Towards a Causal Theory and Test of Network Effects: Structural Holes, Alliance-Network Externalities, and Organizational Innovation

Exequiel Hernandez
The Wharton School
University of Pennsylvania
exequiel@wharton.upenn.edu

Jason Lee
The Wharton School
University of Pennsylvania
lkwjason@wharton.upenn.edu

J. Myles Shaver
Carlson School of Management
University of Minnesota
mshaver@umn.edu

Authors contributed equally and are listed alphabetically

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ABSTRACT: Assessing whether network position causes organizational outcomes is difficult because networks are usually the result of firm choices (i.e., firm agency). Rather than adopting a statistical approach to address this challenge, we offer a theory-driven solution. Using structural causal modeling, we integrate canonical network theory with the concept of alliance-network externalities. This distinguishes self-driven changes in a focal firm's network position from other-driven changes—the latter of which suppress the agency of the focal firm. Therefore, under certain assumptions that we can evaluate, assessing how other-driven changes affect organizational outcomes can be interpreted as a causal test of network position. Examining the biotechnology industry alliance network (1995-2012), we find that structural holes increase firm innovation only under conditions of self-driven network change, but *not* under conditions of other-driven network change. We thus do not find support for a causal effect of structural holes *per se* on innovation. One interpretation is that the effect of network position is spurious. Another is that canonical theory requires updating to account for agency as a factor that activates the benefits of structural position. Our theory and results have profound implications for how scholars theorize and test network effects.

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A preponderance of empirical evidence demonstrates that certain positions within an alliance network (e.g., structural holes, centrality) are associated with desirable organizational outcomes (e.g., Ahuja, 2000a; Phelps, Heidl, and Wadhwa, 2012; Balachandran and Hernandez, 2018). Many of these studies conclude that managing a firm's network to realize such positions can be advantageous. Nevertheless, several scholars also rightfully acknowledge that other factors allowing a firm to obtain advantageous network positions—such as its objectives, actions, and capabilities—are hard to separate from the positive outcomes that might stem from those positions (Manski, 2000; Rider, 2009, 2012; Baum, Cowan, and Jonard, 2010). Do firms benefit because of their network position, or do they occupy that position for reasons that allow them to be obtain the benefit in the first place? Answering this question is essential before one can effectively draw conclusions from these studies, and not just from an empirical or practical perspective. We emphasize that the answer is central to identifying the theoretical mechanisms that explain why network positions affect organizational outcomes.

Employing structural causal modeling (e.g. Pearl 2010; Pearl and Mackenzie, 2018), we offer a novel theoretical approach to address this issue. We apply the approach to re-assess the impact of structural holes on innovation—one of the most studied relationships in interorganizational networks (Phelps, Heidl, and Wadhwa, 2012 offer a review). We make this reassessment by integrating two streams of research and mapping the underlying causal structure of their theoretical arguments. These two streams are (a) the aforementioned theory of structural holes and innovation and (b) recent theoretical ideas on alliance-network externalities. The former provides the canonical argument linking structural holes to innovation outcomes *given* that the focal firm is already in a position spanning structural holes. However, it does not offer an account of *how* the firm achieved that structural position in the first place. This is where the latter stream is useful. It offers a conceptual foundation, rarely highlighted in the literature, to explain whether firms' observed network positions are the result of their own actions or the result of others' actions. Precisely integrating and mapping the underlying causal arguments of

these two theories reveals a novel theory-driven approach—in contrast to a statistical approach—to assess *when* the effect of network position on innovation can be considered causal.

We adopt the insight from recent advances in the study of alliance network dynamics showing that interorganizational networks can change through various corporate actions. These include alliance formation and dissolution, acquisitions, and divestitures (Hernandez and Menon, 2018, 2021; Hernandez and Shaver, 2019). By distinguishing between self-driven and other-driven actions, this work also introduces the notion of alliance-network externalities: how the actions of *other* firms modify the structural position of a focal firm through changes in the focal firm's alliance network that it did not initiate. For instance, a third party's acquisition might rewire some of the focal firm's ties or eliminate a previous partner of the focal firm in ways it did not intend. In contrast, the actions of the focal firm that modify its alliance network (e.g., its own acquisitions or alliance dissolutions) are difficult to separate from the intentions behind its corporate transactions. The key conceptual difference is that the locus of agency differs across the two determinants of network position.[1]

Causal mapping allows us to identify and develop a powerful theoretical implication of alliance-network externalities. Whereas a network position obtained through a firm's own corporate actions (self-driven) is difficult to separate from its own agency, the same position obtained through others' corporate actions (other-driven) are not subject to this concern—under certain assumptions that we carefully consider. This insight provides a path to hypothesize that, under conditions of other-driven network change, structural holes will have a positive causal

[1] Our main idea does not rely on firms acting as hyper-rational structural architects of their own or others' networks. Nevertheless, we do assume that a focal firm's own actions and their effects on its network are congruent with some overarching corporate intention that influences its alliance portfolio, whereas the changes in other firms' network positions do not factor into the focal firm's intentions (see Hernandez and Menon, 2021). We justify this assumption later in the paper.

effect on the focal firm's innovation. Such a statement cannot be made about structural holes obtained under conditions involving self-driven network change.

This theoretical foundation provides guidance for a novel research design to test the relationship between network position and organizational outcomes. We implement the test through the following steps. First, we obtain comprehensive data on the alliance network for a specific industry. Second, we account for how each firm's own corporate actions (alliance formation and dissolution, acquisitions, divestitures, entry, and exit), plus the same actions by other firms, modify the structure of the industry alliance network in each period (year). Third, we categorize changes in a firm's ego network as driven by their own actions, others' actions, or a mix of the two. Fourth, we regress measures of firm-level innovation on these sources of network change. This allows us to observe whether the effect of network position on organizational outcomes is statistically significant under distinct self-driven or other-driven network change conditions. If the preceding theoretical edifice is correct, and if we can empirically infer the presence of certain necessary conditions, we can conclude that the effect of the other-driven network position on the organizational outcome is plausibly causal.

Our empirical context is the life sciences (biotechnology) industry. We use data on alliances, acquisitions, and other corporate actions between 1995 and 2007, and data on patents between 1996 and 2012. This well-researched setting is advantageous because it allows us to replicate prior work before adopting our novel approach. Our replication confirms the positive relationship between structural holes and innovation reported in prior studies. Yet once we apply our research design, the positive relationship between structural holes and innovation holds only under conditions of *self-driven* network change. Under conditions of *other-driven* network change, structural holes have no effect on innovation (positive or negative). These findings are not driven by a variety of alternative explanations that we carefully assess.

Our central conclusion is that we cannot claim that the effect of structural holes on firm innovation, commonly documented in existing studies, is distinct from the firm's agency (which

drives its innovation goals, strategies, and capabilities). Thus, we are unable to draw causal conclusions that managing one's alliance network, in ways described in the current literature, leads to increased innovation *per se*. We discuss the theoretical implications of this conclusion for existing and future research. One possibility is that existing findings might reflect network effects that are spurious. Another possibility, which significantly enriches and redirects the literature, is that network position is necessary but not sufficient to produce a beneficial outcome; it needs to be paired with a firm's agency—its attributes or actions—to be activated (c.f. Smith, Menon, and Thompson, 2012). We highlight the theoretical and empirical implications of these possibilities to rigorously advance future research on networks and organizational performance.

