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Modeling History Dependence in Network-Behavior Coevolution

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Spatial interdependence—the dependence of outcomes in some units on those in others—is substantively and theoretically ubiquitous and central across the social sciences. Spatial association is also omnipresent empirically. However, spatial association may arise from three importantly distinct processes: *common exposure* of actors to exogenous external and internal stimuli, interdependence of outcomes/behaviors across actors (*contagion*), and/or the putative outcomes may affect the dimensions along which the clustering occurs (*selection*). Accurate inference about any of these processes generally requires an empirical strategy that addresses all three well. From a spatial-econometric perspective, this suggests spatiotemporal empirical models with exogenous covariates (common exposure) and spatial lags (contagion), with the spatial weights being endogenous (selection). From a longitudinal network-analytic perspective, the same three processes are identified as potential sources of network effects and network formation. From that perspective, actors' self-selection into networks (by, e.g., behavioral *homophily*) and actors' behavior that is contagious through those network connections likewise demands theoretical and empirical models in which networks and behavior *coevolve* over time. This paper begins building such models by, on theoretical side, extending a Markov type-interaction model to allow endogenous tie-formation, and, on empirical side, merging a simple *spatial-lag* logit model of contagious behavior with a simple p^*-logit model of network formation. One interesting consequence of network-behavior coevolution—identically, endogenous patterns of spatial interdependence—emphasized here is how it can produce history-dependent political dynamics, including equilibrium *phat* and path dependence (Page 2006). The paper concludes with an illustrative application to alliance formation and conflict behavior among the great powers in the first half of the twentieth century.

1 Introduction

Networks—whether speaking of friendship or other relations among individuals, trade or conflict relations among states, predator–prey relations in ecosystems, or any other relations (ties, connections, edges, etc.) among units (nodes, agents, actors, etc.)—are everywhere. And these ubiquitous networks

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matter. *Network effects*, arising from structural aspects of the network, or from actors' positions in the network, or from other actors via the network of connections importantly impinge upon the behaviors, opinions, outcomes, or other characteristics of units. Networks are also often endogenous. Units typically choose or affect their ties, that is, the edges that structure their network. A major challenge empirically for researchers interested in the theory and substance of network effects and *network formation* is that network effects on nodes and the formation of ties between nodes tend to be mutually endogenous and, at the same time, both may be caused by outside factors, that is, by a third mechanism, we call *common exposure*. Archetypically from social-network analysis, for example, we may observe clusters of (non)smokers because (non)smoking is contagious—one acquires or avoids the habit from friends who smoke or abstain—or because (non)smokers choose to hang with (non)smokers: homophilic selection by behavior type—or we may observe clustering of (non)smokers because both the behavior of (non)smoking and the connections between mutually (non)smoking behavior types are caused by actors' common exposure to outside conditions, such as shared sociodemographics, that affect both the propensities to smoke and to become/stay friends. Or, expanding a more political example from Koger, Masket, and Noel (2009, 2010): Representatives who sit together may vote similarly because, sitting by party, they have similar constituencies (common exposure), or because they talk and influence each other (contagion), or they may choose to sit together because they like each other maybe for some of the same reasons they vote similarly (selection). Or, as in our empirical application, international conflict may be contagious through alliance connections, but nations with similar conflict behavior patterns may also be more likely to ally (selection), and some exogenous conditions to which particular nation-state dyads are exposed, a natural resource, for example, may affect both alliance and conflict patterns.

From spatial-econometric perspective also, as *Tobler's Law* (Tobler 1970) aptly sums: "Everything is related to everything else, but near things are more related than distant things." Furthermore, as the pithy title by Beck, Gleditsch, and Beardsley (2006) reminds in corollary: "Space is More than Geography." That is, the substantive content of Tobler's *nearness*, so the pathways along which interdependence between units may operate, extends well beyond physical distance, contact, and contiguity. Long literatures in regional science, geography, and sociology elaborate from those disciplinary perspectives the multifarious mechanisms by which contagion may arise.[1] In fact, as Brueckner (2003) showed, strategic interdependence (*contagion*) arises any time, some unit(s)'s actions affect the marginal utility of other(s)'s actions. Given such externalities, *i*'s utility depends on both its policy and that of *j*. Theoretically, substantively, then, spatial interdependence is ubiquitous. Empirically, clustering or correlation of outcomes on some dimension(s) of proximity (*spatial association*) is also obvious across a vast array of substantive contexts. However, and this is the crux of the great empirical challenge/opportunity represented by the substantive and theoretical ubiquity of interdependence, outcomes may evidence spatial association for at least these three distinct reasons. First, units may be responding relatedly to similar exposure to exogenous internal/domestic or external/foreign stimuli (common exposure) or second unit(s)'s responses may depend on others' responses (contagion, one sort of network effect). States' adoptions of some economic treaty, for example, may cluster geographically or along other dimensions of proximity, for example, bilateral trade volume, because proximate states experience similar exogenous domestic or foreign political-economic stimuli or because each state's decision to sign depends on whether proximate others sign. A third possibility arises when the putative outcome affects the variable along which clustering occurs (*selection* or network formation). Treaty signatories might also cluster by some variable on which we observe their proximity (bilateral trade volume) because being cosignatories affects that variable (spurs trade between them).

From either network-analytic or spatial-econometric perspective, accurate empirical distinction and gauge of the role and strength of these alternative processes—common exposure, contagion, and selection; that is, node effects, network effects, and network formation—are difficult because the processes manifest empirically similarly, but also crucial because the theories and policy-intervention advice supported by any observed spatial-*cum*-network phenomena hinge critically on whether, or the relative degrees to which, they arise from contagion/network effects, selection/network formation, or common exposure/node effects. The situations' substance and how policies might best intervene depend critically on

[1] Simmons, Dobbin, and Garrett (2006) offer a list for international politics: *coercion*, *competition*, *learning*, and *emulation* (to which add *relocation diffusion*, Hägerstrand 1967, such as migration).

whether state signatories cluster in pockets of dense trade because those states tend to experience similar exogenous conditions that favor signing, or because some states' signing spurs trading partners to sign, or because the treaty fosters trade among cosignatories. Moreover, as we have elsewhere demonstrated analytically, by simulation, and in applications (Franzese and Hays 2006, 2007a, 2007b, 2008a, 2008b; Franzese, Hays, and Schaffer 2010; Hays, Kachi, and Franzese 2010), drawing effective distinctions and obtaining accurate estimates empirically of any of these processes requires careful attention to specification (including measurement) of all three. Regardless of whether interest centers on network effects like the contagiousness of smoking, or on network formation like what determines trade or conflict patterns, one must model well both the network effects/contagion and the network formation/selection and also whatever exogenous factors important to either process.

This article develops a framework for theoretical and empirical modeling of social phenomena with (common exposure and) simultaneous contagion and selection, that is, of mutually endogenous network effects and network selection, that is, of the coevolution of actors' behavior and network ties. Identically from spatial-econometric perspective, this means models with exogenous covariates reflecting common exposure, with spatial-lag contagion, and with patterns of spatial connectivity (spatial weights), that is, networks, which are endogenous to behavior. Our theoretical model of such processes builds from extant Markov type-interaction models, which explain evolving and steady-state profiles of actor types based on probabilities of type switching that depend on the previous period distribution of actor types according to some set of exogenously given (possibly exogenously varying) connections between actors. These models parallel from the theoretical side extant empirical spatial-lag models of spatial econometrics, notably in the exogeneity of the connectivity matrix, that is, of the network of connections between actors, which thereby expressly disallows network formation/selection. Accordingly, we extend such Markov type-interaction models to incorporate endogenous determination of the ties between units, ties made or broken endogenously (to an extent model parameters can vary) by the previous behavior types of those units. Likewise, empirically, we merge extant spatial-lag models of interdependent behavior—specifically, the simplest time-lagged *spatial-lag logit* model —which have typically maintained exogenous connections between units, with extent models of network formation, p^* models—specifically, the simplest p^* model of independent ties—which have typically maintained exogenous unit characteristics, including behaviors, as explanators of network ties. Theoretically and empirically, the emergent models are ones of network-behavior *coevolution*.

