The coevolution of networks and health

Introduction to the Special Issue of Network Science

DAVID R. SCHAEFER
Department of Sociology, University of California Irvine, Irvine, California, USA
(e-mail: drschaef@uci.edu)

JIMI ADAMS
Department of Health and Behavioral Sciences, University of Colorado Denver, Denver, Colorado, USA
(e-mail: jimi.adams@ucdenver.edu)

Historically, health has played an important role in network research, and vice versa (Valente, 2010). This intersection has contributed to how we understand human health as well as the development of network concepts, theory, and methods. Throughout, dynamics have featured prominently. Even when limited to static methods, the emphasis in each of these fields on providing causal explanations has led researchers to draw upon theories that are dynamic, often explicitly. Here, we elaborate a variety of ways to conceptualize the relationship between health and network dynamics, show how these possibilities are reflected in the existing literature, highlight how the articles within this special issue expand that understanding, and finally, identify paths for future research to push this intersection forward.

We organize our discussion of the relationship between networks and health along the separate dimensions of causal ordering and levels of analysis (see Table 1). Many social and behavioral sciences take (for granted) the individual as the focal unit of analysis. This orientation is concerned with providing explanations for individual outcomes, particularly those that can be linked to specific behaviors. In turn, those linkages provide the basis for behavioral interventions that can improve individual outcomes, e.g. better health. This orientation provides a clear framework for incorporating a network perspective into health research: how do networks shape or influence one’s health outcomes (Table 1, Cell 1A). This framework prominently underpins some of the earliest adoptions of network ideas into health research, exemplified by Coleman et al. (1957) demonstrating the diffusion of tetracycline prescription practices among physicians in the Midwest.

Although influence is the predominate focus of health studies, inferences of network influence are frequently overstated (Shalizi & Thomas, 2011). As with many fields that rely principally on cross-sectional data, any claims of causal effects must be carefully tempered (Winship & Morgan, 1999). While assortativity, or similarity between connected nodes, is consistent with influence, it does not necessarily imply contagion as a causal mechanism (Kandel, 1978); such similarity may also be driven by homophilous selection. Network selection, particularly in regards to homophily, has a longstanding theoretical grounding (for a review, see McPherson et al. (2001) despite not receiving the same empirical attention across that period. Questions about the effects of health on networks (Cell 1B) provide a cornerstone of recent efforts to identify the nature of network-based selection effects (Schaefer et al., 2011).
Within individual-level networks research, a burgeoning body of literature aims to disentangle selection from influence effects (Steglich et al., 2010) and as such focuses on both cells 1A and 1B.

Although research on networks and health is focused disproportionately on individual-level outcomes, networks can also inform our understanding of population level phenomenon. Shifting to row 2, population health research focuses not on explaining individuals’ outcomes, nor even simply aggregating up from studies that do. Instead, this orientation (Cell 2A) focuses on an entire population and investigates the network-based processes that account for population-level outcomes, for instance, health disparities between groups within the population (Keyes & Galea, 2016; Bachrach & Daley, 2017). An exemplar contribution to this literature is research on the role of network concurrency in understanding the epidemiology of HIV. For example, Morris et al. (2009) find that small differences in the rates of observed concurrency (2% greater among blacks than whites) is sufficient to produce substantial differences (50% disparity) in epidemic potential between these groups.

This perspective embraces the connection of network research to its complexity counterparts and focuses on explaining nonlinear dynamics and emergent features of population processes that are not reducible to aggregations of individual level attributes or mechanisms. In fact, recent work has shown the limitations that arise from not treating these levels as conceptually distinguishable—even when employing network perspectives. For example, population-level structural features can operate in conjunction with, independently from, or even in opposition to mechanisms operating at the individual level (e.g. Adams & Schaefer, 2016). When focusing on network influence at this level of analysis, questions address what features of observed network structures account for population-level health outcomes (e.g. disease prevalence). Conversely, researchers can also examine how population level attributes lead to different patterns of global network structure (Cell 2B). Health-related empirical examples of this sort are rare; however, Moody (2001) demonstrates how population level race/ethnic heterogeneity contributes to patterns of assortativity on race/ethnicity.

