History Dependence in Network-Behavior Coevolution: A Type-Interaction Model Merging Spatial-Econometric and Network-Analytical Approaches

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Abstract

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 variable 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, we can identify the same three processes as potential sources of network effects and network formation. From that perspective, actors' self-selection into networks (by, e.g., behavioral *homophily*) and contagious behavior likewise demands theoretical and empirical models in which networks and behavior *coevolve* over time. This paper begins building such modeling by, on the theoretical side, extending a Markov type-interaction model to allow endogenous tie-formation, and, on the empirical side, merging a simple *spatial-lag* logit model of contagious behavior with a simple *p-star* logit model of network formation, building this synthetic discrete-time empirical model from the theoretical base of the modified Markov type-interaction model. One interesting consequence of network-behavior coevolution—identically: endogenous patterns of spatial interdependence—emphasized here is how it can produce history-dependent political dynamics, possibly including equilibrium *phat* and path dependence (Page 2006). The paper explores these implications, and then concludes with a(n extremely preliminary) demonstration of the approach to alliance formation and conflict behavior among the great powers in the first half of the twentieth century.

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1 Introduction

From a network-analytic perspective: Networks are ubiquitous. Whether speaking of friendship or familiarity relations among individuals, trade or conflict relations among states, predator-prey relations in ecosystems, or any other sets of relations (*a.k.a.*: ties, connections, edges, etc.) among sets of units (*a.k.a.*: agents, actors, nodes, etc.), networks are essentially everywhere. And networks usually matter. *Network effects*, arising from various structural aspects of the network, or from actors' positions in the network, or from other actors through the network of connections, often importantly impinge upon the behaviors, opinions, outcomes, or other characteristics of units. Networks are also commonly endogenous. The units within some particular network typically choose or influence their connections, which are the edges that structure the network. A large challenge empirically for social scientists interested in the theory and substance of *network effects* and *network formation* is that network effects on nodes and the formation of edges between nodes tend to be mutually endogenous and, at the same time, both may be caused by outside factors, i.e., by a third mechanism that we have elsewhere called *common exposure* (Franzese and Hays 2006, 2007b, 2008b,a; Hays et al. 2010). In one archetypal application of social-network analysis, for example, we may observe clusters of smokers and of nonsmokers because smoking is contagious–one acquires the habit from friends or avoids acquisition because one's friends abstain–or because smokers choose to hang with smokers and nonsmokers with nonsmokers: homophily by behavior-type–or we may observe clustering of smokers and nonsmokers 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 propensity to smoke and friendship formation.

We have made these arguments before from a spatial-econometric perspective (Franzese and Hays 2006, 2007b,a, 2008b,a, 2009; Hays et al. 2010): 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 Beck et al. (2006)'s pithy title reminds in corollary: "Space is More than Geography". The substantive content of the proximity in Tobler's Law, and 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 carefully elaborate from those disciplinary perspectives the multifarious mechanisms by which contagion may arise. Simmons and colleagues (Simmons and Elkins 2004; Elkins and Simmons 2005; Simmons et al. 2006) offer a list for international relations: *coercion*, *competition*, *learning*, and *emulation* (to which one should add *relocation diffusion* (Hägerstrand 1967, 1970). In fact, as, e.g., Brueckner (2003) showed, strategic interdependence, i.e., 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, the 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 similarly to similar exposure to similar 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*). We may find states' adoptions of some economic treaty, for example, to cluster geographically or along other dimensions of proximity, e.g., 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 according to some variable on which we observe their proximity (volume of trade between them) because being co-signatories affects that variable (spurs bilateral trade).

Whether from the network-analytic or the spatial-econometric perspective, accurately distinguishing and gauging empirically the role and strength of these alternative processes–common exposure, contagion, and selection; a.k.a., node effects, network effects, and network formation–is difficult, because the processes manifest empirically similarly, but also crucial because the theories and policy-intervention advice supported by any observed spatial-*cum*-network phenomenon hinges critically on whether (or the relative degrees to which) the observed phenomena arise from contagion/network-effects, selection/network-formation, or common exposure. The substance of the situations and how policies might best intervene in them vary critically depending on whether state signatories cluster in pockets of dense trade relations because those states tend to experience similar exogenous conditions that favor signing, or because the signing by some states spurs their trading partners to sign, or because the treaty fosters trade between co-signatories. Likewise, whether (non)smokers/(non)smoking clusters in social networks because having smoking or nonsmoking friends spur one to adopt the behavior also, because (non)smokers tends to acquire friends who also (do not) smoke, or because some clustered exogenous internal or external conditions, some sociodemographics for instance, affect both one's (non)smoking behavior and with whom one becomes friends.

As we have also argued (and to some extents demonstrated, analytically, by simulation, and/or in applications) elsewhere, drawing effective distinctions and obtaining accurate estimates empirically of any of these separate processes requires great care and attention to specification (including measurement) of all three of components. That is, regardless of whether one's interests center on network effects, the contagiousness of smoking for instance, or on network formation, what determines trade or conflict patterns for instance, one must model well both the network-effects/contagion and the network-formation/selection, and also whatever relevant external factors important to either process.[1]

Our project here aims to develop a framework for theoretical modeling and empirical specification, estimation, and interpretation of social phenomena with (common exposure and) simultaneous contagion and selection, that is, of mutually endogenous network effects and network selection, i.e., of the coevolution of actor behavior and their network connections. Identically from a spatial-econometric perspective, this means models with exogenous covariates reflecting common exposure, spatial-lag contagion, and a pattern of spatial connectivity, i.e., a set of spatial weights, which are (at least in some part) endogenous to behavior. Our theoretical model of such processes builds from

[1]Nor, generally, will causal-inference strategies based on the potential-outcomes framework and assumptions of SUTVA salvage accurate estimation of any of these causal processes without adequate address of all three. SUTVA, in a nutshell, requires that (i) the probability of one unit receiving/taking treatment, (ii) the (constant) magnitude of the treatment, and (iii) the effect of treatment are independent of each other and of any other unit(s) receiving/taking treatment, the sizes of those units' treatments, or the effects of those treatments in those others. These precluded situations are the essence spatial-*cum*-network effects. "The two most common ways in which SUTVA can be violated appear to occur when (a) there are versions of each treatment varying in effectiveness or (b) there exists interference between units" (Rubin 1990:, p. 282). The first of these is called spatial heterogeneity, the second is spatial interdependence: i.e., these are merely the network or spatial effects argued in those literatures to be ubiquitous and central to (at least) all social phenomena.