The combination of network effects, specifically of behavioral contagion,[2] and of network formation with self-selection of actors into networks, specifically of actors' choosing their ties according to some (dis)similarity or other function of the actors' behaviors or types (*heterophily/homophily*), implies that networks and behavior coevolve over time. This paper emphasizes one interesting consequence of such network-behavior coevolution, showing how it can produce history-dependent political dynamics, including Page's (2006) *phat*, path, and/or equilibrium dependence (Jackson and Kollman 2007; Jackson 2008; Page 2006, 2007; Walker 2007). Using our Markov type-interaction model extended to allow endogenous tie formation, we establish that, and derive the conditions under which, coevolutionary systems generate multiple steady-state equilibria, and we show the connection of this multiple steady-state generation to the various forms of history dependence. Our proposed combination of the simplest spatial-lag logit and p^*-logit models yields a discrete-time Markov model that can estimate the empirical magnitude and substantive and statistical significance of such coevolutionary dynamics. A strength of this empirical approach is its direct connection with the theoretical Markov type-interaction model, which, inter alia, provides strong foundation for statistical tests of history dependence generated by coevolution. We give one such test below. The most developed (perhaps only) extant alternative approach to network-behavior coevolution is Snijders and colleagues' (Snijders 1997, 2001, 2005; Steglich, Snijders, and West 2006; Snijders, Steglich, and Schweinberger 2007) *stochastic actor-oriented models* for longitudinal social network-analysis: (*simulation investigation for empirical network analysis* (*Siena*). The paper briefly introduces the Siena coevolutionary model and estimation technique and summarizes our Monte Carlo

[2]Network effects subsume effects on nodes of the network (e.g., its *density* or *hub & spoke* structure), of the nodes' positions within the network (e.g., *centrality* or *betweenness*), and of other nodes' characteristics through their network connections (i.e., of alter on *ego*). We focus for now on this last commonly labeled contagion in the relevant literatures.

evaluations and comparisons of the two strategies.[3] Materials published on the journal Web site online elaborate this introduction and details these simulation results.

The rest of the paper proceeds thus. Section 2 presents the theoretical Markov type-interaction model, modified to allow endogenous tie formation. We propose our statistical model, specified to reflect that proposed theoretical model, and compare it to Siena in Section 3. Section 4 offers an illustrative application examining the coevolution of the great powers' military alliances and conflict behavior in the first half of the twentieth century, again comparing our proposed simple logistic strategy with Snijders and colleagues' Siena. Section 5 concludes summarily.

2 A Discrete-Time Markov-Chain Theoretical Model of Network-Behavior Coevolution

This section gives a theoretical model of network-behavior shaping (contagion) and tie-formation (selection) effects comprised of two sets of Markov chains. In this model, a group of actors are of certain behavior types, types which change over time as actors are influenced by others (and exogenous factors). Such behavior type contagion occurs only if the actors are *connected*, where the explicit notion of networks (or spatial weights) characterizes such *connectedness*. Simultaneously, the connectivity of actors also changes over time, not only due to exogenous factors but also as a function of types taken by actors in the previous period. We particularly highlight behavior type *heterophily/homophily*, where network ties more likely form and persist among actors whose behavior types are less/more similar the previous period. The key features of, and the additional sources of complexity due to, this extension of extant type-interaction models are: (1) it introduces the details of which actor interacts with which to represent network effects on actors' behavior, specifically, contagion effects, and (2) it describes how those interaction patterns, that is, the networks cum spatial-weights matrice, evolve endogenously based on actors' types in the previous period, which reflects homophily by behavior type.[4]

We then demonstrate that this model can produce long-run steady-state (LRSS) equilibria[5] that depend on starting values and history. With coevolution, multiple steady-state distributions of types are consistent with a single behavior-switching rule. The type/behavioral-rule combination that emerges at a given point in time depends on actors' prior types, so the evolution of behavioral types is history dependent and may be specifically path, *phat*, or initial-conditions equilibrium dependent (Page 2006).

2.1 *A Minimal Coevolutionary Model*

We offer a theoretical model minimally sufficient to incorporate both contagion in node behavior and behavioral homophily in network-tie formation and show that and how this suffices to generate steady-state path dependence. Consider a discrete-time process with actors $i \in \{1, \ldots, N\}$ and time periods $t \in \{1, 2, \ldots\}$. Distinguish an actor's *behavior* from her behavior type, with type being the actors' probability of taking behavior 1. Let behavior be observed and dichotomous, whereas type is continuous and unobserved by analysts but observed by actors, with contagion and selection occurring by type. Actors i choose behavior 1 or 0 (whether to smoke, vote, take an aggressive interstate behavior, democratize, etc.) each period t, denoted $s_{it} \in \{0, 1\}$. Denote the behavior type of i in t, that is, $\text{pr}(s_{it} = 1)$, by $\sigma_{it} \in [0, \ldots, 1]$. The state of the system at the end of period t, which actors observe, is thus an N-dimensional vector of types $\sigma_t = (\sigma_{1t}, \ldots, \sigma_{Nt})$, a corresponding vector of behaviors, $\boldsymbol{s}_t = (s_{1t}, \ldots, s_{Nt})$, and a matrix of latent and observed ties between actors to be described.

The system incorporating both contagion and selection comprises two sets of Markov chains, N explaining type and $\frac{1}{2}N(N-1)$ explaining tie formation. We focus first on the behavior-type Markov chains that incorporate network contagion effects among the N actors. Equation (1) describes actor i's

[3] These may be the first Monte Carlo evaluations of Siena and more certainly the first comparisons to an alternative (Leenders 1997 did evaluate his precursor models).

[4] Homophily refers to phenomena where ties more likely form/persist between actors similar in some characteristic(s).The homophilic-selection bases could be exogenous or endogenous. *Behavioral homophily*, network selection by similarity of the endogenous behavior (types), plus behavior being contagious by those enodgenously selected ties, equals coevolution.

[5] *Equilibrium* here means consistency between actors' behavioral types and their behavior-switching rules: *system steady state* or *fixed-point*; Nash strategic equilibrium is not implied.

probabilities of transitioning type from period t to $t+1$:

$$\begin{pmatrix} \sigma_{i,t+1} \\ 1-\sigma_{i,t+1} \end{pmatrix}' = \begin{pmatrix} \sigma_{it} \\ 1-\sigma_{it} \end{pmatrix}' \begin{pmatrix} c_{0i}\sigma_{it} + (1-c_{0i})\frac{\sum_{j\neq i}(\delta_{ij}\sigma_j)}{N-1} & 1-\text{Left} \\ 1-\text{Right} & c_{1i}(1-\sigma_{it}) + (1-c_{1i})\frac{\sum_{j\neq i}(\delta_{ij}(1-\sigma_j))}{N-1} \end{pmatrix} \quad (1)$$

The left-hand side (LHS) is a row vector of next period's types, $\sigma_{i,t+1} = \Pr(s_{i,t+1} = 1|\sigma_{it}, \delta_{ij,t})$ and $1-\sigma_{i,t+1} = \Pr(s_{i,t+1} = 0|\sigma_{it}, \delta_{ij,t})$, where $0 \leqslant \delta_{ij,t} \leqslant 1$ indicates the probability (or, isomorphically in this model and perhaps more substantively appealing, the strength) of dyad ij connection last period. At far, right-hand side (RHS) is the transition probability matrix, which, premultiplied by t's types, yields $t+1$'s types. For example, cell $(1,1)$ gives the probability i chooses behavior 1 given her propensity toward 1 last period, $\Pr(s_{i,t+1} = 1|\sigma_{it})$. Because each new state arises from one of the two possible previous states, this matrix is *row* (or *right*) *stochastic*: its rows sum to 1. Therefore, defining any one element of each row, for example, the "staying probabilities" in cells $(1,1)$ and $(2,2)$, suffices to complete the transition matrix.

We separate each transition probability in two components. A temporal-autoregressive aspect first: an actor is more likely to maintain behavior 1 at $t+1$ the nearer her latent type at t is to 1. The probability of "staying" in $s_i = 1$ is higher the greater was σ_{it}, the propensity toward action 1 last period. The transition matrix's first-row (second-row) elements—giving the respective staying and "switching" probabilities from behavior 1 (behavior 0)—capture this temporal-dependence component by $c_{0i}\sigma_{it}$ and $c_{1i}(1-\sigma_{it})$, respectively. Second, a contagion component: each actor's behavioral decisions are also influenced by others' types. Actors in a given dyad $(\{i,j\}, i \neq j)$ influence each other's behavior type only insofar as they are connected. Let $0 \leqslant \delta_{ij,t} \leqslant 1$ denote the probability that a tie exists or, isomorphically in this model, the strength of the tie between i and j in period t, that is, $\Pr(d_{ij,t} = 1)$.[6] We express this probability/strength of connection as $\sum_{j\neq i}(\delta_{ij}\sigma_{jt})/(N-1)$, which normalizes the j's tied to i by $(N-1)^{-1}$ to bound this to the 0–1 interval.[7] The second terms in the transition probabilities from behavior 1, $(1-c_{0i})\frac{\sum_{j\neq i}(\delta_{ij}\sigma_{jt})}{N-1}$, relate to contagion effects. By this term, i's probability of staying in behavior 1 increases with the weighted sum of the others' propensities to take action 1, $\frac{\sum_{j\neq i}(\delta_{ij}\sigma_{jt})}{N-1}$. The analogous contagion component of the probability i stays in behavior 0 is seen in cell (2,2) as $(1-c_{1i})\frac{\sum_{j\neq i}(\delta_{ij}(1-\sigma_{jt}))}{N-1}$. Thus, the behavior is contagious, and contagion effects operate via actors' propensities toward action 1 or 0, σ_{jt} or $1-\sigma_{jt}$, and strengthen with the latent dyadic tie strengths, δ_{ij}.