In general, while we can conceptually distinguish processes of network influence on health from those whereby health affects networks, empirical reality is likely to exhibit these processes operating in both directions simultaneously, giving the possibility of bi-directional feedback effects. In what follows, we elaborate how both the causal direction and levels of analysis described above are reflected in the literature, including the articles of this special issue. We choose to organize the

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Table 1. Prototypical types of network and health studies.

<table>
<thead>
<tr>
<th>Causal direction</th>
<th>Networks → Health</th>
<th>Health → Networks</th>
</tr>
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<tbody>
<tr>
<td>Level of Analysis</td>
<td>Individual 1A: Influence</td>
<td>1B: Selection</td>
</tr>
<tr>
<td></td>
<td>Population 2A: Diffusion</td>
<td>2B: Network topology</td>
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discussion primarily along the causal direction dimension since this is the question most central to network literature.

1 Networks → health

To begin, how do networks influence health? One approach to addressing this question is to focus on purely structural aspects of networks and ask how they shape health outcomes. Studies of this sort ask how network features ranging from the local level (e.g. degree or reciprocity) to those informed by the global network structure (e.g. centrality or clustering) contribute to individual health outcomes. In this issue, Copeland, Bartlett, and Fisher (CBF) take this approach in asking how several theoretically-informed operationalization’s of isolation affect one’s smoking behavior. They observe that there are different forms of isolation, each with its own causal antecedents that can affect smoking behavior in distinct ways. In this issue, Elmer, Boda, and Stadtfeld (EBS) ask a similar question about how embeddedness in networks of strong or weak ties affects emotional well-being. Their findings push us to think more deeply about how the processes relating networks to health may work differently depending upon the type of relation under study. The Lienert et al. paper also offers a new twist on this question. They ask whether cancer patients who share time together in a cancer treatment ward experience similar survival outcomes. Their findings are suggestive and call for more research on the social processes transpiring within shared treatment environments that may operate above and beyond the effects of the individual-level biomedical treatment alone.

Beyond the individual level, studies have demonstrated the utility of network structure—and the dynamics therein (e.g. Bansal et al., 2010)—for explaining population-level health outcomes. For example, Moody et al. (2017) show how the overall volume and shape of a degree distribution combine to determine population-level epidemic potential. Other studies consider how features such as cohesion, clustering, and density alter epidemic trajectories (Salathe & Jones, 2010). However, given the scope of network data needed to identify these structural features, much of this work to date has relied on data that are static in nature. In this issue, Armbruster, Wang, and Morris overcome this limitation by developing a new method for identifying “forward-reachable sets” as a means to operationalize how network structure and dynamics combine to alter diffusion potential.

Another way to examine how networks shape health is to consider both network structural features and individual properties. This approach is represented most commonly by studies of peer influence, which investigate how alters’ health properties and/or behaviors affect ego’s health outcomes. EBS provides an example of this form of inquiry, by showing how the emotional well-being of one’s friends affects one’s own well-being. Though they find limited evidence of such contagion, their distinction between the types of ties where influence can occur is provocative. In a similar vein, Perry, Pullen, and Pescosolido (PPP) investigate the moderating effect of networks on perceptions of health care. In particular, they find that individuals embedded in networks with positive beliefs about the medical profession experience more successful treatment for mental health. Of note, both EBS and PPP fit the call by Veenstra and colleagues (2013) for more research on how relational attributes may moderate network effects.
Networks and health are also associated in the opposite causal direction. Calls for attention to the effects of health on networks have been made periodically (House et al., 1988), however empirical investigations have been less prominent than those focused on network influence. This question is critical as selecting one’s network is in effect selecting those individuals who will later influence oneself or, in the case of diffusion, serve as a transmission vector.