Markov type-interaction models in the extant literature, 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 in a manner that depends on an exogenously given (possibly exogenously varying) set of connections between actors. These models parallel theoretically extant empirical spatial-lag models of spatial econometrics, notably in the exogeneity of the connectivity matrix, i.e., of the network of connections between actors.[2] In other words, these models expressly disallow network-formation/selection. Accordingly, we extend these extant Markov type-interaction models to incorporate endogenous determination of the connections between units, connections being made or broken endogenously (to an extent that we can vary with parameters of the model) by the previous behavior-types of those units. Likewise, empirically, we merge extent spatial-lag models of interdependent behavior–specifically, a very simple version of the *spatial-lag logit* model[3]–which have typically maintained exogenous connections between units, with extent models of network formation, *p-star* models–specifically the simplest p-star model of independent ties[4]–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, and of network formation with self-selection of actors into networks, specifically of actors' self-selection of the ties between actors according to some (dis)similarity or other function of the actors' behaviors or types (e.g., *homophily*), implies that networks and behavior *coevolve* over time. Both network effects and network formation, i.e., both contagion and selection, are ubiquitous and frequently important across the social sciences (as are the usual plethora of exogenous conditions relevant to both). Therefore, in longitudinal-network or spatiotemporal analyses, scholars must take seriously the modeling of all three processes–common exposure, contagion, and selection–if they are to understand the nature of and properly model and estimate the structure of the coevolutionary dynamics in their data, i.e., if they are to explain accurately the network formation and dynamics and the behavioral decision-making evidenced in their data. In this paper, we emphasize that one of the more interesting consequences of such network-behavior coevolution is that it can produce history-dependent political dynamics, possibly including what (Page 2006) defines as *phat*, path, and/or equilibrium dependence. We first establish theoretically that systems with coevolution can easily generate multiple equilibria (i.e., multiple steady states of the system), using the aforementioned modified Markov type-interaction model extended to allow endogenous tie-formation. The potential of multiple equilibria raises a very difficult empirical question–how sensitive are equilibrium distributions (over types) to the past states? Nor are the stakes in this question merely academically nontrivial. What can be achieved by potential policy interventions today and how we should design policy interventions for the future depend critically on whether and how history matters in phat- or path-dependent ways for the equilibrium attained in the society. To evaluate the empirical magnitude and substantive and statistical significance of coevolutionary dynamics, therefore, we combine as just noted the spatial-lag logit and p-star logit models to develop discrete-time Markov models that

[2]In practice, most theoretical Markov type-interaction models have employed very simple, uniform and universal, and therefore anonymous, connections between actors; i.e., all actors are equally or equiprobably connected to all others and therefore are anonymously exchangeable in this sense. The extension of our model to endogenous selection of ties on the basis of past behavior-types must foresake this anonymity; reformulating the model to keep track of these individuals and dyads itself proved a nontrivial extension.

[3]For now, to start, we employ only a time lag of the spatial-lag dependent variable, and assume this adequate model of the spatiotemporal dynamics of contagion, to evade the multidimensional integration complications of simultaneous spatial-lags in latent-variable models.

[4]Given the assumed conditional independence of the network ties across ties and actors in that simplest model—again, for now, to start—this amounts very simply to a set of $\frac{1}{2}N(N-1)$ simple logits.

can estimate the empirical magnitude and significance of any coevolutionary dynamics in the data. One strength of this empirical approach lies in its direct connection with the theoretical Markov type-interaction model, which allows us to assess the full substantive content of history dependence in observed data and which can provide a foundation for developing statistical tests for history dependence generated by coevolution.

[Somewhere, perhaps here but maybe later or maybe split up some here and some later, goes some further lit review and such. On SIENA (Steglich et al. 2006; Snijders et al. 2007; Snijders 2005); on spatial-econometric approaches to some of these issues (Franzese and Hays 2009; Hays et al. 2010) (AND CITES FOR SPATIOTEMPORAL AND SPATIAL LATENT-VARIABLE); and on empirical models of path dependence (Jackson and Kollman 2007; Jackson 2008; Page 2006, 2007; Walker 2007)]

The rest of the paper proceeds as follows. In the next section, we present a theoretical Markov type-interaction model for N actors, modified to allow allow endogenous tie-formation. In the third section, we propose our statistical model, specified to reflect the theoretical model proposed in the preceding section. The fourth section provides an illustrative application that examines the coevolution of the military alliances and conflict behavior of major powers in the first half of the twentieth century, comparing our proposed model and estimation strategy with Snijders and colleagues' coevolutionary actor-oriented longitudinal-network model, SIENA (Snijders 2005; Steglich et al. 2006; Snijders et al. 2007).[5]

2 The Markov-Chain Model

In this section, we introduce a relatively simple theoretical model of behavior-shaping (network contagion effects) and network-formation (selection) that results in a set of Markov chains. In this model, a group of actors are of certain types and their types change over time as actors are influenced by other actors. Such contagion of actors' types occurs only if the actors are *connected*. We employ the explicit notion of networks (or spatial weights) to characterize 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 focus on behavior-type *homophily* (or *heterophily*), i.e., where network ties are more (or less) likely to form among actors whose types are more (or less) similar in the previous period. The key features of and the additional sources of complexity due to this extension of extant type-interaction models are the following: (1) it introduces the details of which actor interacts with which to represent the effects–specifically, the contagion effects[6]–of networks on actors' behavior and (2) it describes how those interaction patterns, i.e., the networks (or spatial-weights matrices), change endogenously over time based on actors' types in the previous time period, which reflects homophily by behavior-type.

[5] In future work, we intend to compare more fully our empirical model and estimation strategy to Snijders and colleagues' SIENA, including Monte Carlo simulation of properties of parameter, dynamics and steady-state effect estimates. For now, the fourth section contains only a comparison of coefficient estimates for a single substantive application.

[6] As previously mentioned, network effects more generally include three sorts of effects: (i) effects of the network structure (*density* or *hub-and-spoke* structure for instance) on nodes, (ii) effects of nodes' positions within the network (their *centrality* or *betweenness* for example) on nodes' behaviors, and effects through the network of connections of other nodes' characteristics or behaviors on nodes' behaviors (of *alter* on *ego* in network terminology). We focus, for now at least, at first, on the last of these, commonly labeled *contagion* in much of the relevant literatures.

We then demonstrate that the model can produce multiple equilibria. By equilibrium, we mean consistency between actors' behavioral types and their behavior-switching rules (i.e., steady state or fixed point). In coevolution models, multiple equilibrium (distributions of) types are consistent with a single behavior-switching rule. The particular type/behavioral-rule combination that emerges at a given point in time is a function of actors' prior types. In this way, the evolution of behavioral types is history dependent and may be specifically phat, path, or equilibrium dependent.[7]

2.1 The Model

Consider the following discrete-time longitudinal process with N actors. Let $i \in \{1, \cdots, N\}$ denote these N actors and $t \in \{1, 2, \cdots\}$ denote time periods. We distinguish between the *behavior* of an actor and her *behavioral type* (or simply *type*), understanding the latter as the actors' probability of taking action 1. In our initial model here, we assume that behavior is observed and dichotomous, whereas behavior-type is continuous and unobserved by analysts but observed by actors, with contagion and selection occuring by type.[8] In each period, actors choose between *behavior* 1 and 0, e.g., smoking or not, voting or not, taking an aggressive interstate behavior or not, democratizing or not, and so forth. We denote *behavior* of actor i in period t by $s_{it} \in \{1, 0\}$. *Behavioral type* is the probability that an actor chooses behavior 1. We denote the *behavioral type* of actor i in period t by $\sigma_{it} \in [0, 1]$. The state of the system that actors can observe at the end of period t is therefore an N-dimensional vector of types, $\boldsymbol{\sigma}_t = (\sigma_{1t}, \cdots, \sigma_{Nt})$ and a matrix of latent and observed ties between actors to be described subsequently.[9]