Notice how the parameter $c \in [0,1]$ captures the extent to which i's own behavior type in time t influences her behavior in $t+1$ and $1-c$ indicates the remaining relative role of contagion, that is, of j's time-t type in determining i's $t+1$ behavior. The parameters $\mathbf{c}$ thus gauge the strength of contagion versus exogenous internal or external (here, autoregressive) factors. At one extreme where $c_{0i} = 1$, for example, i's time-$t+1$ behavior choice remains, as her time-t choice was, solely determined by her behavior type, $\sigma_{i,t}$ and not at all affected by any others' to whom she is connected: that is, the strength of contagion is 0.[8]

The behavior choice being dichotomous, one conditional probability suffices to describe those Markov chains, $\Pr(s_{i,t+1} = 1|\sigma_{it}) = \sigma_{i,t+1}$ (the other is just $1-\sigma_{i,t+1}$):

$$\begin{aligned} \Pr(s_{i,t+1} = 1|s_{it}) = \sigma_{i,t+1} = \sigma_{it}&\left[c_{0i}\sigma_{it} + (1-c_{0i})\frac{\sum_{j\neq i}(\delta_{ij}\sigma_j)}{N-1}\right] \\ &+ (1-\sigma_{it})\left[1 - \left\{c_{1i}(1-\sigma_{it}) + (1-c_{1i})\frac{\sum_{j\neq i}(\delta_{ij}(1-\sigma_j))}{N-1}\right\}\right]. \end{aligned} \quad (2)$$

[6]The model assumes undirected ties/symmetric spatial-weights matrices and one basis for connection between units. Extension to *directed networks*/*asymmetric weights* and multiple ties remains (very importantly, if perhaps not very easily) for future work (Franzese and Hays 2006 and Hays, Kachi, and Franzese 2010 have made such extensions in linear model systems).

[7]The row standardization common in spatial econometrics or the recommended spectral normalization of Kelejian and Prucha (2010) would also bound $0 \leqslant \sum_{i\neq j}(\delta_{ij}\sigma_{jt}) \leqslant 1$; our less orthodox $1/(N-1)$ weighting is equally functional and greatly facilitates the model's accounting.

[8]These two components enter in convex combination and the actor's types, σ, are probabilities (that they choose $s = 1$). This properly bounds all transition probabilities to $[0, \ldots, 1]$.

Equation (3) gives the tie formation Markov chains. The unit of analysis is now a dyad (i, j). The probability that i and j are connected in period $t+1$ is denoted $\delta_{ij,t+1} \in [0, \ldots, 1]$:

$$\begin{pmatrix} \delta_{ij,t+1} \\ 1-\delta_{ij,t+1} \end{pmatrix}' = \begin{pmatrix} \delta_{ij,t} \\ 1-\delta_{ij,t} \end{pmatrix}' \begin{pmatrix} \delta_{ij,t} & 1-\text{Left} \\ 1-\text{Right} & c_{2,ij}(1-\delta_{ij,t}) + (1-c_{2,ij})(\sigma_i-\sigma_j)^2 \end{pmatrix}. \quad (3)$$

Extant type-interaction models assume tie formation probabilities exogenous (in fact, often fixed and uniform), but the potential for endogenous ties is core to coevolution. We allow actors to prefer ties to others who behave (dis)similarly, *behavioral (heterophily) homophily*. To simplify, we build this behavioral selection component directly into the transition probabilities only in the time-t , $d_{ij,t} = 0$.[9]

The second term of transition matrix element (2, 2), $(1-c_{2,ij})(\sigma_i-\sigma_j)^2$ gives this behavioral selection effect: for homophily (heterophily), $0 < c_2 < 1$ ($-1 < c_2 < 0$), as the distance between two actors' behavior types increases, the dyad is less (more) likely to connect. So, with $0 < c_2 < 1$, our model exhibits homophilic tie formation by behavior type: two (non) smokers are more likely to be friends, two countries of similar conflict behaviors more likely to ally, two representatives of closer ideologies more likely to cosponsor bills, etc.

As before, the other terms, $\delta_{ij,t}$ and $c_{2,ij}(1-\delta_{ij,t})$ in (1,1) and (2,2), reflect temporal autoregression—again: standing in for all common exposure factors that affect tie formation. Analogously to c_1, c_2 reflects the strength of temporal autoregression (common exposure) relative to behavioral selection effects in the transition probabilities, and $(1-c_{2,ij})$ reflects the remaining extent to which hetero- or homophilic selection determines tie formation. The combinatorial forms of the various weights again serve to bound probabilities properly $[0, \ldots, 1]$.

A single conditional probability again suffices to specify the tie formation chains:

$$\Pr(d_{ij,t+1} = 1 | d_{ij,t}) = \delta_{ij,t+1} = \delta_{ij,t}^2 + (1-\delta_{ij,t})[1 - \{c_{2,ij}(1-\delta_{ij,t}) + (1-c_{2,ij})(\sigma_i-\sigma_j)^2\}]. \quad (4)$$

The systems of difference equations (1) and (3) complete our theoretical model of network-behavior coevolution, that is, of jointly endogenous contagion and selection. The steady-state equilibrium of this system consists of a vector of each actor's type and each dyad's tie forming probability, (σ, δ). This steady state solves equations (1) and (3) for σ for $\sigma_{i,t+1} = \sigma_{i,t}$ and $\delta_{ij,t+1} = \delta_{ij,t}$, $\forall i, j \in \{1, \ldots, N\}$. In particular contexts, our interests may lie primarily in the steady states and/or the intertemporal dynamics of actors' types or of dyads' ties, σ or δ; regardless, either can only be characterized deriving vectors of types and tie probabilities both due to the endogeneity generated by homophily and contagion (Our exposition will highlight behavior type and network-tie steady states, suppressing dynamics for compactness.)

To illustrate the multiple steady states of this coevolutionary system, consider a two actor, $i = \{1, 2\}$, one undirected edge, δ_{12}, example. This gives a system of three equations of motion, two for the actors' behavior type and one for their dyad's tie formation processes:

$$\begin{cases} \sigma_{1t} &= \sigma_{1,t-1}[c_{01}\sigma_{1,t-1} + (1-c_{01})\delta_{12,t-1}\sigma_{2,t-1}] \\ &\quad + (1-\sigma_{1,t-1})[1 - \{c_{11}(1-\sigma_{1,t-1}) + (1-c_{11})\delta_{12,t-1}(1-\sigma_{2,t-1})\}] \\ \sigma_{2t} &= \sigma_{2,t-1}[c_{02}\sigma_{2,t-1} + (1-c_{02})\delta_{12,t-1}\sigma_{1,t-1}] \\ &\quad + (1-\sigma_{2,t-1})[1 - \{c_{12}(1-\sigma_{2,t-1}) + (1-c_{12}))\delta_{12,t-1}(1-\sigma_{1,t-1})\}] \\ \delta_{12,t} &= \delta_{12,t-1}\delta_{12,t-1} + (1-\delta_{12,t-1})[1 - \{c_2(1-\delta_{12,t-1}) + (1-c_2)(\sigma_{1,t-1}-\sigma_{2,t-1})^2\}]. \end{cases} \quad (5)$$

Solving this system for its LRSS $\boldsymbol{\sigma}$ and δ yields: $\{\sigma_1 = \sigma_2, \delta_{12} = 1\}$. Any $\sigma_1 = \sigma_2$ and $\delta_{12} = 1$ is a candidate steady state; in the LRSS, 1 will be tied to 2 but at different $\sigma_1 = \sigma_2$ depending (at least) on initial conditions. Page (2006)'s rigorous definitions of history dependence distinguish sensitivity to initial conditions, to the set or sequence of past conditions, or to immediate past conditions in shaping equilibria (that is, steady states) and, distinctly, outcomes along the path. As we shall illustrate, at which type the actors in this model will settle (equilibrium) depends on where they start (initial conditions) and the immediate past but also on the set (*phat*) and sequence of past conditions (*path*).