THEORETICAL DEVELOPMENT

The networks perspective has become one of the most important lenses to understand how the external environment affects firms. Among the many outcomes affected by networks, innovation has received significant attention. Scholars often conceptualize innovation as a process of knowledge recombination, whereby firms obtain multiple bits of knowledge and put them together in novel applications (Fleming, 2001). Networks factor into this process because the structural position a firm occupies affects the amount of knowledge that flows to the firm, the variety of knowledge to which the firm is exposed, and the exclusivity of the knowledge available to the firm. These are crucial inputs into the process of recombination. While these considerations apply to multiple kinds of networks at different levels of analysis, we are interested in interfirm alliance networks.

A prominent application is the well-studied prediction that structural holes positively influence firm innovation. The theory is well known (Burt, 1992, 2004; Ahuja, 2000a). A firm that spans more structural holes has more disconnected partners, which likely belong to different knowledge communities. Because knowledge across communities tends to differ more than within communities, each disconnected partner exposes the focal actor to distinct bits of

knowledge. Because the partners are not connected, the focal firm is in the unique position of being the only one with access to the intersection of those distinct knowledge sources. Hence, networks with many structural holes expose a focal firm to a greater amount and variety of knowledge with greater exclusivity. Brokers, or those spanning structural holes, will have an advantage because they identify and access non-redundant ideas more frequently than other network participants.

Causal Structure of the Canonical Theory

Our approach to derive a novel research design and test of the causal link between network structure and organizational outcomes requires that we focus carefully on the underlying theory relating structural holes to innovation. To aid our theorizing, we employ structural causal diagrams (e.g. Pearl, 2010; Pearl and Mackenzie, 2018). We favor this approach because it requires us to be transparent about the underlying theoretical mechanisms. Moreover, it also requires us to be explicit about the assumptions necessary to consider empirically estimated relationships as causal. We emphasize that we are not offering a statistical solution as a testing strategy. Rather, we carefully assess and develop theory, which guides us to a novel research design and testing strategy.

Figure 1 presents the straightforward causal relationship expressed in the extant theory and tests. In our setting, spanning more structural holes leads to more innovation because the alliance network position inherently enables the mechanisms expressed in the canonical theory. Note that the established theory begins with the premise that the focal firm is already in a structural hole spanning position, but it does not tell us *how* it ended up in that position to begin with. Therefore, two issues complicate assessing the causal relationship in Figure 1. The first is that alliance network positions are not systematically imposed on firms; rather they reflect choices that firms make (i.e., they reflect firm agency). Existing research clearly shows that firm characteristics affect both their motivation to seek alliance partners (e.g., Shan, 1990; Hagedoorn, 1993; Dollinger, Golden, Saxton, 1998; Baum, Cowan, and Jonard, 2010) and their

attractiveness to potential alliance partners (e.g., Eisenhardt and Schoonhoven, 1996; Ahuja, 2000b; Baum et al., 2010).

The second complicating issue is that factors other than network position also affect innovation outcomes. Organizational theory points to firm characteristics—goals, strategies, and capabilities—as determinants of innovation outcomes (Cohen and Levinthal, 1990; Subramaniam and Youdt, 2005; Lavie and Rosenkopf, 2006). For example, a firm with scientific research capabilities will innovate more than a firm lacking these capabilities (Fleming and Sorenson, 2004; Fabrizio, 2009).

Figure 2 augments the causal diagram in Figure 1 by acknowledging that firm characteristics have direct effects on both network position and on innovation outcomes. Such characteristics are a confounder of the relationship between network position and innovation outcomes. They will lead to a correlation between network position and innovation outcomes even if no such causal relationship exists. The empirical implication is that an estimated relationship between network position and innovation can only be considered a causal effect if one controls for all firm characteristics that affect innovation without measurement error. Although conceptually straightforward, this is difficult to enact.

Organizational scholars are keenly aware of this issue and engage in many efforts to control for firm characteristics. First, recognizing the potential for confounding effects, many papers include controls of observable characteristics in regression analyses. Second, studies employ panel data to control for unobservable time-invariant firm characteristics through fixed-effects estimators (e.g., Powell, Koput, and Smith-Doerr, 1996; Ahuja 2000a). Third, some studies employ instrumental variables (e.g., Phelps, 2010; Ozmel et al., 2017; Chakravarty, Zhou, and Sharma, 2020).

Each of these approaches is valid and reflects progress in getting meaningful casual estimates. Nevertheless, they are largely statistical approaches with limitations in application. For instance, resource-based theories that predict linkages between firm characteristics,

network structure, and innovation outcomes emphasizes that such characteristics are often intangible and difficult to measure (e.g., research competence or capability) (Godfrey and Hill, 1995). Moreover, the set of firm characteristics that affect network structure and innovation outcomes is manifold, and the controls would have to be exhaustive to allow causal claims. Directly controlling for those characteristics is thus nearly impossible. This is a key motivation for scholars to employ fixed-effect estimators, which relax the need to identify and measure the underlying firm characteristics. However, this approach only accounts for time-invariant firm characteristics over the period of study. If unmeasured firm characteristics change over the panel, then causal inference might be lost.[2]

The causal diagram in Figure 2 demonstrates the requirements of a valid instrumental variable. It must not directly affect innovation (otherwise, it is another confounding variable) and it must not be determined by firm characteristics. Should this occur, we have the same problem as with network position. There would exist a "backdoor" path from firm characteristics to innovation—this time via the instrument. These are demanding assumptions to satisfy. For example, some studies use network-based measures like status to instrument for structural holes (e.g. Vasudeva, Zaheer, and Hernandez, 2013). The trouble with this approach is that it assumes that firm characteristics simultaneously affect and do not affect a firm's network. Other studies use firm characteristics to instrument for network position (e.g. Phelps, 2010), but this approach is subject to the assumption that the firm characteristics valued by potential alliance partners do not affect firm innovation.

A Theory-Driven Approach

Alliance-Network Externalities. Rather than focusing on a statistical solution to assess if there is a causal relationship between network position and innovation outcomes, we rely on a theory-driven approach. We leverage the insights from recent work showing that corporate

[2] We demonstrate that our data violate the assumption of stable unobservable effects.

actions (alliances, acquisitions, divestitures) initiated by one firm can indirectly influence the network position of other firms (Hernandez and Menon, 2018, 2021; Hernandez and Shaver, 2019), which we will refer to as “alliance-network externalities.” We define these externalities as changes in a focal firm’s ego network structure caused by the actions of another firm in the alliance network. We provide examples of such externalities later in the paper (see Figures 6a and 6b). Similar externalities could arise in other types of networks, but we are concerned with alliance networks—hence the “alliance-network” label.

Integrating the concept of alliance-network externalities with existing network theory highlights that a firm’s observed network position is the net effect of its own actions (self-driven) and the actions of other firms in the network (other-driven). Self-driven network change occurs when the process resulting in an observable network position (e.g. structural holes) is the result of a firm’s own actions. For example, a firm might achieve structural holes through a sequence of alliances, acquisitions, or divestitures it initiates. Other-driven network change occurs when the process is the result of other firms’ actions. For example, a sequence of alliances, acquisitions, or divestitures initiated by third parties might alter a focal firm’s position in the network such that it spans greater or fewer structural holes than before.