We focus first on the Markov chains that explain behavioral type, taking into account the contagion effects of networks among the N actors. We separate three component terms that together determine a *switching probability*–the probability that an actor's behavior at $t + 1$ becomes $s_{i,t+1}$ or $1 - s_{i,t+1}$ from $s_{i,t}$ or $1 - s_{i,t}$. First, we have a component of the probability that the actor chooses behavior 1 that does not depend on the state of the world, which includes both the actor's own and others' types, that she observes at the end of t ($\boldsymbol{\sigma}_t$). Let c_1 denote this part of the probability; i.e., the probability that an actor takes action 1 exogenously to the state at the end of period t. Similarly, let c_0 denote the probability that an actor takes action 0 exogenously of the state of the world she observes at the end of period t. In terms of the three processes that may produce spatial/network association, c_0 and c_1 are the theoretical placeholders for the exogenous external and internal conditions to which actors may have *common exposure*. Lastly, each actor's behavioral decisions (i.e., *ego*'s choices, in network-analytic terminology) can also be influenced by others' (*alters*') types; this is the *contagion* in our terminology. Actors in a given dyad (i and j, where $i \neq j$) influence each other's behavioral type only if they are *connected*. This *connectivity*

[7] Ultimately, we aim to characterize the forms of history dependence–outcome and equilibrium state, phat, path dependency (Page 2006)–that emerge from alternative parameter and starting values and the mappings from the latter to the former.

[8] We envision eventually a range of possible models, with continuous or discrete behavior, observed or unobserved by analysts and/or by actors, with contagion or selection by behavior or type. The theoretical, substantive, and empirical appeal of these alternative models would presumably vary with the application context. We begin with the model described here because we believe it an appealing one for the application to be offered in section four, because this theoretical model maps well into the empirical model to be offered in section three, and because we must begin somewhere.

[9] There is also a corresponding vector of behaviors, $\boldsymbol{s}_t = (s_{1t}, \cdots, s_{Nt})$, which is less germane to the theoretical model here than to the empirical models to come since this theoretical model has type observed and the basis of contagion and selection.

among actors, the *ties* between them, could be friendship, military alliance, trade partnership and so on. Let $\delta_{ij,t} \in [0,1]$ denote the probability that a tie exists between the two actors in dyad ij in period t; we can also interpret this probability, isomorphically in this model, as the strength of the tie.[10] Ties are undirected; undirected ties are equivalent to symmetric spatial-weights matrices. (Extension to the *directed-network/asymmetric-weights-matrices* case is obviously an important next step for the theoretical model.) In this N-actor system, we express contagion, the extent to which others' types influence i's type, as $\sum_{j=1,(j\neq i)}^{N}(\delta_{ij}\sigma_j)/(N-1)$, where we have weighted alters' (j's) influence on ego (i) by $(N-1)^{-1}$ to bound the value of this positive term by 1.[11] This ensures that the total effect of others' types $\sum_{j\neq i}(\tilde{\delta}_{ij}\sigma_j)$ lies in $[0,1]$; then, the way we combine these three components of an actor's type assures that the whole expression for σ_i, which is the *probability* the actor chooses behavior, s_i, equal to 1, is likewise bounded $0 \leq \sigma_i \leq 1$.

Equation (1) describes the transition of actor i's type from period t to $t+1$. This system results in a set of N Markov chains. The second matrix on the right-hand side is the transition-probability matrix. For example, cell $(1,1)$ of the transition-probability matrix represents the probability that actor i chooses behavior 1 given that i chose 1 in the past period, $Pr(s_{i,t+1}=1|s_{it}=1)$. The first term is the probability that i choose behavior 1 exogenously of the state, c_1 and the second term is the weighted average expressing how others' types affect i's choice of behavior. Notice how, by this construction, the weight $(1-c_1-c_0)$ captures the extent to which others' types matter for i's behavioral choice in $t+1$. This gives us parameters within the transition model by which to vary the overall strength of contagion. For instance, in an extreme case where i chooses 1 with probability $c_1 = 0.5$ and 0 with probability $c_0 = 0.5$ exogenously of others' types, then $1-c_1-c_0 = 0$, meaning that i's decision will not be affected by any others to whom she is connected: i.e., the general strength of contagion is 0.

$$
\begin{aligned}
&\begin{pmatrix} \sigma_{i,t+1} \\ 1-\sigma_{i,t+1} \end{pmatrix}' \\
&= \begin{pmatrix} \sigma_{i,t} \\ 1-\sigma_{i,t} \end{pmatrix}' \begin{pmatrix} c_1 + (1-c_1-c_0)\frac{\sum_{j\neq i}(\delta_{ij}\sigma_j)}{N-1} & 1-\left[c_1 + (1-c_1-c_0)\frac{\sum_{j\neq i}(\delta_{ij}\sigma_j)}{N-1}\right] \\ 1-\left[c_0 + (1-c_0-c_1)\frac{\sum_{j\neq i}(\delta_{ij}(1-\sigma_j))}{N-1}\right] & c_0 + (1-c_0-c_1)\frac{\sum_{j\neq i}(\delta_{ij}(1-\sigma_j))}{N-1} \end{pmatrix} \qquad (1) \\
&= \begin{pmatrix} \sigma_{1,t} \\ 1-\sigma_{1,t} \end{pmatrix}' \begin{pmatrix} z_t^{11} & 1-z_t^{11} \\ 1-z_t^{00} & z_t^{00} \end{pmatrix}
\end{aligned}
$$

We focus next on the Markov chains that explain tie-formation probabilities (Equation (2)). The unit of each entry is now a dyad (i,j). The probability that the two actors i and j in a dyad form a tie (e.g., "are friends") in period $t+1$ is denoted by $\delta_{ij,t+1} \in [0,1]$. In conventional type-interaction models, these tie-formation probabilities are assumed exogenous. By contrast,

[10] These alternatives are mathematically identical in this particular model because contagion and selection occur by the continuous, observed type and strength, not the dichotomous, unobserved behavior and tie. In models with other combinations of these conditions, the alternative interpretations may differ slightly due to the distinction between a behavior or tie of strength 1 with probability p and strength 0 with probability $1-p$ versus a behavior or tie of strong p.