[9]Indirect, time-lagged network selection effects nonetheless manifest in both states, and the qualitative conclusions of the model do not depend on the simplification.

2.2 *Illustrations of History Dependence in Coevolutionary Models*

History dependence refers to phenomena where past conditions alter a system's future course. This broad notion is often conflated with the much narrower concept, *path dependence*, but following Page (2006) we define history dependence most broadly and differentiate three increasingly restrictive cases within it: *state*, *phat*, and *path dependence*. The most restrictive path dependence means that a system's future history depends on the path, that is, the *sequence* or *order*, of past conditions, and not merely on the set, which is the less-restrictive *phat* or *set* dependence. The least-restrictive state dependence is where a system's trajectories can be partitioned into a finite number of states that contain all relevant information for the future of the system regardless of events outside that partition (meaning that the system's future depends on its current state not the path or set of earlier conditions). We also distinguish *outcome* from *equilibrium* history dependence. In the former, each period's outcome (e.g., $\boldsymbol{s}_t$) depends somehow on outcome(s) in past period(s) (e.g., $\boldsymbol{s}_{t-v}$) or on the time index. Equations (1) and (3) show that our model, like any temporal-autoregressive model, is outcome history dependent. More interesting here is steady-state dependence: whether the LRSS of behavioral types, $\boldsymbol{\sigma}$, and strength-of-ties, $\boldsymbol{\delta}$, depend on their history (sequence, set, or state). We illustrate the forms of history dependence our model may exhibit by a series of numerical exercises in a two-actor system with given sets of initial and/or sequences of behavior type and tie probability values, $\boldsymbol{\sigma}$ and δ, and/or exogenous parameters, $\mathbf{c}$.

Figure 1 shows the sequence of actor 1's behavior type (σ_{1t}) over the first 11 periods, with the Markov chains (equation (5)) solved recursively from two sets of initial conditions (starting values), $\{\sigma_{11} = \sigma_{21} = \delta_{12,1} = 0.4\}$ or $\{\sigma_{11} = \sigma_{21} = \delta_{12,1} = 0.7\}$, but with all parameter values fixed in both cases at $c_{01} = c_{11} = 0.9, c_{02} = 0.1, c_{12} = 0.9, c_2 = 0.5$. The LRSS behavior types are ~0.6173 and 0.6244, respectively (with $\sigma_2 = \sigma_1$ in both, as concluded above). The LRSS types depend on starting values of the endogenous variables, $\boldsymbol{\sigma}$ and δ, even with the exogenous parameters (c's) fixed; that is, in Page (2006) terms, the system exhibits *initial conditions equilibrium dependence*. This does not necessarily indicate either of the stricter forms of history dependence, phat or path, but conventional type-interaction models do not exhibit even this weakest initial conditions, dependence.

To analyze stricter forms of history dependence, we consider changes in parameters $\mathbf{c}$ over the system's history. As mentioned above, the temporal-autoregressive parameters, being the only exogenous noncontagion or nonselection terms in the respective equations of the theoretical model, serve also as placeholders for all the exogenous conditions to which the actors may be exposed (i.e., analogously to the exogenous $\mathbf{X}\boldsymbol{\beta}$ of a regression model). As such, history dependence on these $\mathbf{c}$ may be substantively more interesting and practically more important than dependence on type starting values because one could more easily imagine intervention on and relate substantively to variation in some actor/dyad-specific attributes, $\boldsymbol{x}$, that is, theoretical-model conditions $\mathbf{c}$, than manipulating or varying initial states.[10]

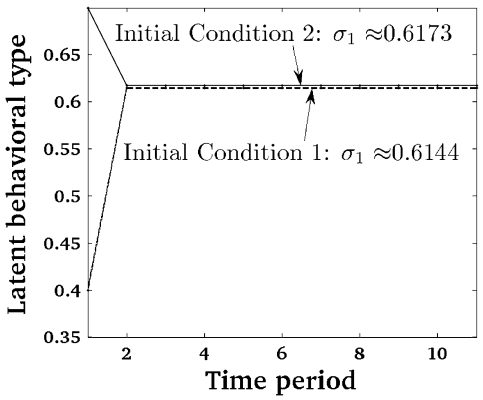

Fig. 1 Initial-conditions steady-state sensitivity in a coevolutionary system.

[10]Our emphasis on history dependence relating to the parameters of a nonlinear system of equations resonates with results in Jackson and Kollman (2007).

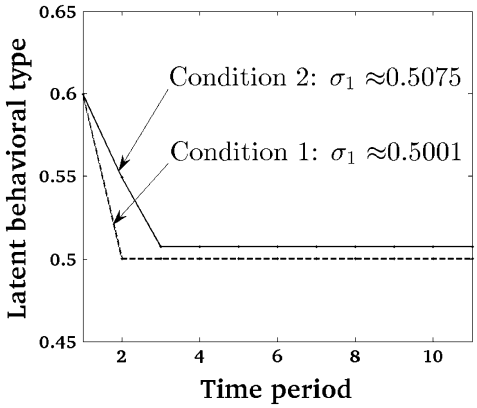

Fig. 2 Early conditions steady-state sensitivity (Phat dependence).

We start with the simplest form of phat dependence, *early conditions sensitivity*. Figure 2 plots the path of actor 1's LRSS behavior type under two different sets of parameters. Condition 1 remains at $\{c_{01} = 0.9, c_{11} = 0.1, c_{02} = 0.1, c_{12} = 0.9, c_2 = 0.1\}$ for all 11 periods. Condition 2 instead starts with parameters $\{c_{01} = 0.1, c_{11} = 0.6, c_{02} = 0.1, c_{12} = 0.1, c_2 = 0.3\}$, but some intervention changes the parameters to $\{c_{01} = 0.9, c_{11} = 0.1, c_{02} = 0.1, c_{12} = 0.9, c_2 = 0.1\}$ from $t = 2$ onward. Starting values for the endogenous behaviors and ties, $\boldsymbol{\sigma}$ and δ, are the same in both scenarios; the only differences lie in their sets of **c**. Conclusion: the set of past conditions, specifically early conditions, of the exogenous parameters, **c**, matter; the system with behavioral contagion and homophily is (at least) phat dependent.

Finally, the top-left graph of Fig. 3 shows that, with both behavior-type contagion and behavior-homophilic selection (coevolution), our model exhibits true equilibrium path dependence (Page 2006): the LRSS behavior types $\boldsymbol{\sigma}$ and tie strength δ depend on the order, not just set, of past events. The graph plots the dynamics and LRSS of actor 2's behavior type under alternative Path 1 and 2 scenarios that differ only by the sequence of exogenous values, $c_{01}, c_{11}, c_{02}, c_{12}, c_2$, with the first two vectors **c** order reversed. The history is constant within and equal across scenarios from $t = 3$, and both paths share endogenous variable starting values: $\{\sigma_1 = 0.6, \sigma_2 = \delta_{ij} = 0.4\}$. The LRSS behavior types are $\sigma_1 = \sigma_2 \approx 0.5141$ under Path 1 and $\sigma_1 = \sigma_2 \approx 0.5008$ under Path 2 ($\delta_{12} = 1$ in both). The two paths differ only in the sequence of past conditions but generate different LRSS: true path dependence.

Figure 3 reveals a core result from our model. The coevolutionary system exhibiting path dependence plotted at top-left has both behavioral contagion and behavior-homophilic section. We can set $\mathbf{c}_0$ and $\mathbf{c}_1$ to 1, eliminating contagion, and/or $\mathbf{c}_2$ to 1, eliminating selection, and compare responses the same alternative paths of conditions. At top-right, with contagion but without homophilic selection, 1's LRSS behavior type is $\sigma_1 = 0.6429$ regardless of path, and at bottom-left, with homophilic selection but without contagion, 1's behavior type remains as initially assigned: $\sigma_1 = 0.6$. Path dependence is also eliminated with no contagion and no homophilic selection of course. The crucial upshot is that behavioral contagion and behavioral-homophilic selection are both required to generate path dependence; this suggests a direct empirical test for path dependence in the empirical model below.