Agency. The key difference between self-driven and other-driven network change is in the locus of agency. In the case of self-driven change, the firm’s agentic intent is reflected in the resulting network, even if the various actions that resulted in the specific network position were not primarily meant to affect the network structure. For example, a firm might have conducted an acquisition to obtain a certain asset unrelated to the alliance network, but the resulting position in the network is still congruent with the broader corporate objective (Hernandez and Menon, 2021). In the case of other-driven network change, the focal firm’s agency is not manifest—the structural change reflects another party’s agency. Therefore, it does not reflect the intentions of the focal firm, even if the change is beneficial for the focal firm. For example, another firm’s acquisition in pursuit of a certain asset, unrelated to the alliance network, might

increase the focal firm's structural holes. Even if the focal firm desires a more open or diverse network, the fact that it now has one is the result of someone else's agency.

Agency has been a slippery concept in the networks literature, but it is at the heart of whether network theories (and empirical results) can be understood as causal. Some studies assume that network actors are purposeful and strategic in the pursuit of specific network positions (e.g., Jarillo, 1988; Jackson and Wolinsky, 1996; Buskens and Van de Rijt, 2008). Many studies in organizational theory, heavily influenced by the sociological origins of the literature, assume that social structure is too complex and invisible for any single network actor to control (e.g., Pachuki and Breiger, 2010; Tatarynowycz, Sytch, and Gulati, 2016). The former studies assume strong control over the network; the latter assume no control. Other scholars have taken an intermediate approach. They view firms as boundedly rational actors whose network actions support certain objectives (e.g. innovation, profit), with an imperfect understanding of the social structure, and some control within their immediate network neighborhood but little control beyond it (e.g. Gulati and Srivastava, 2014; Hernandez and Menon, 2021).

These agency assumptions are at the core of our efforts. If the network structure is fully outside the control of the focal firm, it is by definition exogenous: any theory or empirical result linking network structure to organizational outcomes would be causal. But when the process leading to the observed network position involves even partial agency by the focal firm (due to its capabilities, strategies, or goals), network effects cannot be considered causal *per se*. Scholars typically do not explicitly lay out their assumptions regarding network agency. But in the case of alliance networks, processes involving some degree of agency—even if constrained—are the most likely (Gulati and Srivastava, 2014). The literature on the antecedents of alliances and on innovation via alliances, which we cited earlier, makes the case strongly.

Thus, the key to identifying the causal effect of network position on organizational outcomes is to develop a theory that *suppresses* a focal firm's agency from the process resulting in its observed network structure. As we discuss next, the distinction between self-driven and other-driven change allows us to do so—under certain assumptions.

A Theoretical Path to Identify Causal Network Outcomes. Figure 3 adds the concept of alliance-network externalities to the causal diagram depicted in Figure 2. The top half of the figure replicates the relationships in Figure 2. Without loss of generality, we label these as 'self-driven' because, for a focal firm, its characteristics affect both its network position and its innovation outcome, while its network position affects its innovation outcome. The bottom half of the figure shows the same set of relationships for another firm. We label these as 'other-driven' because, for this other firm, its characteristics similarly affect its network position and innovation outcome and its network position affects its innovation outcome. The theoretical underpinning of alliance-network externalities is that each firm's network position can affect the other's network position. The arrows in both directions, from focal-firm network position to other-firm network position, reflect this.

The causal diagram in Figure 3 suggests a novel approach to theorize about and estimate the causal relationship between the focal firm's network position and its innovation outcome. The thick gray arrows in Figure 4 present this approach. The theory of alliance-network externalities predicts that other-firm network actions can affect a focal firm's network position. Established theories of network structure (such as structural holes theory), in turn, predict that the focal firm's network position affects its innovation outcomes. Integrated, these two theories predict that other-driven network change affects a focal firm's innovation. As long as there is no confounding path in this relationship—which we address later—this relationship represents a causal effect of network position on innovation.

Of course, one possible confounding path might arise from the focal firm's characteristics, which in Figure 3 are depicted as simultaneously affecting the focal firm's

network position and its innovation. However, the causal diagram in Figure 4 shows that, if we can find a way to block the path between a focal firm's characteristics and its network position (as depicted by the X over the path), then a focal firm's network position would only change because of a change in *other* firms' network positions. The estimated effect of ego network position on innovation in this situation would be the causal effect because there is no "backdoor" path to the focal firm's innovation (Pearl, 2010).

Figure 4 thus reflects a critical theoretical assumption: the other firms' network choices affect a focal firm's network position, but they are not confounded by a focal firm's characteristics. We describe how we operationalize this approach when we present our research design. We wish to emphasize that integrating theory linking network structure and innovation with theory on alliance-network externalities provides the insight for this novel estimation strategy—without theoretical guidance, this approach would not be apparent.

This integration of the two theories leads to our central hypothesis, which is a causal expression of the prediction made by the original theory of structural holes.

> *Hypothesis: Under conditions of other-driven network change, structural holes increase a focal firm's innovation.*

We do not predict a relationship between structural holes and innovation under conditions of self-driven change, which is the commonly tested hypothesis in the literature. As previously discussed, it is difficult to claim that this relationship can capture the theoretically proposed causal relationship that structural holes increase innovation.

Necessary Assumptions. The causal diagram in Figure 4 makes explicit assumptions that must hold for our hypothesis to reflect a causal test.

Figure 5 presents two relationships (depicted by red arrows) that would invalidate our approach. Our hypothesis assumes that these relationships do not exist or are not material. The first problem would arise if the focal firm's characteristics affected the other firm's network position. The existence of this path would reflect that, when engaging in actions that shape its

network, the other firm considers the capabilities, objectives, or other attributes of the focal firm. There are plausible scenarios under which this could happen. For example, if firms consider their competitors' network actions when building their own network positions. In our case, this would be manifested in other firms' actions systematically increasing or decreasing the focal firm's structural holes—such as rivals intentionally closing each other's structural holes to undermine their innovation capabilities. When such linkages exist, focal firm characteristics not only affect their own network positions, but also do so through others' network positions. We end up with a concern like the one originally depicted in Figure 2. Focal firm characteristics affect innovation directly, but also *indirectly* through the focal firm's network position because of alliance network externalities.

The second red arrow represents the possibility that other firms' innovation outcomes affect the focal firm's innovation outcomes directly. This path reflects the possibility of technological spillovers within the industry, among other mechanisms. For example, firms observe one another's innovation outcomes, which in turn aids their innovation efforts—as suggested by research on positive knowledge spillovers (e.g. Audretsch and Feldman, 1996). This path could also exist if innovation is characterized by patent races (e.g. Baum, Calabrese, and Silverman, 2000). In this case, when a firm receives a patent (i.e., innovation outcome of the other firm) it precludes granting another firm obtaining this patent (i.e., innovation outcome of the focal firm). While research has documented these types of positive and negative innovation spillovers, the existence of this path leads to spurious outcomes in our case only if changes in the *other* firm's network correlate with the alliance-network externalities it imposes on the focal firm. For example, if firms reduce the structural holes of their competitors when they increase their own structural holes. Under this scenario, the path that we test between focal-firm structural holes and innovation would also capture the relationship between other-firm structural holes and innovation plus the impact of other-firm innovation on focal-firm innovation. Absent the correlation, such as spurious relationship would not be manifest.