Health can affect the structure of a network in terms of both the presence and configuration of relations. Many health conditions limit one’s capacity for social interactions, by imposing cognitive or physical restrictions, thereby reducing the size of one’s network (Haas et al., 2010). An important distinction here is whether the effect of health on networks is direct or indirect. For instance, both CBF and EBS distinguish a direct effect—whether smoking and emotional well-being, respectively, affect the number of friends one chooses—from an indirect effect, whereby health affects one’s attractiveness as a friend to others (e.g. avoiding or being drawn to smokers as friends). Distinguishing these effects has implications for the design of interventions to alter networks (Aral et al., 2013).

Health can also affect the content of one’s network, such as when one’s own health determines the kinds of people in one’s network. Homophilic preference is a prominent example of this process, where the joint status of ego’s and alter’s health determines selection tendencies, with ties between similar individuals (e.g. smokers) more likely to exist. EBS extends the study of homophilous selection to the case of emotional well-being, finding notable differences in the magnitude of this process for strong versus weak friendships.

The aforementioned studies, like most prior research examining how health shapes networks, focuses at the individual level (Cell 1B). Thus, Cell 2B is comparatively less populated in empirical studies compared to the other cells in Table 1. However, shifting the unit of analysis from individual health to healthcare organizations reveals some examples. Healthcare organizations are one field that have demonstrated the benefits of assessing population-level network structure as shaped by health conditions. For example, those hospitals with complementary types of expertise are likely to form collaborative relationships, which in turn should promote better coordination of care for the population of patients served across those organizations (Lomi & Pallotti, 2012). Moreover, the requirements of practitioner specialization that govern how patient transfers occur among hospitals, leads to networks that are less hierarchical and centralized than the language often used to describe hospital systems would suggest (Iwashyna et al., 2009).

The relationship between networks and health can involve processes operating in both directions simultaneously. From a causal inference standpoint, considering bi-directional pathways is preferable, since the same health-network association may arise through multiple processes, including those representing different causal orders. In this issue, Schneider et al. take coevolution as a sequential set of related questions. They begin by examining the stability of confidant and sexual relationships.
exploring several predictors of this stability, they turn to investigating which health effects are explained by network stability, finding effects on both attitudinal, and behavioral risk factors.

As with the examples above, structure may combine with attributes to generate bi-directional causal effects. For instance, a pattern of assortativity on a health factor can arise through homophilous selection (health → network) and through peer influence (network → health). Though assortativity is the more commonly studied network pattern given interests in peer influence, the same principle holds true for other network–behavior associations. Take popularity correlated with an attribute—this could arise because certain behaviors enhance one’s attractiveness, such as smokers being more attractive friends, or because having more friends enhances the likelihood of the behavior (Schaefer et al., 2012). In such cases, inferring that an observed pattern is the result of an effect in one direction requires controlling for the corresponding effect in the opposite causal direction. In addition, this approach may be preferred on theoretical grounds, such as when one hypothesizes the presence of feedback mechanisms (Ip et al., 2013).

Methodologically, evaluating coevolution requires a more sophisticated approach than conventional linear models, with possibilities including multivariate models or, increasingly, simulations. CBF uses an autoregressive latent trajectory model for their study of the coevolution of smoking and isolation. Such a method is suitable to the extent that network dependencies are limited (which they suggest is the case for isolation). When network interdependencies are more extensive, then methods such as the stochastic actor-oriented model (SAOM; Snijders et al., 2010) that explicitly model network structures and behavior change mechanisms are more applicable. As one example, EBS take advantage of the SAOM’s ability to model both network and health outcomes, while allowing each outcome to be endogenously affected by the other.

4 Future directions

The papers in this special issue address several aspects of health-network coevolutionary dynamics. Each reflects the more general tendency of the field to focus on how network structure and process affect individual and population health outcomes. Additionally, three of the papers combine this form of question with some attention to the reverse-causal process—how health shapes network structure. We see several additional directions in which future research may fruitfully continue to inform our understanding of health-network dynamics.