[11] The row standardization common in spatial econometrics, or the spectral normalization that Kelejian and Prucha (2009) recommend instead, would also serve to bound $0 \leq \sum_{i\neq j}(\delta_{ij}\sigma_j) \leq 1$.

the potential for endogenous ties—for instance via behavior-type homophily: types seeking like types—is a crucial aspect of our model. In our model, similarity or dissimilarity of actors' types at t partially determines the tie-formation probabilities in the next time period. For example, the transition probability for a dyad to be connected in period $t+1$ given that they were already connected at t is $c_2 + (1-c_2)(1-(\sigma_i - \sigma_j)^2)$. Analogously to c_0 and c_1, the term c_2 reflects exogenous factors' contributions to these two actors' forming a tie, regardless of the state. The component $1-(\sigma_i - \sigma_j)^2$ of the remaining term captures the extent to which similarity of i's and j's types at the end of period t affect their probability of being connected in period $t+1$. This quantity increases as the two types, σ_i and σ_j, become more similar; so, to the degree that $c_2 < 1$, our model exhibits homophiletic tie-formation by behavior-type: two individuals are more likely to form a friendship tie if both smoke, two countries more likely to ally if their conflict behaviors are similar, two members of Congress more likely cosponsor a bill the more similar their political ideologies, etc.

$$
\begin{aligned}
&\begin{pmatrix} \delta_{ij,t+1} \\ 1-\delta_{ij,t+1} \end{pmatrix}' \\
&= \begin{pmatrix} \delta_{ij,t} \\ 1-\delta_{ij,t} \end{pmatrix}' \begin{pmatrix} c_2 + (1-c_2)(1-(\sigma_i-\sigma_j)^2) & 1-\{c_2+(1-c_2)(1-(\sigma_i-\sigma_j)^2)\} \\ 1-\{c_2+(1-c_2)(\sigma_i-\sigma_j)^2\} & c_2+(1-c_2)(\sigma_i-\sigma_j)^2 \end{pmatrix} \\
&= \begin{pmatrix} \delta_{1,t} \\ 1-\delta_{1,t} \end{pmatrix}' \begin{pmatrix} w_t^{11} & 1-w_t^{11} \\ 1-w_t^{00} & w_t^{00} \end{pmatrix}
\end{aligned}
\tag{2}
$$

The systems of differential equations (1) and (2) complete our theoretical model of network-behavior coevolution, i.e., of jointly endogenous contagion and selection. The long-run steady-state (LRSS) equilibrium of this system consists of a vector of each actor's type and each dyad's tie-forming probability, $(\boldsymbol{\sigma}, \boldsymbol{\delta})$. This LRSS equilibrium can be obtained by solving (1) and (2) for $\boldsymbol{\sigma}$ by setting $\sigma_{i,t+1} = \sigma_{i,t}$ and $\delta_{ij,t+1} = \delta_{ij,t}$, $\forall i, j \in \{1, \cdots, N\}$. In any given social-science context, our interests may lie primarily in the LRSS equilibria and/or the intertemporal dynamic of actors' types, $\boldsymbol{\sigma}$, and/or in the LRSS and/or dynamics of tie-formation probabilities, $\boldsymbol{\delta}$. Of course, our interests may also involve all of the above equally. In any case, the states and the equilibria are fully and best characterized by vectors of types and tie-probabilities both, due to the endogeneity generated by homophily. For compactness only, our exposition will highlight the equilibria of behavior types and (network) ties.[12]

2.2 Example: Three-Actor System

To illustrate the existence of multiple equilibria, consider the following example with three actors, $(1, 2, 3)$, and so three possible undirected edges, $(12, 13, 23)$. With probability 0.5—-perhaps better conceived as: *to an extent of 50%*—actor i chooses behavior 0 exogenously of the state at the end of t, $(c_0 = 0.5)$. To an extent of 0.2, actor i takes behavior 1 exogenously of the state, $(c_1 = 0.2)$.

[12] Exploration and discussion of our illustrative example is currently incomplete. In future drafts, we plan to develop the example further and characterize insofar as possible the mapping from parameter and starting values to paths and equilibria.

To the remaining extent, $1-c_0-c_1=0.3$, actor i's type in $t+1$ will be influenced by the types of others to whom i is connected. In any dyad, the two actors, i and j, form an undirected tie with/to probability/extent 0.5 exogenously of the state, ($c_2=0.5$). To the remaining 0.5 extent, actors' homphiletic preferences determine their ties. This leaves the following system of equations, a set of six equations of motion, one for each of the three actors' types and one for each of the three dyads' tie-formation processes:

$$\begin{cases} \sigma_{1,t+1}=\sigma_{1,t}\Big(0.2+0.3\frac{(\delta_{12,t}\sigma_{2,t}+\delta_{13,t}\sigma_{3,t})}{2}\Big)+(1-\sigma_{1,t})\Big(1-\Big(0.5+0.3\frac{\{\delta_{12,t}(1-\sigma_{2,t})+\delta_{13,t}(1-\sigma_{3,t})\}}{2}\Big)\Big) \\ \sigma_{2,t+1}=\sigma_{2,t}\Big(0.2+0.3\frac{(\delta_{12,t}\sigma_{1,t}+\delta_{23,t}\sigma_{3,t})}{2}\Big)+(1-\sigma_{2,t})\Big(1-\Big(0.5+0.3\frac{\{\delta_{12,t}(1-\sigma_{1,t})+\delta_{23,t}(1-\sigma_{3,t})\}}{2}\Big)\Big) \\ \sigma_{3,t+1}=\sigma_{3,t}\Big(0.2+0.3\frac{(\delta_{13,t}\sigma_{1,t}+\delta_{23,t}\sigma_{2,t})}{2}\Big)+(1-\sigma_{3,t})\Big(1-\Big(0.5+0.3\frac{\{\delta_{13,t}(1-\sigma_{3,t})+\delta_{23,t}(1-\sigma_{2,t})\}}{2}\Big)\Big) \\ \delta_{12,t+1}=\delta_{12,t}\Big(0.5+0.5(1-(\sigma_{1,t}-\sigma_{2,t})^2)\Big)+(1-\delta_{12,t})\Big(1-(0.5+0.5(1-(\sigma_{1,t}-\sigma_{2,t})^2))\Big) \\ \delta_{13,t+1}=\delta_{13,t}\Big(0.5+0.5(1-(\sigma_{1,t}-\sigma_{3,t})^2)\Big)+(1-\delta_{13,t})\Big(1-(0.5+0.5(1-(\sigma_{1,t}-\sigma_{3,t})^2))\Big) \\ \delta_{23,t+1}=\delta_{23,t}\Big(0.5+0.5(1-(\sigma_{2,t}-\sigma_{3,t})^2)\Big)+(1-\delta_{23,t})\Big(1-(0.5+0.5(1-(\sigma_{2,t}-\sigma_{3,t})^2))\Big) \end{cases} \tag{3}$$