Table 1 summarizes the crucial conclusions of the proposed discrete-time Markov chain type-switching model of coevolutionary dynamics: endogenous coevolution of network (spatial) connections, which depend in part on the behaviors of the connected nodes, and of node behaviors shaped in part by others' behaviors through that network generates systems of nonlinear difference equations that can easily produce initial condition, state, phat, and path steady-state history dependence.[11] We specified the transition probabilities of an example system with parameters reflecting temporal autoregression and, implicitly, other exogenous (or predetermined) covariates on the one hand (embodying common exposure factors), and on the other hand, contagion through network connections in the behavioral model and homophily

[11]To appreciate how easily coevolution introduces complexity, note how minimally Page's *Rule of Six* (2007)—systems must have numbers of actors plus choices of six or more to have multiple equilibria—is met here: 2 actors plus 3 dichotomous equations, 2 behaviors and 1 tie, suffice because the *symmetric* network-tie choices, ij and ji contribute just 1 equation.

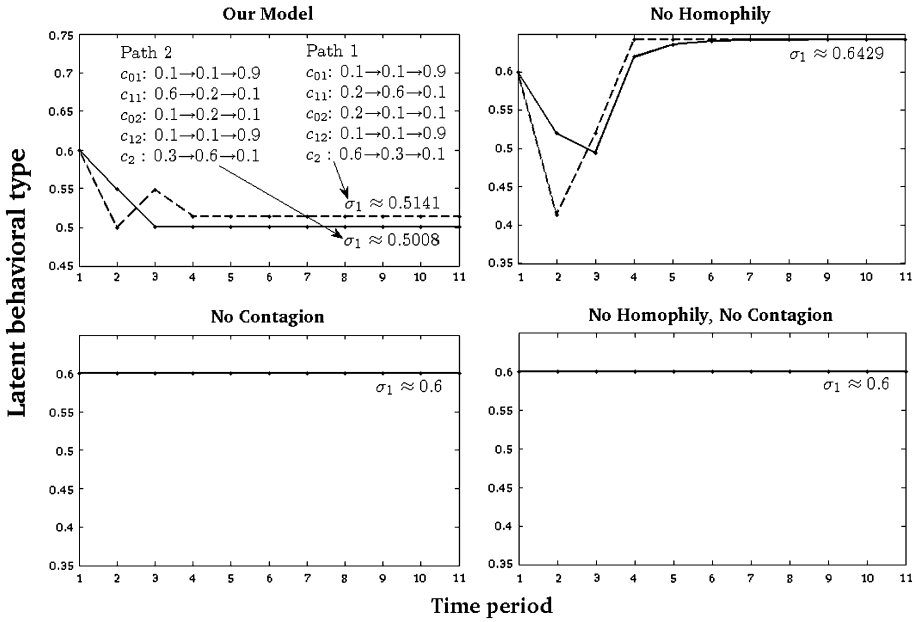

Fig. 3 Equilibrium (steady-state) path dependence in a coevolutionary system.

by behavior type in the network-tie formation model (selection). Our analysis indicated that the existence of steady-state history dependence depended on the *joint* presence of contagion and selection. Dynamic models of ties and/or behavior without one or both processes do not exhibit steady-state path dependence. This will suggest the form of a test for path dependence in the empirical model to come.

3 Empirical Strategies

This section uses our theoretical model, which expresses two kinds of transition probabilities, one in behavior and one in network ties, as functions of three kinds of conditions—exposure to exogenous factors, contagion, and selection—to suggest a simple empirical strategy for estimating models of social phenomena that can distinguish these inputs as sources of network-cum-spatial association, correlation, or clustering. Our logistic discrete-time Markov model for empirical analysis of coevolutionary processes combines the simplest (time-lagged) *spatial-lag* models of behavioral contagion from spatial econometrics with the simplest (independent) p^* models of behavior-homophilic selection in tie formation

Table 1 Path dependence in type-interaction models

	No contagion ($c_1 = 1$)	*Contagion* ($c_1 \neq 1$)
Exogenous tie formation ($c_2 = 1$)	No contagion of behavior types. Tie formation is also exogenous to behavior type LRSS: $\sigma^*_{it} = \sigma_{i0}, \forall i, \delta^*_{ij,t} = \delta_{ij,0}, \forall i, j$ Path independent	Behavior type is contagious, but tie formation is exogenous to behavior type LRSS: $\sigma^*_{it} = 1, \forall i, \delta^*_{ij,t} = \delta_{ij,0}, \forall i, j$ Path independent
Endogenous tie formation: homophily ($c_2 \neq 1$)	No contagion in actors' behavior types, but tie formation is endogenous to behavior types, with actors more likely to form ties with similar behaviortypes (homophily) LRSS: $\sigma^*_{it} = \sigma_{i0}, \forall i, \delta^*_{ij,t} = 1, \forall i, j$ Path independent	Behavior type is contagious, and tie formation is endogenous to behavior type, with actors more likely to form ties with similar behavior types (homophily) LRSS: $\sigma^*_{it} = \sigma^*_{jt}, \forall i, j, \delta^*_{ij,t} = 1, \forall i, j$ Path dependent

Note. LRSS, long-run steady state. Time index "0" in σ_{i0} and $\delta_{ij,0}$ indicate starting values.

from network analysis. The section also briefly introduces the extant alternative, Snijders' *actor-oriented* continuous-time coevolution model, Siena (Snijders 1997, 2001, 2005; Snijders, Steglich, and Schweinberger 2007; Ripley and Snijders 2010) and summarizes our Monte Carlo evaluations of Siena's sMoM and our simple-logistic estimators' performances.[12]

3.1 *A Simple Logistic Discrete-Time Markov Model Strategy*

Our discrete-time Markov empirical model has contagion in behavior (not behavior type) and selection by observed ties (not latent tie strengths). We give the behavior-shaping probabilities as N simple spatial-lag logit models, with only time-lagged (and not simultaneous) spatial-lags, and tie formation probabilities as the simplest p^* model, one with independent dyads (similarly reducing tie formation to $N(N-1)$ conditionally independent logits):[13]

$$\begin{cases} \Pr(s_{i,t}=1|\mathbf{s}_{t-1},\mathbf{d}_{t-1}) = \text{logit}(\beta_0+\beta_1 s_{i,t-1}+\beta_2 \mathbf{d}_{i,t-1}\mathbf{s}_{t-1}), \\ \Pr(d_{ij,t}=1|\mathbf{s}_{t-1},\mathbf{d}_{t-1}) = \text{logit}(\gamma_0+\gamma_1 d_{ij,t-1}+\gamma_2 \cdot I(s_{i,t-1}=s_{j,t-1})), \end{cases} \tag{6}$$

where $\mathbf{d}_{i,t-1}$ is a row vector of size N containing the $(N-1)$ binary tie formation indicators between i and each other actor at the end of period $t-1$ (and 0 in element i for dyad ii), and $I(s_{i,t-1}=s_{j,t-1})$ indicates whether given dyad's behaviors were the same in the previous period, capturing homophily. As noted, this model's contagion and selection, that is, network effects and formation, both operate through observed behaviors, not latent behavior type.

Estimating equation (6) is straightforward; behavior and tie formation can be estimated separately or as a seemingly unrelated system of logit equations. If the disturbances—that is, the extreme-value disturbances from the underlying choice models—are correlated across equations, separate estimation would produce consistent, though inefficient, estimates of parameter values, and standard error estimates would be inaccurate. The standard error inaccuracy issue can be redressed by robust (i.e., consistent) standard errors using a systems sandwich estimator of the variance-covariance matrix. The sandwich matrix in this formulation, the outer product of the gradients of the likelihoods, provides estimates of the parameter covariances across equations, which are incorporated into the variance estimates.[14]

3.2 *Siena's sMoM Continuous-Time Markov Model*

In the network-analytic tradition, Snijders and colleagues (op. cit.) have advanced furthest in empirical modeling of dynamic, endogenous contagion, and selection.[15] In Siena, N actors are connected by an observed, binary, potentially endogenous, and time-variant matrix, $\mathbf{x}$, of ties, $x_{ij,t}$. A vector of N observed, binary behaviors, $\mathbf{z}$, at time t has elements $z_{i,t}$. Additional exogenous explanators may exist at unit or dyadic level, $\mathbf{v}_{i,t}$ or $\mathbf{w}_{ij,t}$. Opportunities arise for actors to change their network ties, switching at most 1 tie on or off, at continuous-time fixed rate, $\rho^{\text{net}}_{i,t}$, according to an exponential model. Likewise, opportunities to switch or leave unchanged the dichotomous behavior arise at rate $\rho^{\text{beh}}_{i,t}$.[16] When an opportunity to change network ties arrives for some i, she may choose to switch *on* or *off* any one of her $N-1$ ties by comparing the values of her objective function, $f^{\text{net}}_i(\mathbf{x},\mathbf{x}',\mathbf{z}) + \varepsilon^{\text{net}}_i(\mathbf{x},\mathbf{x}',\mathbf{z})$, under the existing behaviors, $\mathbf{z}$, and network, $\mathbf{x}$, to the existing behaviors and the network under the considered tie change, $\mathbf{x}'$. The weights on the various network statistics in these objectives are the coefficients, β^{net}_h, to be estimated. Assuming $\varepsilon^{\text{net}}_i$ extreme value distributed, independently across i and t, yields a multinomial-logit categorical choice model. Similarly, when a chance to change behavior arrives, i compares values of an analogous objective

[12] Materials published on the journal Web site online elaborate and detail the introduction and comparisons.