We emphasize that we are not simply assuming away the two mechanisms depicted by the red arrows. Indeed, we have provided realistic scenarios—some documented in prior literature—under which they might be in play. The advantage of the causal mapping exercise is that it makes these assumptions explicit and forces us to assess if they hold, which we do in the empirical part of this paper. This provides transparency to our process of determining if the causal claim of our hypothesis is valid.

Further, Figure 5 reveals that the assumptions underlying our approach are implicit in *all existing studies* that examine how network position affects innovation outcomes. Violating these assumptions creates mechanisms by which the path between structural holes and innovation can reflect a confounding effect rather than a direct causal effect. The prevalence of these implicit assumptions in existing work only becomes clear by formally integrating theory on how structural holes affect innovation with theory on how structural holes arise in the first place (i.e. via alliance-network externalities, in our case). We now turn to the data to test our hypothesis and validate the necessary assumptions.

RESEARCH DESIGN

As we document in Figure 4, our hypothesis expresses a causal relationship between structural holes and innovation outcomes if we can block the path between self-driven network change and innovation outcomes (subject to the specified assumptions). Our approach to do this is directly through measurement. We identify periods when firms enact changes to their network and identify periods when they do not (self-driven vs. no self-driven change). We also identify periods when other firms nearby the focal firm in the network enact changes to their networks and periods when this does not occur (other-driven vs. no other-driven change). Juxtaposing these observations, we can isolate periods in which the focal firm's network is subject exclusively to conditions of other-driven change from conditions involving self-driven change. The latter observations block the path between self-driven network change and

innovation outcomes, whereas the former do not. This becomes the basis for assessing the effects of structural holes under both conditions.

Before describing the data and measurement, we provide tangible examples that map to our approach. Figure 6a provides an example of other-driven network change. The focal firm, Oxford Molecular, did not initiate any structure-modifying actions between 1998-1999. However, two of its network neighbors did: Polymasc merged with Valentis, while Celltech established a pair of new alliances. As a result, Oxford Molecular's network constraint decreased (its structural holes increased) through no action of its own. In this year, the path between self-driven network change and innovation outcomes is blocked because the focal firm did not initiate any changes. However, the path between other-driven network change and innovation is 'opened' as a result of Valentis and Celltech's actions. Figure 6b provides examples of how other-driven network change can increase or decrease a focal firm's constraint. Between 2002 and 2003, two of Lifespan Biosciences partners ended their alliance, increasing the structural holes of Lifespan. A year later, two of its other partners formed a tie with each other, decreasing Lifespan's structural holes.

Figure 6c illustrates a case of self-driven change for Watson Pharmaceuticals between 1996 and 1997. Watson began the period as a highly disconnected firm, on the periphery of a cluster controlled by firm #45. By undertaking two acquisitions and initiating two alliances, Watson put itself in the center of a network spanning three distinct clusters in the alliance network. In this example, the path between self-driven network change and innovation outcomes is open. Measuring self-driven changes is the common approach in the literature. We also note that the literature overwhelmingly focuses on self-driven changes from alliance formations and dissolutions. It has not consistently considered self-driven network changes resulting from acquisition activities, which this figure highlights, or from divestitures. We account for all of these in our measures.

Empirical Setting and Data

We perform our analysis in the context of the life sciences (biotechnology) industry, which we choose for the following reasons. Using a well-researched context in which alliance network structure is associated with innovation (Sytch and Bubenzer, 2008) has advantages when implementing a novel test. We can first replicate the results of prior work to provide confidence that our findings are not driven by the choice of setting. Our approach also requires accounting for multiple types of corporate actions—alliances, acquisitions, divestitures, entries, and exits—and the life sciences industry exhibits substantial activity in all of them. In addition, alliance networks play a crucial role in the innovation and performance outcomes of firms in this industry because technological development is too complex for firms to go it alone (Baum, Calabrese, and Silverman, 2000). Life sciences firms value innovation because it is directly associated with performance, and they systematically file patents for any significant innovation they create. This provides a measurable form of innovation output and allows us to capture most firm innovations. Finally, excellent sources of data on firms' networks, corporate actions, and patents are available for this industry.

We construct the alliance network for the period spanning 1995-2007, using data from the *Recombinant Capital* (Recap) database. Every entry in Recap is defined by an agreement between two or more firms to cooperate on a life sciences activity. The firms in the sample are small to medium biotechnology firms and large pharmaceutical firms. We define an alliance as any form of voluntary collaboration to exchange, share, or co-develop resources in which the two firms remain independently owned (Gulati, 1998) because our interested is knowledge-related collaborations that plausibly affect patentable innovations. Research shows that many kinds of alliances help firms develop new knowledge, so we include various types of collaborations (e.g. R&D, licensing, manufacturing, etc.) and drop those that clearly have no potential for knowledge transfer (see Alcacer and Oxley, 2014). The eliminated deals include categories such as asset purchases, loans, and settlements.

We identify 19,131 alliances initiated between 1991 and 2007 involving 7,910 unique firms. Consistent with prior research, we assume that each alliance has a five-year lifespan, after which it is terminated (e.g., Gulati, 1995; Stuart, 2000). To have a full five-year alliance duration in our first year of observation, our sample begins in 1995 (with alliances formed between 1991 and 1995). We then capture the alliance network in each subsequent year through rolling 5-year windows. Our final year of observation for the network is 2007 because we have Recap data only until that year.

Although alliance ties define the network, a central aspect of our research design is the fact that other corporate actions—acquisitions, divestitures, entries, and exits—can restructure the network. Like prior work, we assume that firms enter or exit the industry network based on their appearance and disappearance from the Recap database. A firm enters the network in the first year in which it appears in Recap. If a firm has not been active in Recap for 5 years, we consider that it is no longer active in the alliance network, consistent with the assumption made in prior work.[3] To account for acquisitions and divestitures, we obtain data on those events from *SDC Platinum* for the years 1995-2007. Please see Appendix A for an explanation of how we accounted for those events when constructing the alliance network.

We obtain data on firms' patents from the USPTO's *PatentsView* database. Because we observe patenting outcomes in the 5-year period following the observation of the alliance network, we gathered patent data for the years 1996-2012. For instance, if we observe a firm's network position in 2007, we capture the patenting outcomes for that firm during 2008-2012—in line with prior research (e.g., Fleming, King, and Juda, 2007; Balachandran and Hernandez, 2018). Results are robust to using other time windows.

[3] We note that firms could still be active in the industry even if they are not actively involved in alliances, for example by engaging in internal R&D. A firm can re-enter the network if it establishes an alliance more than five years after its previous alliance in Recap, although this is highly unusual in our data.

Measures

Innovation. We measure innovation using the two most common metrics in the literature: *patent counts* and *citation-weighted patent counts* (e.g. Ahuja, 2000a; Sampson, 2007; Vasudeva, Zaheer, and Hernandez, 2013). We calculate patent counts by summing the number of patent applications made during the five-year window after the focal year, as explained above. Like all prior work in this area, we keep only patents that were eventually granted, but we consider the year of application as the moment in which the invention was created. We measure citation-weighted patent counts by multiplying each patent by the number of citations it receives during the five-year window following the application date (e.g., Vasudeva, Zaheer, and Hernandez, 2013; Funk, 2014) and summing all the firms' citation-weighted counts for the five-year period following the focal year.