Solving this system for the steady-state σ_i's and δ_i's, we get at least the following four sets of equilibria:

$$\begin{cases} 1.\{\sigma_1=\sigma_2=\sigma_3=\frac{5-3\delta_{12}}{13-6\delta_{12}}\} \text{ with } \{0\le\delta_{12}=\delta_{13}=\delta_{23}\le 1\}, \\ 2.\{\sigma_1=\sigma_3=\frac{206-69\delta_{13}}{559-138\delta_{13}},\ \sigma_2=\frac{197+159\delta_{13}-552\delta_{13}^2}{559-138\delta_{13}}\} \text{ with } \{\delta_{12}=\delta_{23}=\frac{1}{2},\ 0\le\delta_{13}\le\frac{1735-7\sqrt{45121}}{1104}\}, \\ 3.\{\sigma_1=\frac{7}{23}+\frac{3}{23}\sigma_2,\ \frac{137}{421}\le\sigma_2=\sigma_3\le\frac{206}{559}\}, \text{ with } \{\delta_{12}=\delta_{13}=\frac{1}{2},\ \delta_{23}=\frac{206-559\sigma_3}{69-138\sigma_3}\}, \\ 4.\{\sigma_1=\sigma_2=\frac{206-69\delta_{12}}{559-138\delta_{12}},\ \sigma_3=\frac{197-51\delta_{12}}{559-138\delta_{12}}\} \text{ with } \{0\le\delta_{12}\le 1,\ \delta_{13}=\delta_{23}=\frac{1}{2}\}. \end{cases} \tag{4}$$

Notice that the LRSS include both *pooling* and *separating* equilibria. The first equilibrium category exhibits complete pooling of behavior and of type, whereas the others all exhibit the same partial-pooling pattern of separation. In equilibria 2, for instance, actors 1 and 3 choose common behavior-type and actor 2 will connect with them on a $50-50$ basis. Equilibria 3 and 4 are analogous, with two actors pooling behavior-type and the other connecting to each of them with probability 0.5.

2.3 Comparison with the No-Coevolution Model: Three-Actor Case

Comparing the LRSS equilibrium results from our type-interaction model to those from conventional models, which do not allow for the coevolutionary dynamic generated by simultaneous homophily and contagion, may also be useful. Table 1 summarizes the results of LRSS equilibrium analyses for the four combinations of c_0, c_1, and c_2 values that produce scenarios with the four combinations of with and without behavior-type contagion and behavior-homophiletic selection in our model.

Table 1: Comparison of Type-Interaction Models

	No Contagion ($c_0 + c_1 = 1$)	**Contagion** ($c_0 + c_1 \neq 1$)
Exogenous Tie-Formation ($c_2 = 1$)	*No contagion in actors' behavior-types, and tie formation is also exogenous to behavior type.* **Unique equilibrium**: $\sigma_i = 1 - c_0$, given any $0 \leq \delta_i j \leq 1$, $\forall ij$.	*Behavior type is contagious, but tie formation is exogenous to behavior type.* **Unique equilibrium**: σ_i varies, given $\delta_i j$, $\forall ij$.
Endogenous Tie-Formation ($c_2 \neq 1$) (homophily)	*No contagion in actors' behavior-types, but tie formation is endogenous to behavior types, with actors more likely to form ties with similar behavior-types (homophily).* **Unique equilibrium**: $\sigma_i = 1 - c_0$, with any $0 \leq \delta_i j \leq 1$, $\forall ij$.	[Our model] *Behavior type is contagious, AND tie formation is endogenous to behavior type, with actors more likely to form ties with similar behavior-types (homophily).* **Multiple equilibria**: examples given in Equation 4.

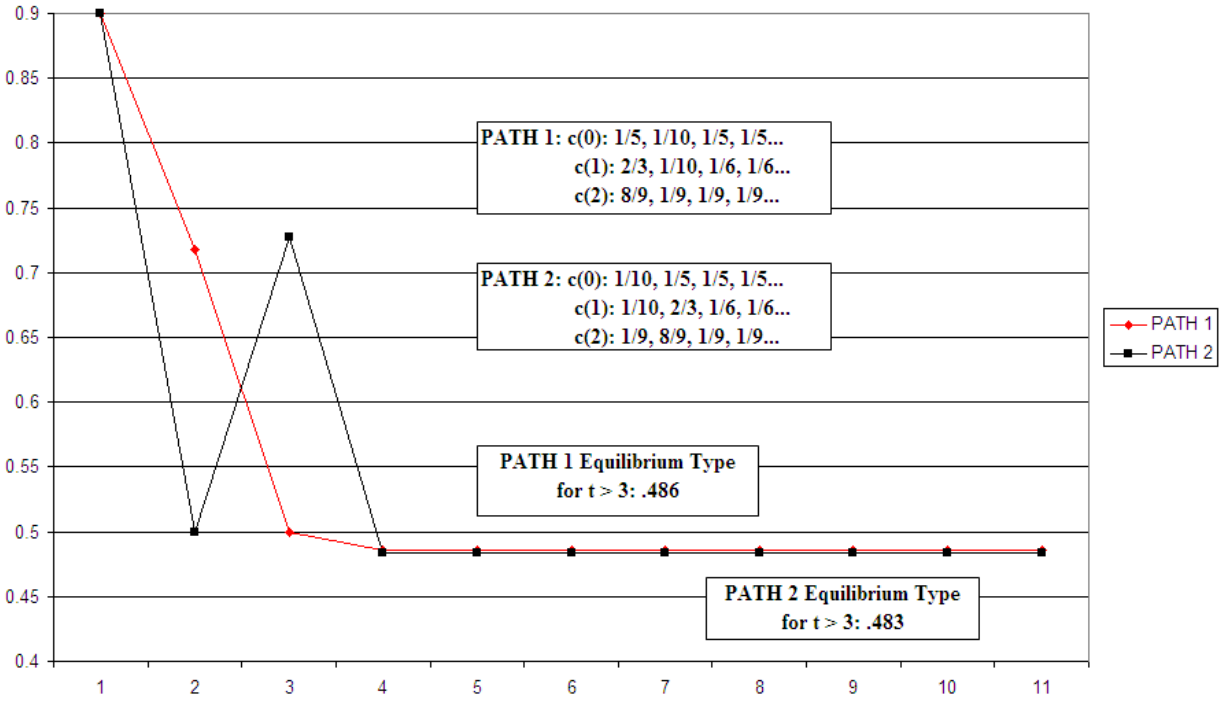
The upshot is simple: multiple equilibria arise ifthe model exhibits both behavior-type contagion and behavior-homophiletic selection. This will suggest the form of a possible test for evidence of equilibrium history-dependence (Page 2006) in the empirical model proposed below.

2.4 Illustration of the Equilibrium Dependence in Types

Figure 1 illustrates the equilibrium path-dependence (Page 2006) possible in our model, given behavior-type contagion and behavior-homophiletic selection. The figure illustrates the dynamics and LRSS equilibrium behavior-type for actor 2 in a three-actor system like the one in our example. We consider two scenarios, labeled Path 1 and Path 2, that differ only by the sequence of exogenous values for c_0, c_1, and c_2 fed actor 2 our three actors. The early part of the sequence differs—namely: the first two vectors (c_0, c_1, and c_2) reverse order for actor two–but the history of exogenous factors from period 3 onward is constant in and the same across both scenarios.