[13] Lazer (2001) takes similar approach to modeling network-behavior coevolution. Important future extensions include enriching these models to simultaneous spatial-dependence and nonindependence p^* cases (feasibly, computational demands of both can be very high).

[14] *Stata* gives these system sandwich estimates at one postestimation command: *suest*.

[15] Wasserman (1980a, 1980b), Leenders (1997) presage. Bayesian latent-space longitudinal networks (Hoff, Raftery, and Handcock 2002; Hoff and Ward 2004; Hoff and Westveld 2007) may also relate.

[16] Although Siena can accommodate richer parameterizations, both ρ are held constant across i but allowed to differ arbitrarily by t here. These rates of intraobservational event occurrence can vary freely, so the assumption of one i making one 1-unit change at a time is inconsequential. The strong assumption (we also make) of conditional independence of the choices does remain though.

function under alternative actions (here, binary): switch to 1 or 0 or leave unchanged. Again assuming i.i.d. extreme value stochastic components ($\boldsymbol{\varepsilon}_i^{\text{beh}}$), the logistic form emerges.

The behavior and network objective functions (and also the rate functions if desired) can include any of a number of commonly supposed social-network phenomena. For instance, importantly for our purposes, *covariate-related dissimilarity*, which is "defined by the sum of absolute covariate differences between i and the others to whom he is related" (371):

$$\text{covariate-related dissimilarity: } s_i(\mathbf{x}) = \sum_j \mathbf{x}_{ij} |v_i - v_j|. \tag{7}$$

Entering $s_i(\mathbf{x})$ in the tie formation equation with covariates v_i and v_j being i's and j's behaviors gives a behavioral homophilic (or, rather, heterophilic) selection term. RSiena estimates such models by sMoM. That is, it simulates network-behavior outcomes according to the processes of the proposed model and estimates the parameters of that model (along with estimated variance-covariances for those parameter estimates by the delta method) by optimizing fit of simulated to observed sample statistics.[17]

As a theoretical model and estimation strategy for simultaneous tie formation and behavioral choices, Siena is the state of the art. Yet, notice also the many caveats stressed:

- "Although in our experience, these equations mostly seem to have exactly one solution, they do not always have a solution" (Snijders 2001, 374).
- "[The moment-conditions stated are] far from implying the statistical efficiency of the resulting estimator, but it confers a basic credibility to [. . . it and. . .] ensures the convergence of the stochastic approximation algorithm. . . " (Snijders 2001, 373).
- ". . . the method proposed here is not suitable for observations. . . too far apart in [. . . the number of intraobservational changes]. For such [. . . cases, dependence of one observation on the previous. . .] is practically extinguished, and it may be more relevant to estimate the parameters of the process [. . . separately]" (Snijders 2001, 374).
- "It is plausible that these estimators have approximately normal distributions, although a proof is not yet available" (Snijders 2001, 375).

This is a small subset of the statements acknowledging various aspects of the estimation strategy performance as unknown or maybe problematic, but we do not highlight them as criticism. Siena seems the currently best developed tool capable of addressing coevolution, which we think is common and important in social science, and its approach to modeling network formation and behavioral choice shares our emphasis on affording address of a theoretically and substantively central empirical challenge of distinguishing and distinctly estimating the common exposure, contagion, and selection effects in generating social outcomes that ubiquitously exhibit network/spatial association. Our point is instead to underscore how little is known regarding Siena's performance as an estimator. Understandably given its complexity, little has been proven analytically about its properties; nor, also understandably given its computational demands and its specialized implementing software until RSiena's recent advent, has its performance been explored much in Monte Carlo analysis.

3.3 *Estimation Strategy Evaluation and Comparison*

Next, we summarize our evaluation and comparison of the simple time-lagged spatial-lag logistic-regression strategy proposed here and Siena's sMoM strategy for estimating models of network-behavior coevolution, that is, with contagion and selection. These evaluations and comparisons are elaborated and detailed in the web materials.

Our simulations followed (Snijders 2001) to specify a data-generating process (DGP) exactly replicating a Siena model of coevolution with the behaviors of N actors contagious through a network of ties

[17]The online appendix elaborates; see also Snijders (2001) and Ripley and Snijders (2010) for further estimation procedure details and options.

generated by behavioral homophily. Using this DGP, we generated 100 trials each of eight different scenarios: varying the number of actors $N \in \{30, 50\}$, the number of observed periods, $T \in \{5, 11\}$, and the rates of event occurrence, $\rho_{\text{net}} = \rho_{\text{beh}} \in \{1, 5\}$. The coefficient magnitudes are not directly comparable; nevertheless, we can conclude already from the raw parameter estimates on several points. First, in contexts with higher event rates, that is, where intraobservational changes in networks and behavior are likely to have been great reliable estimation of coevolutionary processes by either strategy seems impossible. For both strategies, statistical power to discern behavioral contagion or behavioral homophilic selection was negligible at the higher rates, and parameter estimates exhibited very large biases and/or mammoth inefficiency. Standard error accuracy was also problematic. At lower rates, either estimator reports reasonably honestly about the certainty of its parameter estimates. Siena seems essentially unbiased in lower-T samples but suffers some downward or deflationary bias in its estimates at larger T (oddly). The spatial-logistic parameter estimates seemed roughly to parallel Siena's in magnitude, although bias could not be gauged since the "true" parameters of this incorrect model under the Siena DGP were unknown. At these lower rates, the simpler spatial-logistic strategy seemed to have some edge in efficiency and, thereby, in power, with this advantage growing more noticeable with lower T and smaller samples. Judging by the parameter estimates, therefore, one could summarize: at low event rates, both strategies work generally acceptably and roughly comparably well, with an efficiency advantage and simplicity perhaps favoring the logistic strategy; at higher event rates, neither strategy managed to gain any appreciable traction on contagion or selection.

To evaluate the performance of Siena and our simple logit more effectively, we should calculate estimated effects on behavior or tie formation by each estimator of some common hypothetical. We consider the following hypothetical regarding contagion. If all i's network partners behave in one way (all 0 or 1), what are the odds that i will choose the network consistent over the network inconsistent behavior? In the Siena DGP, we can get these odds thus: if all i's ties are initially to dissimilar behavior types (so her average similarity score is 0) and i switches her behavior to match her network partners, her average similarity will go to 1, and the corresponding odds of going from inconsistent to consistent behavior, assuming i is chosen to act, are $\exp(\beta_{\text{beh}})$ to 1 (≈ 2.714). In the simple-logit model, if i's network partners switch their behavior from all-0 to all-1, then for i, behavior 1 likewise goes from being network inconsistent to network consistent, and the odds of choosing behavior 1 gives the equivalent contagion effect, here as the spatial-lag variable goes from 0 to 1. For a comparable homophilic-selection effect of behavioral similarity on network ties, we ask: what are the odds that i will choose to connect to another actor who behaves similarly over to one who behaves dissimilarly? In the Siena model, if all i's ties are between dissimilar behavioral types (average similarity 0), choosing to connect to a similar behavior type increases covariate-(behavior)-related similarity from 0 to 1, and the odds of forming such a tie (relative to choosing a tie with a dissimilarly behaving actor) are $\exp(\beta_{\text{net}})$ to 1. In the simple logit model, an indicator variable turns on (off) when a potential network partner behaves similarly (dissimilarly), so the relevant odds calculation is straightforward.

