties and individual behavior, mutually reacting on one another. This coevolution is crucial for a variety of research topics that currently receive a lot of attention. Examples are studies about the spread of health-related behaviors, like the smoking and drinking behavior studied in our example, about the spread of deviance and crime, about the effects of communication interaction on individual

attitudes and performance, about the formation of alliances between firms, and their effects on firm performance, or about the formation and effects of social capital of employees in organizations. The social influence processes involved are hardly ever limited to conveniently bounded groups of actors, but often a meaningful approximation can be made by focusing on groups that contain within them a large part of the social influence processes relevant to the behavior in question, so that a complete network study within such a group will uncover a major part of the dynamic interplay of structural network properties and individual behavior. In our example, this group was a school cohort.

Statistical inference about what drives the coevolution of a network and behavioral variables from empirical data is complicated. Difficulties reside in the endogenous nature of the two components, network and behavior, and in the strong influence of feedback processes on their dynamics. A longitudinal design is necessary to distinguish between what drives the network change and what drives the behavior change. In this paper we have assumed a panel design, where a network (binary) and behavior (measured on an ordinal scale) are observed at two or more discrete time points, but what happened in-between is unobserved. This type of design is frequent in the study of networks representing affective or interactional relations between individuals, such as friendship, regular communication, or advice, and also occurs regularly in the study of relations between organizations. The intermittent nature of the observations, combined with the feedback processes intrinsic to the dynamics under study, makes it necessary to postulate a model for what happens between the observations. We have found an actor-based model, with changes happening between observations in small steps, to be most natural for this purpose. The network together with the behavior of all actors jointly constitute the “state” of this model, which evolves dynamically in continuous time. The model specification has separate components for what drives network change and what drives behavior change, thus allowing us to draw separate conclusions about selection and influence while assuming that both of these processes occur simultaneously. We hope that the availability of these new methods of statistical inference will be an impetus for research on network-behavior interactions in diverse fields—including, but not limited to, those listed above. The software to estimate these models is freely available on the Internet.

Important scholarly work has been published in the econometric literature about the difficulty of identifying endogenous social interactions, defined by Manski (1993: 532) as effects “wherein the propensity of an individual to behave in some way varies with the behavior of the group.” Our model presents a special case of endogenous social interactions, where the group is defined as the personal network (the collection of members of the total network to whom the focal actor has an outgoing tie) and determined endogenously. In this case, it is possible to obtain meaningful estimates of these effects, due to the longitudinal design, the observation of the ties in the network, and the model assumptions being postulated.

As in any statistical model, the validity of the conclusions depends on the tenability of the model as, at least, an adequate approximation. A crucial assumption in our general modeling approach is that the probabilities of the changes in network and behavior depend only on the current value of network and behavior (the “state” of the model) and on the observed exogenous actor-bound and dyadic variables—in mathematical terms, that network and behavior jointly evolve as a Markov process (Norris 1997). Making such an assumption for modeling network dynamics was first proposed by Holland and Leinhardt (1977), and our approach shares this assumption with many actor-based models as well as with many statistical models. It should be noted that it is not assumed that the observed system is in equilibrium. The specification of the objective functions (cf. Tables 2 and 3) offers much flexibility to specify how exactly the probabilities of change depend on the current state and the independent variables, while staying within the class of Markov processes. In the empirical analyses, we therefore included a relatively large number of independent variables—to obtain a good fit between model and data, and to alleviate concerns about the model assumptions.

In the empirical part of the paper, we showed how persistent puzzles surrounding network autocorrelation in substance use among adolescents could be solved for the case of a study of a secondary school cohort in Glasgow by applying our method. We showed that in our data set, there was evidence for peer influence on both substance use dimensions, stronger for alcohol consumption but still significant for smoking. The effects of partner selection, on the other hand, were identified to be only weakly related to substance use, hinging more on mechanisms of network closure, structural balance, and selection based



on sociodemographic properties. Simulation studies served to quantify the “amount” of network autocorrelation that could be ascribed to the different processes of trend (in behavior and network evolution), control mechanisms, substance use based selection, and influence. These studies served to improve on earlier, similar attempts in the literature, in which the role of trend and control mechanisms in particular was neglected, and which arrived at diverging conclusions about the importance of selection and influence effects.