Structural Holes. We use Burt's (1992) network constraint measure to capture a firm's access to structural holes:

$$C_i = \sum_j c_{ij}, \quad i \neq j$$

$$c_{ij} = (p_{ij} + \sum_q p_{iq} p_{qj})^2, \ i \neq q \neq j$$

where C_i is the network constraint of node i and c_{ij} is node i's dependence on its contact j. The contact-specific dependence, c_{ij}, is calculated from the proportion of i's ties invested in contact j, both directly (p_{ij}) and indirectly ($\sum_q p_{iq} p_{qj}$). Higher constraint indicates fewer structural holes, so the canonical theory predicts a negative relationship between constraint and innovation.

Measuring self-driven and other-driven network change

Assessing if self-driven changes impact a firm's network position is straightforward. It entails tracking the formation or dissolution of alliance ties plus acquisition and divestiture activity involving the focal firm. Therefore, for each year we assess if a firm engaged in actions that changed its own network and the extent to which it changed.

In contrast, determining whether a firm was affected by another party's actions is more complicated because the potential for alliance–network externalities varies according to the proximity of the focal firm to *other* firms involved in alliances, acquisitions, or other relevant activities. For instance, an acquisition by a directly connected partner potentially causes a much greater change in the focal firm's network compared to an acquisition by a firm several degrees away in the network. The same is true for the alliance formation or dissolution actions of others. Like with an earthquake, the distance to the "epicenter" determines whether a firm is truly subject to an alliance-network externality.

Because network constraint is an ego-network measure, its "radius" involves only one degree. We thus follow the rule that a focal firm's structural position is potentially impacted when it is one degree of separation from another firm directly involved in an acquisition (either the acquirer or the target) or a firm forming or terminating an alliance (to either of the allying parties). This radius includes changes in the ties between the focal firm and its direct partners as well as the ties among the focal firm's partners. We note that we measure the potential for change—if any action by another firm happened within one degree, we include it in our measurement. Hence, it is possible that the actions by a focal firm's partners do not result in actual changes to the focal firm's network position. The results are unaffected if we only include actual changes in the structure in our measures.

Building from these data definitions we can classify every firm-year along two dimensions. The first is whether the focal firm enacts network changes or not. The second is whether firms within one degree radius of the focal-firm enact changes in their networks or not. This results in a 2x2 matrix. The cells in the matrix capture four mutually exclusive and collectively exhaustive categories of network change: (1) *self-driven-only* (i.e., determined only by a focal firm's actions), (2) *other-driven-only* (i.e., determined only by another firm's actions), (3) *simultaneously self-driven and other-driven* (i.e. determined by both a focal firm's actions

and others' actions), or (4) *no change* (i.e., neither the firm nor its other firms within one degree of separation initiated an action).

With respect to the third category (simultaneously self-driven and other-driven network change), we would ideally like to separate the portion of change resulting from the firm's own actions from the portion resulting from others' actions. However, this is not feasible because we cannot identify exactly which of the focal firms' versus others' actions lead to modifications in each individual tie comprising the focal firm's ego network. But this is not problematic because the main distinction is between *other-driven-only*, which we can isolate cleanly, and any condition involving self-driven change. Whether *self-driven-only* or *simultaneous self-driven and other-driven*, they are "contaminated" by the focal firm's agency and their impact on innovation cannot be considered causal.

Table 1 shows the incidence of all four categories of network change. In all, 26,963 firm-year observations involve other-driven changes, and 13,650 observations involve self-driven changes. There is a meaningful number of *other-driven-only* changes in the data—our category of main interest (14,012 or 44 percent of all firm-year observations). Although there are not many cases of self-driven-only change (699 firm-years), we observe a large number of cases of simultaneous self-driven and other-driven change (12,951).

**** INSERT TABLE 1 ABOUT HERE ****

Because the same firm can (and does) appear in different network change categories across years, one might be concerned that systematic sequences of switching among categories could influence our findings. For instance, if firms always oscillate from *self-driven-only* to *other-driven-only* (or any other systematic sequence), the causal effect of *other-driven-only* for which we advocate might instead be a spurious artifact of the sequence. To check for this, we mapped out all the observed sequences by which firms moved across categories, as reported in Appendix C. We found no evidence of systematic sequences. The concern about such sequences contaminating the results is stronger the longer the time window over which we

measure our dependent variable. We thus also verified the robustness of our results to using shorter DV windows (see Appendix B).

Validating our first assumption. Recall that one of our key assumptions is that network changes initiated by other firms are not performed to affect the focal firms' structural holes in a systematic manner. To assess this, we investigate the magnitude and variance of self- vs. other-driven changes.

One potential concern is that other-driven network changes might be so small as to make it difficult to find effects in empirical tests. It is not necessary that other-driven and self-driven network changes be similar in magnitude. Sufficient for our purposes is that meaningful variance exists across both types for empirical testing to be feasible. Table 2 shows that both *self-driven-only* and *other-driven-only* events produce meaningful variance in network constraint from year to year. The top panel reports statistics on potential changes (used in our main analysis), while the bottom panel reports statistics on actual changes (results are robust if we use these measures). Unsurprisingly, *self-driven-only* changes create larger average modifications in constraint than other-driven actions. Note that the vast majority of potential *other-driven-only* changes do not result in actual changes. But the variance of actual changes produced by *other-driven-only* is larger than the variance of *self-driven-only* actions. We also find meaningful variance when network changes are simultaneously other- and self-driven.

**** INSERT TABLE 2 AND FIGURE 7 ABOUT HERE ****

We depict the distribution of actual changes caused by *self-driven-only* and *other-driven-only* change in Figure 7. Virtually all (97%) of the self-driven constraint changes resulted in decreased network constraint. This reflects that firms initiating network changes tend put themselves in positions to span more structural holes, which according to theory is beneficial. It is also highly consistent with work showing that in high-technology industries, like biotechnology, firms exhibit a clear tendency to pursue structural holes (Tatarynowycz, Sytch, and Gulati, 2016). Crucially for our purposes, other-driven network changes cluster around zero,

suggesting that they do not systematically benefit or harm a focal firm's network structure: 47.5 percent lowered the network constraint of the focal firm and 52.5% increased it. Of course, most cases in which a focal firm is exposed to *other-driven-only* change result in no actual change in the focal firm's constraint. This further reinforces the notion that other firms' actions are not intentionally aimed at the focal firm's network position. There is no obvious pattern in the direction of other-driven changes, consistent with our key assumption.

EMPIRICAL RESULTS

Replication and Extension of Prior Results

The standard approach to estimate the effect of structural holes on innovation is to regress a measure of innovation on structural holes and rely on within-firm variation to identify the coefficient estimates (i.e., include firm-fixed effects). We replicate this standard approach in Appendix A. We confirm the results of many studies in models 1 and 2 of Table A1: structural holes are associated with increased innovation. Note that we present most results without time-varying control variables for simplicity of exposition. The findings and conclusions remain qualitatively unchanged in models where we add several control variables, which we report in Appendix B2. We note that there is no clear agreement in previous research as to what control variables are essential—other than including firm and year fixed effects, which we include in all specifications. In models 3 and 4 of Table A1, we demonstrate consistent results when including changes in network structure driven by acquisitions and divestitures. These analyses situate our test within the current literature.