As one can see, the equilibria differ, and this difference does not fade in time, no matter how far into the future we may have taken the figure. Notice that the starting behavior-type and the set of values of (c_0, c_1, and c_2) are the same. Showing that differences in either of these produced differences in LRSS equilibria would have shown equilibrium initial-conditions sensitivity or equilibrium *phat*, i.e. *set*, dependence. Only the sequence of (c_0, c_1, and c_2) differs, so this is true path-dependence, according to Page (2006).

Figure 1: Illustration by Simulations: Equilibrium Dependence in Actor i's Behavioral Type



	PATH 1					PATH 2				
Time period→	1	2	3	4	⋯	1	2	3	4	⋯
$c_0(1)$	$\frac{1}{5}$	$\frac{1}{10}$	$\frac{1}{5}$	$\frac{1}{5}$	⋯	$\frac{1}{10}$	$\frac{1}{5}$	$\frac{1}{5}$	$\frac{1}{5}$	⋯
$c_0(2)$	$\frac{1}{5}$	$\frac{1}{10}$	$\frac{1}{5}$	$\frac{1}{5}$	⋯	$\frac{1}{10}$	$\frac{1}{5}$	$\frac{1}{5}$	$\frac{1}{5}$	⋯
$c_0(3)$	$\frac{1}{5}$	$\frac{1}{10}$	$\frac{1}{5}$	$\frac{1}{5}$	⋯	$\frac{1}{10}$	$\frac{1}{5}$	$\frac{1}{5}$	$\frac{1}{5}$	⋯
$c_1(1)$	$\frac{2}{3}$	$\frac{1}{10}$	$\frac{1}{6}$	$\frac{1}{6}$	⋯	$\frac{1}{10}$	$\frac{2}{3}$	$\frac{1}{6}$	$\frac{1}{6}$	⋯
$c_1(2)$	$\frac{1}{6}$	$\frac{1}{10}$	$\frac{1}{6}$	$\frac{1}{6}$	⋯	$\frac{1}{10}$	$\frac{1}{6}$	$\frac{1}{6}$	$\frac{1}{6}$	⋯
$c_1(3)$	$\frac{2}{3}$	$\frac{1}{10}$	$\frac{1}{6}$	$\frac{1}{6}$	⋯	$\frac{1}{10}$	$\frac{2}{3}$	$\frac{1}{6}$	$\frac{1}{6}$	⋯
c_2	$\frac{8}{9}$	$\frac{1}{9}$	$\frac{1}{9}$	$\frac{1}{9}$	⋯	$\frac{1}{9}$	$\frac{8}{9}$	$\frac{1}{9}$	$\frac{1}{9}$	⋯

Initial values for the endogenous parameters:

$a_1 = a_2 = 0.9$, $a_3 = 0.1$, $t_{12} = t_{13}0.1$, $t_{23} = 0.9$.

3 Empirical Strategies

In this section, we develop two discrete-time Markov models for empirical analysis. Both combine the *spatial-lag* model from spatial econometrics and the *p-star* model from network analysis. We also compare these with the *actor-oriented* continuous-time coevolution models developed by Snijders and colleagues (Steglich et al. 2006; Snijders et al. 2007; Snijders 2005).

3.1 Discrete-Time Markov Models

Our first discrete-time Markov model is one with behavior (not behavior-type) contagion and observed ties (not latent-strengths of ties). We define the behavior-shaping probability in a simple *spatial-lag* logit model, one with only time-lagged (and not simultaneous) spatial-lag, and define the tie-formation probability in the simplest *p-star* model, one with independent dyads (which likewise reduces the tie-formation model to a set of conditionally independent logits);[13]

$$\begin{cases} Pr(s_{i,t} = 1|\mathbf{s}_{t-1}, \mathbf{d}_{t-1}) = logit(\beta_0 + \beta_1 s_{i,t-1} + \beta_2 \mathbf{d}_{i,t-1}\mathbf{s}_{t-1}) \\ Pr(d_{ij,t} = 1|\mathbf{s}_{t-1}, \mathbf{d}_{t-1}) = logit(\gamma_0 + \gamma_1 d_{ij,t-1} + \gamma_2 \cdot I(s_{i,t-1} = s_{j,t-1})), \end{cases} \tag{5}$$

where $\mathbf{d}_{i,t-1}$ is a row vector of size N that contains the set of $(N-1)$ dichotomous tie-formation indicators between i and all the other actors 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 the behaviors in the given dyad were the same in the previous period. The term captures homophily. In this model, contagion (network effects) and the homophiletic selection (network formation) both operate through behaviors rather than through behavior type.

Our second empirical model is instead a latent-type and strength-of-tie model, which connects much more closely to the theoretical model presented above. The behavior-switching and tie-formation/dissolution rules in this model take the form:

$$\begin{cases} Pr(s_{i,t} = 1|\boldsymbol{\sigma}_{t-1}, \boldsymbol{\delta}_{t-1}) = logit(\beta_0 + \beta_1 s_{i,t-1} + \beta_2 \boldsymbol{\delta}_{i,t-1}\boldsymbol{\sigma}_{t-1}) \\ Pr(d_{ij,t} = 1|\boldsymbol{\sigma}_{t-1}, \boldsymbol{\delta}_{t-1}) = logit(\gamma_0 + \gamma_1 d_{ij,t-1} + \gamma_2 (\sigma_{i,t-1} - \sigma_{j,t-1})^2), \end{cases} \tag{6}$$

where $\boldsymbol{\delta}_{i,t-1}$ is a row vector of size N that contains probabilities that i forms ties with, or the proportionate strengths of the ties between i and, each of the others, j, at the end of period $t-1$ (appropriately zeroed for i's self-reflexive dyad).

In this model, connections are by (observed) continuous strength of tie, $\boldsymbol{\delta}$, not dichotomous tie or indicator of tie, $\mathbf{d}$, and contagion and selection operate through (observed) behavior type, $\boldsymbol{\sigma}$, not dichotomous behavior or indicator of behavior, $\mathbf{s}$. One appealing way to conceive this substantively is that actors' types are influenced by other actors' underlying types and not by ephemeral short-run behavioral manifestations. Substantively, this reflects a proposition that, for example, if my friend who rarely votes happens to vote in one election by chance, that behavior is unlikely to influence the rate at which I turn out to vote. Likewise, homophily/heterophily is driven by type rather than

[13] Important extensions for next steps in this project of course include enriching these two models closer to the start of their arts: simultaneous spatial-dependence in dichotomous outcomes and p-star models beyond simple independence.

current-behavioral manifestations, which could be attributable to chance. For instance, a lifelong liberal senator is much more likely to cosponsor legislation with another committed liberal senator than with a conservative senator who just happened to vote liberal on the previously considered piece of legislation.