As the field continues to embrace a focus on the bi-directional health-network association, we encourage researchers to carefully reconsider the nature of their causal relationship. One step is to move beyond simply thinking of each causal direction as a separate process, and instead consider them as complementary elements of a complex system characterized by feedback effects (e.g. Martin et al., 2013). DiMaggio and Garip (2011) provide an example of such a conceptualization in their network-based model of cumulative advantage. They argue that when individuals selectively affiliate based on shared attributes that are correlated with an outcome; those outcomes are reinforced, which can increase inequality at the population level. For example, if individuals affiliate based on similarities in SES that are correlated with smoking, then as those networks facilitate influence on smoking cessation, smoking at the population
level will become increasingly polarized. Such a perspective allows for a broader focus that encompasses both individual and population level outcomes, which are largely separated in the current literature (as depicted in Table 1). Evaluating such possibilities will require the use of inherently dynamic models, such as agent-based models (DiMaggio & Garip, 2011; Zhang et al., 2015) or similar statistical models (Steglich et al., 2010). We also encourage future research to carefully consider the operationalization of attributes included in these causal models. Research generally focuses on a single behavior at a time, examining its coevolution with a network. However, there are growing reasons to believe that network selection and influence operate simultaneously across multiple behavioral dimensions, and that such behaviors can be interdependent (Rambaran et al., 2017). Indeed it may be that examining behaviors in isolation is a mis-specification of how networks operate and that a more holistic view is needed, such as examining the effects on clusters of behaviors that combine into “health lifestyles” (Mollborn et al., 2014).

In calling for more research on health and network coevolution, we also encourage reconsidering the conceptual nature of selection. As noted above, beyond simply being an alternative to influence in explaining network assortativity, selection is also part of a feedback process, setting the stage for outcomes related to health. For instance, when people are socially marginalized or isolated based on a health condition, there is little room for contagion to operate (CBF). Alternatively, individuals may be embedded in networks that provide opportunities for modeling health-related behaviors, and where local norms can promote healthy and/or unhealthy behavior. As the role of networks in health becomes clearer, the a priori question of how those networks come about becomes increasingly important to answer. There are many avenues to pursue in this regard. Although homophilous selection on health factors is the de facto meaning of selection in many studies, attribute-based selection can take other forms. Health conditions can limit one’s sociality (CBF; Schaefer et al., 2011) or popularity (Crosnoe et al. 2008). Or, multiple attributes may be involved. For instance, Schaefer (2017) tests whether individuals with particular values on one attribute (i.e. low self-control) are more likely to select friends with particular values on a second attribute (i.e. substance use involvement), thereby explaining how adolescents come to be at risk for negative peer influence. Indeed attribute-based selection is likely to be more complex than simply homophily on one or more attributes.

A common critique of network studies is their lack of context, which can be particularly problematic if “similar configurations may produce different outcomes” when examined across different settings (Borgatti et al., 2014). Recent research has begun to take this criticism seriously, a shift we encourage future research to build upon. For example, Adams and Schaefer (2016) found that different initial population-level conditions can cause the same effects (e.g. of peer influence) to generate divergent population-level changes in smoking behavior. Other studies have shown that different contextual norms can lead to variable strength of peer influence and selection effects at the level of peer groups (Delay et al., 2013) or the classroom (Rambaran et al., 2013). As researchers continue to gather network data from a growing number of contexts and population subsamples simultaneously, the field should increasingly scrutinize how these and other context-dependent explanations account for the relationships between networks and health.
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The studies constituting this special issue represent an advance in our understanding of the coevolution of health and networks. We call for pushing these and other ideas further in three broad directions. First is continuing to strengthen our conceptualization of health-network dynamics, both by theorizing the 4 cells in Table 1 as part of one overarching system and by refining our definitions of the “behaviors” on which network selection and influence operate. Second is to broaden our awareness of the forms that selection can take. No doubt homophily is important in most cases, but it is likely to be only a partial explanation. Third is to consider how health–behavior dynamics may vary across contexts. This is especially key to the extent that research on networks and health will inform health promotion efforts in the natural world (Valente, 2012).

References