Several issues remain. First, the Siena effects described above assume that i is chosen to make a behavioral or network change, but not all actors will be selected in that DGP. With rate of event occurrence set to 1, the probability an actor i is selected during an interobservational period is about .63 (the negative exponential cumulative distribution evaluated at 1). The selection-adjusted odds ratio is $.63 \times 2.714 = 1.71$. Second, the logit models are dynamic in a way the Siena DGP is not. Specifically, the logit parameter estimates determine transition probabilities for a first-order Markov chain. Accordingly, the comparable odds ratios would derive from the steady-state (stationary) distribution of the Markov chain. Finally, even with these adjustments, the logit models are still misspecified, especially the network model because the true DGP only allows actors to make one change at a time, a restriction the simple logits do not impose. Consequently, the logit model will likely underestimate the size of the relevant selection effects. Many ties that would have formed among similarly behaving actors absent this restriction, will not be formed in the Siena DGP.

Table 2 compares these behavioral homophilic selection and behavioral contagion effects using the estimates from the lower event rate scenarios. We provide the mean effect estimate and the standard deviation and root mean squared errors (RMSE) for these estimates. The relative bias and efficiency we summarized regarding the structural parameter estimates transfer to the effects estimates. While the mean Siena effect estimates frequently exhibit less bias, the simple logistic strategy outperforms the Siena

Table 2 Monte Carlo simulation results for comparable effects (true effect = 1.72)

Sample: $N = 30$, $T = 5$				*Sample:* $N = 30$, $T = 11$			
Parameter	*Result*	*SIENA*	*Simple logit*	*Parameter*	*Result*	*SIENA*	*Simple logit*
Network	Mean	2.068	1.512	Network	Mean	1.580	1.410
Selection CF	SD	1.836	0.320	Selection CF	SD	0.491	0.172
$(0 \rightarrow 1)$	RMSE	1.869	0.381	$(0 \rightarrow 1)$	RMSE	0.510	0.353
Behavior	Mean	1.763	1.800	Behavior	Mean	1.732	1.735
Contagion CF	SD	1.621	0.922	Contagion CF	SD	0.740	0.577
$(0 \rightarrow 1)$	RMSE	1.622	0.926	$(0 \rightarrow 1)$	RMSE	0.740	0.577
Sample: $N = 50$, $T = 5$				*Sample:* $N = 50$, $T = 11$			
Parameter	*Result*	*SIENA*	*Simple logit*	*Parameter*	*Result*	*SIENA*	*Simple logit*
Network	Mean	1.865	1.539	Network	Mean	1.598	1.462
Selection CF	SD	0.874	0.268	Selection CF	SD	0.392	0.145
$(0 \rightarrow 1)$	RMSE	0.886	0.323	$(0 \rightarrow 1)$	RMSE	0.410	0.294
Behavior	Mean	1.951	1.761	Behavior	Mean	1.778	1.716
Contagion CF	SD	1.353	0.598	Contagion CF	SD	0.854	0.438
$(0 \rightarrow 1)$	RMSE	1.373	0.600	$(0 \rightarrow 1)$	RMSE	0.856	0.438

estimates across the board in RMSE terms, often by large margins. In the small sample case ($N = 30$, $T = 5$), for example, the RMSE from the simple logit model for behavior homophilic selection effect is a little over $\frac{1}{5}$ the size of corresponding RMSE calculated from the Siena estimates.

The summary upshot seems to be: neither strategy can offer much hope of learning anything reliable about coevolution when event rates are high—which may be discernable by high amounts of change in networks and/or behaviors between observational periods that seem substantively far apart in that actors could have undertaken many interim actions. At low event rates, conversely, both strategies work generally acceptably and roughly comparably well, with an efficiency advantage and simplicity perhaps favoring the logistic strategy.

4 Illustration: Military Alliances and Conflict Behavior

We illustrate with an analysis of the alliance formation and conflict behavior of great powers during the first half of the twentieth century (Levy 1981), a period of much variation in conflict behavior (hardly unique to that period) and of multipolarity during which military alliances were in flux (rarer). We suspect alliance ties and conflict behavior coevolve. States self-select into alliances, and these decisions are plausibly driven by homophilic or heterophilic preferences. More aggressive/pacific states may seek likewise aggressive/pacific allies, or the opposite may hold. At the same time, conflict behavior is contagious through alliances. Indeed, that states would be drawn into their allies' conflicts is key to most alliances (e.g., Kimball 2006).

Table 3 presents our estimates. Model 1, columns 1A and 1B, applies our estimator with contagion of dichotomous behaviors, with connection and selection occurring through observed dichotomous ties; that is, the system of equation (6) above. Model 2 adds covariates. Specifically, the conflict behavior model includes regime type (Polity score) and national capabilities (Correlates of War [COW] Composite Index of National Capability [CINC] score). The alliance ties model includes regime similarity, given as one minus the absolute value of the difference in polity scores divided by the maximum difference (20), and the absolute value of the CINC differences, measuring power asymmetry. These are also *covariate similarity* measures, but in an exogenous regressor (as assumed here anyway), unlike our behavioral homophily regressor. The CINC scores are scaled to sum to one across all countries, so both our regime similarity and power asymmetry measures lie between 0 and 1. We suspect the disturbances (from the underlying choice models) correlate across equations in this application (unlike in our simulation DGP), so equation-by-equation estimation would produce consistent, if inefficient, parameter estimates, while conventional standard error estimates would be inaccurate. Accordingly, we will report robust standard errors using a

Table 3 Estimation results: military-alliance ties and conflict behavior

	Discrete-time ours				*Continous-time Snijders et al. (by Siena)*	
(Markov models>>)	*(1A) Alliance networks*	*(1B) MIDs behavior*	*(2A) Alliance networks*	*(2B) MIDs behavior*	*(3A) Alliance networks*	*(3B) MIDs behavior*
Temp lag	4.99**	1.45**	5.04**	1.33**	—	—
	(0.14)	(0.27)	(0.14)	(0.28)		
Dyad-specific						
Previous MIDs	−0.39**	—	−0.42**	—	−4.67	
similarity (behavior)	(0.15)	—	(0.15)	—	(5.14)	
Regime similarity	—	—	0.45	—	—	—
		(0.28)				
Power asymmetry	—	—	6.56**	—	—	—
			(1.51)			
State-specific						
Previous alliance tie	—	0.85**	—	0.74*		1.71
(Network)		(0.31)		(0.33)		(4.06)
Polity	—	—	—	−0.05		—
				(0.03)		
National capability	—	—	—	10.96*		—
				(4.69)		
Loglikelihood	−225.08	−179.50	−223.05	−175.34		

Note. Models (1) and (2) report Seemingly Unrelated Regressions-robust standard errors. These models also include unit fixed effects (not reported).
* .05 level of significance; ** .01 level of significance.

systems sandwich estimator of the variance-covariance matrix. Models 1 and 2 also contain country or dyad unit indicators. Model 3 (3A and 3B) applies the Siena continuous-time Markov model/estimator to "snapshots" at 5-year intervals (1900, 1905, …,1950) of the great powers' alliance networks and conflict behavior. For the network statistic, we used covariate (behavior)-related similarity, and for the behavior statistic, we used the average similarity effect. The former is defined as $s_i^{\text{net}} = \sum_j x_{ij}(\text{sim}_{ij}^z - \widehat{\text{sim}^z})$, where the similarity scores are $\text{sim}_{ij}^z = \frac{\Delta - |z_i - z_j|}{\Delta}$, Δ being the maximum sample difference, and $\widehat{\text{sim}^z}$ is the mean of all similarity scores. The latter behavior statistic is defined as $s_i^{\text{beh}} = \frac{\sum_j x_{ij}(\text{sim}_{ij}^z - \widehat{\text{sim}^z})}{\sum_j x_{ij}}$. (The same statistics as in the Monte Carlo simulations.[18])

With the first two models, we find evidence (1) of heterophily—pacific powers are more likely to ally/maintain alliances with aggressive powers—and (2) that conflict behavior is (positively) contagious through alliances. The Model 1 estimates, for example, imply that the average probability of a great power engaging in a militarized dispute given no involvement in the previous period rises from .55 to .74 when one's allies change from pacific to aggressive behavior in the previous period. The heterophily effects are smaller. The average probability that an alliance of great powers will persist period-to-period is about .92 when the alliance partners behaved dissimilarly. With both parties pacific or both aggressive, this probability drops to below .89. Note that the sustaining influence of asymmetry extends beyond behavior to include capabilities (see Model 2A); relatively, weak countries are more likely to ally and stay allied with relatively powerful partners. This supports theoretical expectations from the alliance formation literature regarding power asymmetry and alliance formation (Morrow 1991). The signs of our Siena-model estimates also suggest behavioral contagion and heterophilic selection, but these estimates are not statistically significant. Model 3's small Wald statistics seem to confirm the finding from our Monte Carlo analysis that Siena is relatively less efficient and powerful than our simpler spatial-logistic strategy.[19]

[18]Snijders (2001) and Ripley and Snijders (2010) offer many alternatives and much further discussion.
[19]We also tried a Siena model with the same covariates as in column 2, but we do not report it because the estimator failed to produce a positive-definite covariance matrix.