Because these types and strength of ties are often unobserved, as in our example application to come, for instance, we need a way to identify and estimate them. We assume that types are always in equilibrium given behavior-switching probabilities, and that tie strength is always in equilibrium given the Markov chain governing tie formation and dissolution from period to period. More formally, we assume

$$\begin{pmatrix} \sigma_{i,t} \\ 1-\sigma_{i,t} \end{pmatrix}' = \begin{pmatrix} \sigma_{1,t} \\ 1-\sigma_{1,t} \end{pmatrix}' \begin{pmatrix} z_t^{11} & 1-z_t^{11} \\ 1-z_t^{00} & z_t^{00} \end{pmatrix} \tag{7}$$

and

$$\begin{pmatrix} \delta_{ij,t} \\ 1-\delta_{ij,t} \end{pmatrix}' = \begin{pmatrix} \delta_{1,t} \\ 1-\delta_{1,t} \end{pmatrix}' \begin{pmatrix} w_t^{11} & 1-w_t^{11} \\ 1-w_t^{00} & w_t^{00} \end{pmatrix} \tag{8}$$

With these assumptions, starting values for $\boldsymbol{\sigma}_{t0}$ and $\boldsymbol{\delta}_{t0}$, and parameter values for $\boldsymbol{\beta}$ and $\boldsymbol{\gamma}$, we can calculate the probability of observing 1's and 0's at time $t = 1$. We can also update each actor's type and the strength of ties across dyads using the implied transition probability matrices. Of course, these difference equations have multiple solutions. We select the solution that emerges from the Markov chain beginning at the previous period's types and tie strengths. With estimated values for $\boldsymbol{\sigma}$ and $\boldsymbol{\delta}$ at time $t = 1$, we can calculate the probability of observing 1's and 0's at time $t = 2$. By repeating this process, we can calculate the joint likelihood for a given sample.

3.2 Comparison with SIENA: Continuous-Time Markov Models

In the network-analytic tradition, Snijders and colleagues (Snijders 1997, 2001; Steglich et al. 2006; Snijders et al. 2007; Snijders 2005) have advanced perhaps furthest in empirical modeling of dynamic, endogenous contagion and selection.[14] They model the coevolution of networks and behavior thus. N actors are connected by an observed, binary, endogenous, and time-variant matrix of ties, $\mathbf{x}$, with elements $\mathbf{x}_{ij,t}$. A vector of N observed, binary behaviors, $\mathbf{z}$, at time t has elements $\mathbf{z}_{i,t}$. 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 connections, switching at most 1 tie on or off, at continuous-time fixed-rate, λ_i^{net}, according to an exponential model. At present, λ_i^{net} is assumed constant for all i, j, t, though in principle one could parameterize it. Likewise, opportunities to increment, decrement, or leave the behavior arise at rate λ_i^{beh}.[15] When an opportunity to change

[14] Leenders (Leenders 1995, 1997) presages. Hoff and colleagues (Hoff et al. 2002; Hoff and Ward 2004; Hoff and Westveld 2007) offer an alternative, Bayesian latent-space approach. Our discussion follows Snijders (Snijders 1997) and Snijders et al. (Steglich et al. 2006; Snijders et al. 2007) most specifically.

[15] Since observation occurs at discrete intervals, the freedom to vary these continuous-time rates render effectively inconsequential the assumptions of one actor making one unit-valued change in his/her network ties or behavior at a time. As greater frequency and/or magnitude of changes are observed, estimates of these occurrence rates at this unobserved instantaneous level simply rise to compensate. This does not, however, relax the strong assumption of conditional independence of these actors' choices (which we make as well).

network ties arrives for some i, i may choose to alter the status of any one of its N-1 ties to *on* or to *off* or to leave all ties unchanged. i makes these choices by comparing the values of some objective function of this form:

$$f_i^{net}(\mathbf{x}, \mathbf{x}', \mathbf{z}) + \varepsilon_i^{net}(\mathbf{x}, \mathbf{x}', \mathbf{z}) = \sum\nolimits_h \left\{ \beta_h^{net} \times \mathbf{s}_h^{net}(\mathbf{i}, \mathbf{x}, \mathbf{x}', \mathbf{z}) \right\} + \varepsilon_i^{net}(\mathbf{x}, \mathbf{x}', \mathbf{z}) \tag{9}$$

where $\mathbf{x}'$ is an alternative network under consideration, which can differ from the existing network, $\mathbf{x}$, only by changing at most one element of (only) row i. $f_i^{net}(\cdot)$ is called the network evaluation function. $s_h^{net}(\cdot)$ is some statistic, i.e., some function of the data, $\mathbf{x}, \mathbf{x}', \mathbf{z}$, that reflects the actor's objectives (ideally, substantively-theoretically derived) regarding the network, $\mathbf{x}$, and behaviors, $\mathbf{z}$. The $\boldsymbol{\beta}_h^{net}$ to be estimated are the relative weights of these objectives. Assuming the $\boldsymbol{\varepsilon}_i^{net}$ extreme-value distributed, independently across actors and over time, yields the multinomial-logit model of categorical choice. Similarly, when an opportunity to change behavior arrives, actor i compares the value of an analogous objective function under each of three possible actions: increment or decrement by one or leave unchanged. Formally, i compares $\mathbf{z}$ to $\mathbf{z}'$ given $\mathbf{x}$ and $\mathbf{z}_{j \neq i}$. Again, the behavior evaluation function, $f_i^{beh}(\cdot)$, is the summed product of weights and statistics, $\boldsymbol{\beta}_h^{beh}$ and $s_h^{beh}(\cdot)$ respectively, and again assuming *i.i.d.* extreme-value stochastic components ($\boldsymbol{\varepsilon}_i^{beh}$), the multinomial logit emerges once more.

4 Illustration: Military Alliances and Conflict Behavior

We illustrate these methods with an empirical analysis of the alliance-formation and conflict behavior of great powers during the first half of the twentieth century (Levy 1981). We suspect that alliance ties and conflict behavior coevolve. States self-select into alliances and these decisions are plausibly driven by preferences homophiletic or heterophiletic in behavior (type). More aggressive (pacific) states may seek likewise aggressive or pacific allies. At the same time, conflict behavior is contagious through alliances. Indeed, that states would be drawn into the conflicts of their allies is usually a key part, if not the core working principle, of alliances.[16] We focus on the first half of twentieth century because it was a period with considerable variation in conflict behavior (hardly unique to that period) and of multipolarity during which military alliances were in flux (rarer in other periods).

We present preliminary estimates of our models in Table 2. Model 1 (columns 1a and 1b) is our model with contagion of dichotomous behaviors with connection and selection by observed dichotomous ties. Model 2 is—as yet, unfortunately only half of—our model with contagion of latent type and connection and selection by observed strength-of-tie. (These particular results were estimated using the assumed-exogenous observed dichotomous ties.)[17] Model 3 (columns 3a and 3b) is the SIENA continuous-time Markov model of coevolution. To estimate this model we used 'snapshots' of the great powers' alliance networks and conflict behavior taken at five-year increments (i.e., 1900, 1905,...1950). 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 $s_i^{net} = \sum_j x_{ij}(\mathrm{sim}_{ij}^z - \widehat{\mathrm{sim}^z})$, where the similarity scores are $\mathrm{sim}_{ij}^z = \frac{\Delta - |z_i - z_j|}{\Delta}$, Δ being the maximum sample difference, and $\widehat{\mathrm{sim}^z}$ is the mean of all similarity scores.The latter behavior-

[16] For a similar argument, see (Kimball 2006).