Important remaining concerns are the possibility of observation errors, the problem of correlated unobservables (Moffitt 2001; Mouw 2006), and the possibility of dependence on past events. These issues will hopefully be treated in future work, and we only briefly discuss each of them here. The first concern is that observation errors are a blind spot in available methods for social network analysis. Although much is known about inaccuracies in social network observations (Marsden 2005), most or all currently available statistical analysis methods assume that observation is perfect. This will be an important area for further methodological developments.

The second important concern is the possibility that there may be nonobserved variables codetermining the probabilities of change in network and/or behavior. These variables should then be added to the state variable of the system in order to satisfy the Markov property. This problem is recognized in a wide variety of models as the endogeneity of important explanatory variables (Manski 1993; Moffitt 2001). The royal road to solving it is the development of better theory and more extensive data collection. An advantage of linear models, compared to models for discrete data like ours, is the availability of methods such as fixed effects that may control, under suitable assumptions, for unobserved time-constant actor differences (Mouw 2006). Such methods have not yet been developed for the model presented in this paper. This concern implies for our approach that conclusions should be phrased cautiously, without strict causal interpretations. An example of correlated unobservables in our setup would be the possible existence of an unobserved individual characteristic  $\mathbf{v}$  with the properties that at time  $t_A$  individuals make enduring ties mainly to others with similar values on  $\mathbf{v}$ , while at time  $t_B$  individuals change their value on the behavioral variable  $\mathbf{z}$  depending on their  $\mathbf{v}$  values. If  $t_A < t_B$ , the process will seem to be influence (persons who are tied become similar on  $\mathbf{z}$ ), whereas if  $t_A > t_B$ , the process will seem to be selection (persons who

are similar on  $\mathbf{z}$  tend to become tied). Such a spurious association is quite conceivable, and it can never be ruled out in a nonexperimental design.

The third concern is the possibility of dependence on past events or past states. This dependence is ruled out by the Markov assumption, which states that change probabilities depend on the current state only; note that in our model this refers to the current unobserved state, not the most recently observed state. The strong point of the continuous-time Markov model is that it expresses feedback in a very convenient and elegant way; the limitation is that it allows dependence on the more distant past only when this is included in covariates or in the state variable. When there is information about, for instance, the duration or closeness of a friendship, this could be included in the state of the model to make the Markov assumption more plausible. It will be very interesting and useful to have such extensions of the model of this paper. When applying the current model, it will be important to optimize the plausibility of the Markov assumption by well-considered choices of the dependent network and behavior variables and by the inclusion of relevant covariates.

For all statistical methods, it is important to assess their accuracy, sensitivity, and robustness. This is as much the case for the method proposed here as for methods proposed earlier in the literature. The criticism of earlier methods that we presented above, hinging on how they deal with the three “key issues,” is of a general, theoretical nature. Other, specific criticisms of individual earlier methods have been raised as well (e.g., see Mouw’s [2006] critique of the cross-lagged panel approach). More evidence is needed, through comparisons for empirical examples as well as simulation studies, about how this translates into operational properties of the various methods, and how these methods compare to each other. In this context, goodness of fit of the proposed model is also an important issue that needs to be elaborated further, complementing work by Schweinberger (2004) for network dynamics, and by Hunter, Goodreau, and Handcock (2008) for cross-sectional network models.

We already now see our model as a useful step toward the joint modeling of network and behavior dynamics in a way that is consistent and faithfully represents what we mean by social influence and social selection processes. Another important point is that the research in this paper was limited to the network formed by one group. More advances

in theory will be possible when the coevolution of social networks and behavior can be studied in many groups, and when a generalization to a population of networks will be possible. This will require a multilevel extension of the methods proposed here, generalizing the multilevel network studies of Lubbers (2003) and of Snijders and Baerveldt (2003). Finally, also within the current framework of one network, several extensions are possible—for example, the design of effects other than those proposed here, which could represent other theoretically derived hypotheses; the analysis of multiplex (multivariate) networks; and other estimation techniques, such as maximum likelihood. We hope that the availability of this new method will stimulate empirical research in the mutually dependent dynamics of networks and behavior in a variety of substantive fields.

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[Note: Corrections were added to the article on 1 October 2010 after first publication online on 15 June 2010. The second part of equation (6) was incorrect and has been amended in this version, equation (7) has been amended to be in line with the corrections made to equation (6).]