We undertake one additional analysis to set-up our ultimate hypothesis test. We use a slightly different approach to estimate the within-firm effect found in current research. Instead of the using a fixed-effect estimator, we use a first-difference estimator. The fixed effects estimator assumes that unobservable firm effects are constant. We are concerned that this assumption may not hold in our data because the duration of the panel is long (13 years, from 1995 to 2007) and the biotechnology industry is fast-changing. It seems unrealistic to assume that firm-specific

characteristics stay the same for over a decade. In a first-difference specification, all the variables (dependent and independent) are subtracted from the values of the firm's previous year's observation. For example, *constraint* in year t-1 is subtracted from constraint year t. The key independent variable now becomes Δ*network constraint*. Like the fixed-effect estimator, this model accounts for unobservable firm effects, but it makes the less-restrictive assumption that unobservable effects can change yet follow a random walk (Wooldridge, 2012).

Table 3 presents these results. In models 1 and 3, We continue to find a statistically significant negative effect of network constraint on the two patenting outcomes. In fact, the statistical significance when estimating citation-weighted patent counts increases. However, the magnitude of the coefficients is much smaller than the fixed-effect estimates in models 2 and 4. The estimate of the impact on patent counts is about one quarter the magnitude and the impact on citation-weighted patent counts is about half the magnitude. The reduction in effect sizes suggests that time-varying unobservable effects, which are not captured in the typical fixed effects model, play an important role in determining the outcomes. Yet we still find that structural holes are positively correlated with patent outcomes.

Effect of Self-Driven vs. Other-Driven Structural Holes on Innovation

We now report the results of our novel approach. Because the four categories of network change reported in Table 1 are mutually exclusive and collectively exhaustive, we can use these classifications to partition Δ*network constraint* to reflect different conditions of network change. We first segment Δ*network constraint* into two variables: Δ*network constraint other-driven-only* and Δ*network constraint not-other-driven-only*. The variable Δ*network constraint other-driven-only* takes the value of Δ*network constraint* when the observation is in the other-driven-only category and zero otherwise. Notice that this variable captures the condition that maps to the causal test of our hypothesis. The path between self-drive network change is blocked (i.e., there is no self-driven network change); however, there is the potential for other-driven network change. We include the variable Δ*network constraint not-other-driven-only* in the specification to

capture all other conditions of network change. This variable takes the value of Δ*network constraint* when the observation is not in the *other-driven-only* category and zero otherwise.

**** INSERT TABLE 4 ABOUT HERE ****

The results in Table 4 demonstrate that the effect of Δ*network constraint* for *other-driven-only* network change does not test different from zero for either of the two innovation measures. Moreover, the coefficient estimate is positive for patent count—contrary to the canonical theoretical expectation. These results fail to support our hypothesis that assesses if the effect of structural holes on innovation is causal.

We find that Δ*network constraint* exhibits a negative and statistically significant effect on innovation for changes that are not-other-driven-only (i.e. "contaminated" by the focal firm's agency). A one-unit decline of network constraint is associated with an increase of about 0.55 patents and 2.6 citation-weighted patents. As we described in the theoretical derivation of our hypothesis, we cannot make causal claims about this statistically significant relationship. In models 2-3 and 5-6, we examine the effect of entering these variables separately to ensure that collinearity does not lead to spurious inference. The coefficient estimates and standard errors are almost identical across these specifications.

We further partition Δ*network constraint* in table 5, which allows us to refine our understanding of what drives the results of network constraint on innovation. We examine the effect of Δ*network constraint other-driven-only,* Δ*network constraint self-driven-only,* and Δ*network constraint simultaneously-other-driven-self-driven*. We drop observations with no change in network constraint because there is no variation in this group (i.e., all have zero values of Δ*network constraint*). The results in Table 5 are consistent with those in Table 4. The coefficient of Δ*network constraint other-driven-only* does not test different from zero, and the sign of the estimate is positive for patent count. The coefficient of Δ*network constraint self-driven-only* exhibits a statistically significant negative effect on innovation: a unit decline of network constraint is associated with an increase of about 0.8 patents ($p < 0.01$) and 6.9

citation-weighted patents ($p < 0.05$). The coefficient of Δ*network constraint simultaneously-other-driven-self-driven* is also negative and significant: a unit decline of network constraint in this condition is associated with an increase of about 0.54 patents ($p < 0.01$) and 2.1 citation-weighted patents ($p < 0.05$).

Validating Our Second Assumption. Recall the second assumption necessary for other-driven constraint to have a causal effect on innovation. The patents of the focal firm and other firms should not affect one another in a manner that explains the effect of the focal firm's constraint on its own innovation. Because this assumption depends on the direction of the estimate of constraint on innovation, we can only assess it after seeing the results. We have documented a null relationship between other-driven network constraint and innovation. Therefore, our second assumption would be violated if the patents of the focal and other firms correlated in a manner that *suppresses* an (in reality) negative effect of focal-firm constraint on focal-firm innovation.

In other words, the effect of other-driven constraint on innovation—which we estimated as null—is actually negative and significant, but we do not observe it because our second assumption is violated. This will occur only if the effect of other-firm constraint on focal-firm constraint is in the *opposite* direction of the effect of other-firm innovation on focal-firm innovation. Because the canonical theory advances a negative relationship between constraint and innovation, then other-firm constraint should positively affect other-firm innovation. In turn, if our assumption is violated, then other-firm innovation must affect focal-firm innovation *negatively*. That negative effect will suppress the effect of focal-firm constraint on focal-firm innovation only if other-firm constraint *negatively* affects focal-firm constraint. To reiterate, other-firm network change creates a positive effect on innovation through focal-firm constraint that is offset by the negative effect through innovation spillovers between firms.

The patterns in our data are not consistent with this possibility. We return to the empirical observation that there is no systematic pattern in how alliance network-externalities

affect the focal firm's structural holes. Recall that the distribution is centered on zero. The relationship that would violate our assumption requires a systematic relationship between focal-firm and other-firm constraint in the opposite direction to a relationship between focal-firm and other-firm patents.

Other Sensitivity Analyses. We run the same models including small set of firms that experienced divestitures (these firms were previously dropped in the main analysis, as explained in Appendix A). The results remain robust with the added observations (see Appendix A3). Second, we use alternative windows of observation for the dependent variables (see Appendix B1). Patent counts and citation-weighted patent counts are aggregated during three- and four-year windows instead of five-year windows. The results remain robust for Δ*network constraint other-driven* in both the four-year and three-year windows.

INTERPRETATION AND IMPLICATIONS

Our findings show that structural holes do not affect patent outcomes under conditions of other-driven change. We conclude that network position, *per se*, does not affect innovation. Nevertheless, we replicate existing findings showing that network position correlates with patent outcomes for the overall sample and for situations involving self-driven changes in network position. Combining these sets of findings lead to two possible interpretations: (a) network effects are spurious or (b) network effects exist, but only in concert with other attributes or actions that reflect the agency of the focal firm. We discuss how these possibilities shape theoretical and empirical directions for future research.

Network effects are spurious

This interpretation reflects the possibility that network position only captures the attributes of firms or dyads in those positions, and these attributes—which are not fully controlled for in empirical tests—affect innovation outcomes. In other words, the specification of empirical tests in the literature is susceptible to omitted variable biases (e.g., Shaver, 1998), and the body of findings reflect this bias.