[17] Obviously, closing this model is a highest priority for the next draft.

statistic is defined as $s_i^{beh} = \frac{\sum_j x_{ij}(\text{sim}_{ij}^z - \widehat{\text{sim}^z})}{\sum_j x_{ij}}$. See Snijders (CITES) for many alternatives and much further discussion.

Overall, we find evidence (1) of heterophily—pacific powers are more likely to ally with aggressive powers—and (2) that conflict behavior is (positively) contagious through alliances.

Table 2: Estimation Results: Military-Alliance Ties and Binary Conflict Behavior

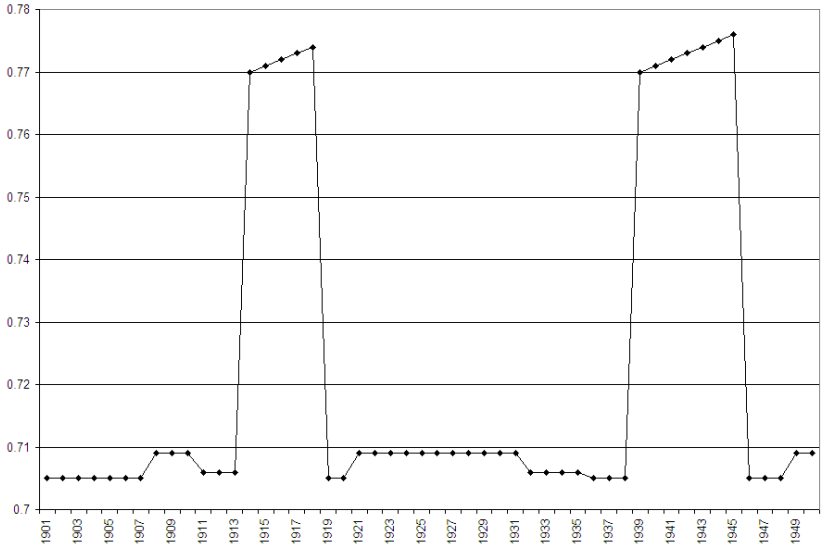
(Markov Model>>)	**Discrete-Time (MATLAB)** Ours			**Cont.-Time (SIENA)** Snijders et al.	
	(1A) Alliance Networks	(1B) MIDs Behavior	(2) MIDs Behavior	(3A) Alliance Networks	(3B) MIDs Behavior
Temp lag	4.93*** (0.27)	1.45*** (0.27)	1.32*** (0.02)	–	–
Previous MIDs similarity	**-0.51*** (0.27)	–	–	**-3.52***** (0.60)	–
Previous alliance tie	–	**0.85***** (0.31)	0.10*** (0.03)	–	**1.06**** (0.53)
Loglikelihood	-237.4	-179.5	-168.4		

From model 2, we have latent type estimates for each of the actors from 1901-1950. Estimates for the U.S. are provided in Figure 2. Unfortunately, as previously acknowledged, this is not a true coevolution model since we assumed exogenous ties (alliances). However, if it were, we would expect complex history dependence in the evolution of this variable. To understand why, remember that in 1910, for example, we have a set of estimated behavior-switching probabilities. These transition probabilities partly determine the latent type for America in 1910. We say partly because there are multiple latent types that are consistent with these switching probabilities. We assume the *correct* type is the one that emerges from a Markov chain beginning at America's latent type in 1909. Therefore, America's type in 1910 is a steady-state solution, one of many to the difference equation implied by the estimated transition probabilities for 1910, that is determined by America's type in 1909, which is itself one of many steady-state solutions, partly determined by America's type in 1908, to the difference equation implied by the estimated behavior-switching probabilities for 1909, etc. etc.

5 Conclusion

Theoretically, this paper attempts to build a Markov type-interaction model in which the behaviors of actors and the networks that connect them coevolve over time. One interesting implication of the model is that it produces history-dependent behavior, possibly including path dependent behavior.

Figure 2: Estimated Behavioral Type for the U.S. (1901–1950)



It seems likely that there are many areas of inquiry in the social sciences where network-behavior coevolution is important. To evaluate this possibility empirically, we combined a simple spatial-lag logit model of contagious behavior with a simple p-star logit model of network formation. We used this statistical framework to analyze the patterns of alliance formation and conflict behavior among the great powers during the first half of the twentieth century.

There is a lot of work left to do. In the short run, we need to refine the theory, exploring important alternatives such as models in which actors observe dichotomous outcomes and adjust their continuous latent traits. There are multiple other options regarding what diffuses and what forms the basis for selection as well. We need to tighten the connection between the theoretical and empirical models. In the medium run, we hope to theorize more fully the relationship between multiple equilibria and history dependence, and characterize the mapping from parameter and starting values to the presence and forms of history dependence. In the longer term, we hope to develop statistical tests for various types for history dependence, including path dependence, and explore the small-sample properties of the estimators, including comparisons to Snijders' SIENA framework from longitudinal networks analysis.

Appendix: Summary of Notations

- Actors: $i \in \{1, \cdots, N\}$.
- Discrete time periods: $t \in \{1, 2, \cdots\}$.
- Behavior: $s_{it} \in \{0, 1\}$. A dichotomous behavior (behavioral action) that actor i takes in period t. Both actors in the model and analysts (or econometricians) outside the model can observe.
- Behavioral type: $\sigma_{it} \in [0, 1]$. This represents the probability that actor i chooses behavior 1 in period t. We assume that, in this model, actors can observe (know) others' behavioral types.
- Network tie: $d_{ij,t} \in \{0, 1\}$. An undirected indicator of whether the two actors i and j in a certain dyad are connected. The indicator $d_{ij,t} = 1$ if they are connected, $d_{ij,t} = 0$ if not.
- Connectivity matrix (discrete): $\mathbf{D}_t$ denotes an $N \times N$ symmetric matrix, in which entry of cell (i, j) is $d_{ij,t}$.
- Tie-formation probability: $\delta_{ij,t} \in [0, 1]$, $\forall i, j$ and $i \neq j$. This denotes the probability that a tie forms between the two actors i and j in a given dyad in period t. This probability can also be interpreted as the strength of the tie between i and j.
- Connectivity matrix (continuous): $\mathbf{\Delta}_t$ denotes an $N \times N$ symmetric matrix, in which entry of cell (i, j) is $\delta_{ij,t}$.
- State of the system: $(\boldsymbol{\sigma}_t, \boldsymbol{\delta}_t) = (\sigma_{1t}, \cdots, \sigma_{Nt};\ \delta_{12,t}, \cdots, \delta_{N-1,N,t})$. We define the state of the system at the end of period t as a vector of every actor's behavioral type and every dyad's tie-formation probability.
- Exogenous components of transition probabilities: c_0, c_1, c_2. The probability c_0 captures the odds of actors' choosing behavior 0 regardless of the state of the system. Similarly, c_1 represents the probability that actors choose 1 regardless of the state. The parameter c_2 is the probability that a given dyad forms a tie regardless of the state.

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