Overall, our empirical results suggest that the conflict behavior of great powers and their military alliance networks coevolve. One significant implication of this is that great power relations may be path dependent. As noted above, our theoretical models suggested that the test of endogenous coevolution, that is, of contagion in behavior and selection by behavior jointly, was also a test of path dependence. Namely, we test whether H_0: $\beta_{\text{contagion}} \times \beta_{\text{homophily}} = 0$. Using a Wald strategy, and the Delta method asymptotic linear approximation for the estimated variance of $\widehat{\beta_c} \times \widehat{\beta_h}$, the χ^2 statistics and associated probabilities are 3.56 and 0.0591 for Model 1, 3.25 and 0.0713 for Model 2: suggestive, if not overwhelming, evidence for path dependence.

5 Conclusion

Theoretically, this paper built a discrete-time Markov type-interaction model in which the behaviors of actors and the networks that connect them coevolve. One interesting implication of the model is that it produces history-dependent behavior possibly including path dependence. We suspect such network-behavior coevolution, and with it the possibility of path dependency, manifests importantly in many areas of social science inquiry. To evaluate this possibility empirically, we built from the theoretical model a spatial-lag logistic model of coevolution that combines a simple time-lagged spatial-lag model of contagious behavior with a simple p^*-logit model of behavioral homophilic network formation (which is also a time-lagged spatial-lag model). We evaluated and compared the performance of this proposed simple estimation strategy and/with the extant alternative from social-network analysis, Snijder's Siena model of node behavior and tie formation. Neither strategy seemed capable of gaining traction in environments where a great deal of change in connectivity and behavior occurs within periods between observations, but either seemed at least somewhat capable of doing so in more favorable scenarios. There, our analyses suggested the simple spatial-logistic strategy had simplicity, efficiency, and power advantages making it an attractive alternative to the more sophisticated Siena. Finally, we demonstrated the feasibility and utility of this theoretical and statistical framework by applying it to analyze the patterns of alliance formation and conflict behavior among the great powers during the first half of the twentieth century. A test for path dependence that we derived from these theoretical and empirical efforts suggests that conflict alliance formation patterns in that period were likely coevolutionary and so path dependent.

References

Beck, N., K. S. Gleditsch, and K. Beardsley. 2006. Space is more than geography: Using spatial econometrics in the study of political economy. *International Studies Quarterly* 50(1):27–44.

Brueckner, J. K. 2003. Strategic interaction among governments: An overview of empirical studies. *International Regional Science Review* 26:175.

Franzese, R. J., and J. C. Hays. 2006. Strategic interaction among EU governments in active labor market policy-making: Subsidiarity and policy coordination under the European employment strategy. *European Union Politics* 7:167–89.

———. 2007a. Empirical models of international capital-tax competition. In *International taxation handbook: Policy, practice, standards and regulations*, eds. C. Read and G. Gregoriou, 43. Oxford, UK: Cima.

———. 2007b. Spatial econometric models of cross-sectional interdependence in political science panel and time-series-cross-section data. *Political Analysis* 15:140–64.

———. 2008a. Empirical models of spatial interdependence. In *Oxford handbook of political methodology*, eds. J. M. Box-Steffensmeier, H. Brady, and D. Collier. Oxford, UK: Oxford University Press.

———. 2008b. Interdependence in comparative politics: Substance, theory, empirics, substance. *Comparative Politics Studies* 41:742–80.

Franzese, R. J., J. C. Hays, and L. M. Schaffer. 2010. Spatial, temporal, and spatiotemporal autoregressive probit models of interdependent binary outcomes: Estimation, interpretation, and presentation. Paper presented at the annual meeting of the American Political Science Association, Washington, DC.

Hägerstrand, T. 1967. *Innovation diffusion as a spatial process.* Chicago, IL: University of Chicago Press.

Hays, J. C., A. Kachi, and R. J. Franzese. 2010. A spatial model incorporating dynamic, endogenous network interdependence: A political science application. *Statistical Methodology* 7:406–28.

Hoff, P. D., A. E. Raftery, and M. S. Handcock. 2002. Latent space approaches to social network analysis. *Journal of the American Statistical Association* 97:1090–9.

Hoff, P. D., and M. D. Ward. 2004. Modeling dependencies in international relations networks. *Political Analysis* 12:160–75.

Hoff, P. D., and A. Westveld. 2007. A Bayesian mixed effects model for longitudinal social network data. Working paper, University of Washington.

Jackson, J. E., and K. Kollman. 2007. Models of path dependence with an empirical application. Paper presented at the annual Political Methodology Conference, State College, PA.

Jackson, M. O. 2008. *Social and economic networks*. Princeton, NJ: Princeton University Press.

Kelejian, H. H., and I. R. Prucha. 2010. Specification and estimation of spatial autoregressive models with autoregressive and heteroskedastic disturbances. *Journal of Econometrics* 157(1):53–67.

Kimball, A. L. 2006. Alliance formation and conflict initiation: The missing link. *Journal of Peace Research* 43(4):371–89.

Koger, G., S. Masket, and H. Noel. 2009. Partisan webs: Information exchange and party networks. *British Journal of Political Science* 39:633–53.

———. 2010. Cooperative party factions in American politics. *American Politics Research* 38(1):33.

Lazer, D. 2001. The co-evolution of individual and network. *Journal of Mathematical Sociology* 25(1):69–108.

Leenders, R. T. 1997. Longitudinal behavior of network structure and actor attributes: Modeling interdependence of contagion and selection. In *Evolution of social networks*, eds. P. Doreian and F. N. Stokman. Amersterdam, Netherlands: Gordon and Breach Publishers.

Levy, J. S. 1981. Alliance formation and war behavior: An analysis of the great powers, 1495-1975. *Journal of Conflict Resolution* 25:581–613.

Morrow, J. D. 1991. Alliances and asymmetry: An alternative to the capability aggregation model of alliances. *American Journal of Political Science* 35:904–33.

Page, S. E. 2006. Path dependence. *Quarterly Journal of Political Science* 1(1):87–115.

———. 2007. Type interaction models and the rule of six. *Economic Theory* 30:223–41.

Ripley, R. M., and T. A. Snijders. 2010. Manual for SIENA, version 4.0. Oxford, UK: Department of Statistics, Nuffield College, University of Oxford. http://www.stats.ox.ac.uk/∼snijders/siena/RSiena_Manual.pdf.

Simmons, B., F. Dobbin, and G. Garrett. 2006. Introduction: The international diffusion of liberalism. *International Organization* 60:781–810.

Snijders, T. A. 1997. Stochastic actor-oriented models for network change. In *Evolution of social networks*, eds. P. Doreian and F. N. Stockman, 149–72. Amsterdam, Netherlands: Gordon and Breach Publishers.

———. 2001. The statistical evaluation of social network dynamics. *Sociological Methodology* 31:361–95.

———. 2005. Models for longitudinal network data. In *Models and methods in social network analysis*, eds. P. J. Carrington, J. Scott, and S. Wasserman, chap. 11. New York: Cambridge University Press.

Snijders, T. A., C. Steglich, and M. Schweinberger. 2007. Modeling the co-evolution of networks and behavior. In *Longitudinal models in the behavioral and related sciences*, eds. K. van Montfort, H. Oud, and A. Satorra, chap. 3, 41–71. Mahwah, NJ: Lawrence Erlbaum.

Steglich, C., T. A. Snijders, and P. West. 2006. Applying SIENA: An illustrative analysis of the co-evolution of adolescents' friendship networks, taste in music, and alcohol consumption. *Methodology: European Journal of Research Methods for the Behavioral and Social Sciences* 2:48–56.

Tobler, W. 1970. A computer movie simulating urban growth in the detroit region. *Economic Geography* 46:234–40.

Walker, R. W. 2007. Path, phat, and state dependence in estimating observation-driven Markov models. Paper presented at the 24th Summer Meeting of the Society of Political Methodology, State College, PA.

Wasserman, S. 1980a. Analyzing social networks as stochastic processes. *Journal of the American Statistical Association* 75:280–94.

———. 1980b. A stochastic model for directed graphs with transition rates determined by reciprocity. *Sociological Methodology* 11:392–412.