This is a plausible explanation because extant research provides evidence that many firm and dyad attributes correlate with innovation outcomes. Firm-level factors include capabilities or resources that enable innovation (e.g. Rothaermel and Hess, 2007), which by their nature are hard to measure (Godfrey and Hill, 1995). Dyadic factors include partner-specific qualities such as trust, absorptive capacity, or routines that play an important role in the benefits that firms get out of alliances (Dyer and Singh, 1998; Dyer, Singh, and Hesterly, 2018). Likewise, a growing stream of research considers alliances as a matching process (Mindruta, 2013; Mindruta, Moeen, and Agarwal, 2016; Fudge Kamal, Honore, and Nistor, 2021) and this implies that capabilities among alliance participants tend to be complementary (Nakamura, Shaver, and Yeung, 1996; Baum, Cowan, and Jonard, 2010). Therefore, the interaction of alliance partner characteristics, which are difficult to isolate and measure, could be important unobserved factors. Moreover, the set of factors we just listed is unlikely to be exhaustive, suggesting that other attributes can also affect innovation.

Additional evidence that unmeasured effects play and important role for innovation outcomes comes from current tests that employ panel data and firm fixed effects. In these studies, the fixed effects often test significant, which indicates the presence of constant unobserved firm attributes. When we relax the assumption that unobserved firm attributes are constant over the panel in our study (i.e., when we move from the fixed effect specification to first difference model), we find the magnitude of the network effect reduces by 75% for patent count and 60% for citation-weighted patent count. This suggests that the unobserved attributes that correlate with innovation change over time, and studies that control for them with firm fixed effects do not fully capture these effects.

The interpretation that alliance network effects are spurious is, of course, problematic for the literature concerned with networks and organizational outcomes. But it does not threaten the vast research program on network antecedents and dynamics. In fact, it adds urgency to such work because it suggests that research on outcomes cannot effectively proceed without a better

understanding of network generating processes (Ahuja, Soda, and Zaheer, 2012). But spuriousness, while plausible considering our results, is not the only interpretation offering a path forward for organizational networks research.

Network effects exist but require agency to be activated

The other potential interpretation is that network position affects organizational outcomes such as innovation; however, network structure alone is insufficient to produce those outcomes. Instead, network effects require some additional factor to be activated (c.f. Smith, Menon, and Thompson, 2012). Our results strongly point to *agency* as the activating factor. As we noted before, the difference between self-driven and other-driven network generating processes is the locus of agency. We find that the effect of structural holes on innovation manifests itself only when the focal firm exerted its agency to achieve its network position. When others' agency is manifest, but not the focal firm's instrumentality, there is no effect. Just being in a certain position is not enough—it appears that the firm needs to be aware, motivated, and able to do something with its position. This has profound implications for how we theorize (and test) network effects.

Back to the future? While the point is more general, it can be usefully illustrated by returning to the example of structural holes and innovation. A careful reading of Burt's formulations of the theory (particularly the in the 1992 book and in the 2004 study of "Structural Holes and Good Ideas") makes it clear that structural explanations are inseparably paired with certain qualities and actions of the individuals who end up in brokerage positions. For example, Burt is explicit about brokers having a "tertius gaudens" behavioral orientation that leads them to consciously seek for personal gains from their position (1992: 30-32). Obstfeld (2005) suggests the "tertius iungens" as a different behavioral orientation for brokers. Either way, specifying some kind of orientation matters. Moreover, Burt discusses "the issue of motivation" by arguing that certain brokerage benefits "require an active hand" and that "a player can respond in ways ranging from fully developing the [brokerage] opportunity to ignoring it" (1992:34). Burt's

theoretical solution to the issue is “to leap over the motivation issue by taking the network as simultaneously an indicator of entrepreneurial opportunity and of motivation” (1992: 35). In other words, agency is baked into the theory.

Years later, commenting on the many empirical studies showing an association between brokerage and performance benefits (mainly for individual-level networks), Burt said that “evidence on the mechanism is not abundant” and, intriguingly, suggested that “the association cannot be causal. *Networks do not act, they are context for action*” (2004: 354, emphasis added). Burt then lays out his theory linking structural holes to good ideas, which upon careful reading includes not just structural mechanisms but also specifications of the attributes and behaviors of brokers (similar to those originally laid out in 1992).

Putting aside the issue of whether Burt’s motivational and behavioral assumptions are correct, it should be evident that the original theory of structural holes is not *purely* structural. However, when imported to organizational-level contexts, the agency assumptions seem to have been lost in translation. The emphasis has been placed almost exclusively on structural explanations. In fairness, scholars have probed several contingencies or interaction effects (as we noted above). But there is a difference between arguing for an interaction effect—which keeps the structural theory of the main effect intact—and incorporating the actor’s agency as a necessary factor to active the effect of network structure on an organizational outcome.

Thus, to properly advance theory, scholars need to go “back to the future” and be clearer about what aspects of agency are necessary and sufficient for network position to affect firm-level outcomes. In that spirit, we next illustrate how two kinds of variables— organizational attributes or organizational actions—that reflect how firm-level agency can be incorporated as activating factors when theorizing networks and innovation.

Attributes. In this view, network position affects innovation outcomes but only in combination with firm or dyad characteristics that are not measured in current empirical analyses and that also influence network position.

For example, consider a firm on the verge of a technical breakthrough in drug development. This firm might be more willing to engage in alliances to leverage this emerging capability, and potential alliance partners could be more willing to ally with the firm due to its emerging capability. Once allied, because of that capability, the firm might be better able to leverage its brokerage position to combine information from other firms' innovation efforts with its emerging capability. This scenario, and other parallel scenarios, would be consistent with our results.

Because this is an emerging technology, it would not be possible to measure with typical patent-based measures. Because it entails a change in capability, it would not be controlled for by a firm fixed effect. And because it leads to increased likelihood of alliances (modifying the motivation and attractiveness of the focal firm), the emerging capability would correlate with changes in alliance activity in such a way that our first-difference approach would not eliminate. However, rather than just reflecting a spurious effect, the emergent capability is what allows the firm to leverage its brokerage position.

We recognize that the existing literature hypothesizes and tests firm contingencies that enhance network positions, of which we highlight a few. One of those is the firm's absorptive capacity, which reflects underlying capabilities (Shipilov, 2009). Other studies highlight the dyadic or relational attributes, such as relative bargaining power (Bae and Gargiulo, 2004; Shipilov, 2009), relative knowledge composition (Phelps, 2010; Ter Wal et al., 2016), and tie strength (Burt, 2000; Tiwana, 2008). Yet other work focuses on macro-level factors, such as the institutions in which the actors are embedded (Lin et al., 2009; Vasudeva, Zaheer, and Hernandez, 2013; Zhu and Chung, 2014). Although the contingency approach to studying network effects is conceptually related to what we describe, findings from these studies do not necessarily provide evidence for the joint effect of position and attribute. Their empirical approach is to interact a firm attribute with network position. But that approach can capture an interaction between the measured attribute and an unobserved effect that leads to network

position, rather than an interaction between the firm attribute and network position. For example, if firms with emerging capabilities are more likely to have favorable network positions, then any of the aforementioned studies might demonstrate an interaction with emerging capabilities rather than network position.

Actions. In this case, a firm must purposefully engage in certain actions to take advantage of a network position it happens to occupy.

For example, to benefit from a brokerage position, a firm must undertake a number of internal processes. It must move appropriate personnel into positions where they can interact with alliance partners and leverage their expertise with the novel information. It must provide these individuals time and resources to assess and integrate into the firm the novel information to which they are exposed. And it must leverage an organizational design that facilitates knowledge transfer and recombination within the firm. Therefore, network position must be paired with managerial actions and organizational processes.

This scenario, and other parallel scenarios, would lead to the result that we demonstrate: that network effects are significant only in cases where firms self-initiate the corporate actions that produce changes in the network structure. While difficult to observe, firms that purposely put themselves into advantageous network positions are also likely engage in these internal processes to leverage their network positions. In contrast, firms exogenously thrust into advantageous network positions are unlikely to initiate the required internal processes. Therefore, they do not benefit from their accidental network positions.

Some prior work provides hints about the importance of such internal processes and tries to identify them. Tiwana (2008) finds that the effects of bridging and strong ties on performance are mediated by the “the process of jointly applying specialized knowledge held by various alliance partners at the project level” (Tiwana, 2008: 255). Studies on alliance management capability also hint at the value of internal organizational design. For instance, Kale, Dyer, and Singh (2002) argue that a dedicated alliance function enhances a firm’s ability

to strategically capture alliance-related knowledge. Such work is useful in suggesting processes that are measurable, though many of the underlying actions that help activate network benefits are likely to be unobservable. And like with measures of attributes, interacting measures of organizational actions with network position might capture interactions with the unobserved effects that lead to network positions.

Principles for advancing future research. The interpretation that agentic activation (vs. spuriousness) is necessary for network effects to causally manifest at the organizational level pushes scholars to turn agency from a "bug" into a "feature" of research. Fully accomplishing this requires refinements and advances in both theory and research design.

Turning first to theory, scholars must build accounts that logically derive the attributes or actions influencing agency in the process generating the observed network position. We relied on the theory of alliance-network externalities, but we recognize that many other theoretical accounts are possible. Such theories would specify how the agentic attributes or actions are conceptually distinct from network position. Separately, the theoretical reasoning would have to advance why these attributes or actions work in concert with network position. Inasmuch as extant network theories include attributes or processes, they often argue that network positions embody them inherently (e.g., structural holes = tertius gaudens = brokerage). This does not satisfy the nature of the theoretical relationship our results suggest might exist. Further, such a theoretical exercise would differ for distinct types of networks operating at different levels of analysis (e.g., a knowledge network composed of individuals vs. teams vs. firms).

Refining or advancing theory in this manner also has profound implications for testing—especially research design requirements. A good test of a theory with this structure requires that we (a) invoke variance in network position, (b) independently invoke variance in the attribute or action, and (c) ensure that the ways in which we invoke variance does not simultaneously invoke variance in other factors that might affect the outcome. Note that this study does not meet all those requirements, nor was that our objective. We have identified one way to invoke

variance in network position not driven by the focal firm's agency (points a and c, above), but our goal was to suppress agency (the "bug" view) rather than embrace it (the "feature" view). Nevertheless, our approach provides a foundation for a test that incorporates agency variables.

Beyond what we do, scholars need to measure the underlying attribute or action advanced by theory development and find a way to invoke variance in the measure without invoking variance in network position (b and c). In other words, if this measure and network position always move together, then it will be difficult to test the interpretation that the outcomes of network position are activated when paired with the measure. Finally, research must find ways to invoke variance in the measure to mitigate the likelihood that it captures other factors that affect the organizational outcome (i.e., the standard endogeneity concern).

We recognize that the theoretical and testing requirements we present are demanding. Nevertheless, the study of interorganizational networks has matured to a point where advancing theory and testing in this manner is warranted. Such a goal is consistent with trends in social science focusing on the development and testing of causal theories.

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FIGURE 1. Causal diagram of the canonical theoretical relationship

FIGURE 2. Underlying theoretical relationship recognizing the confounding effects of firm characteristics

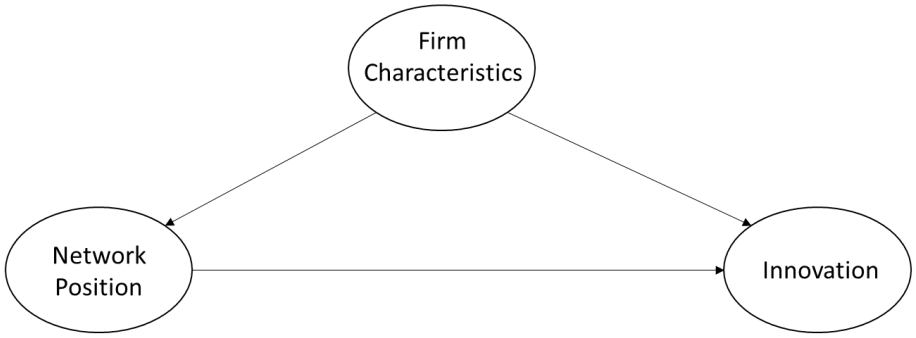

FIGURE 3. Integrating network theory and theory of alliance-network externalities

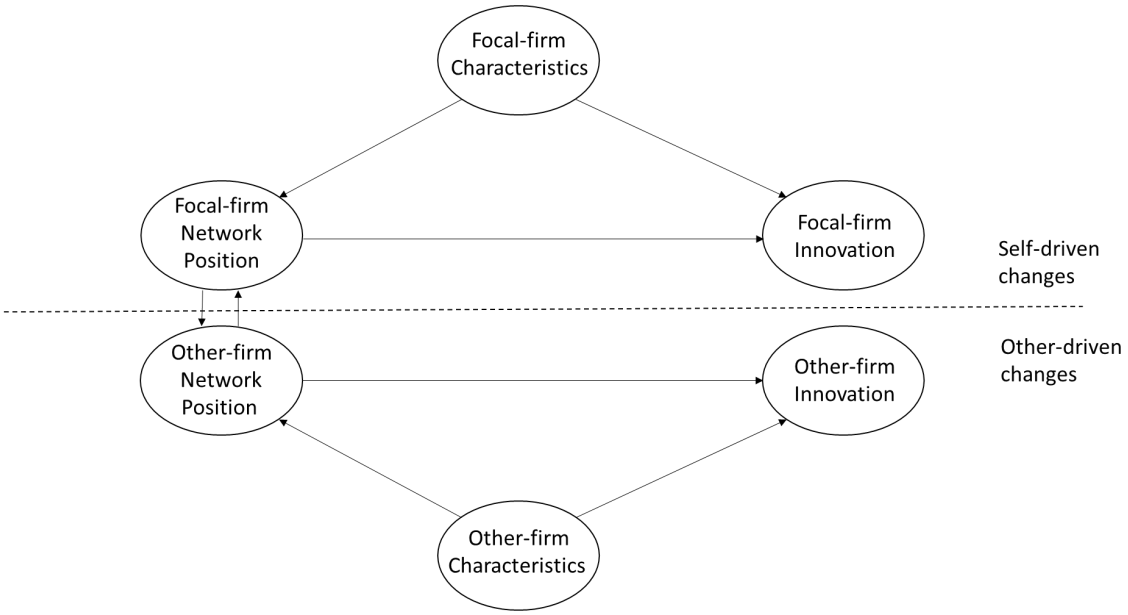

FIGURE 4. Structure of a causal theory in which network position causally affects innovation

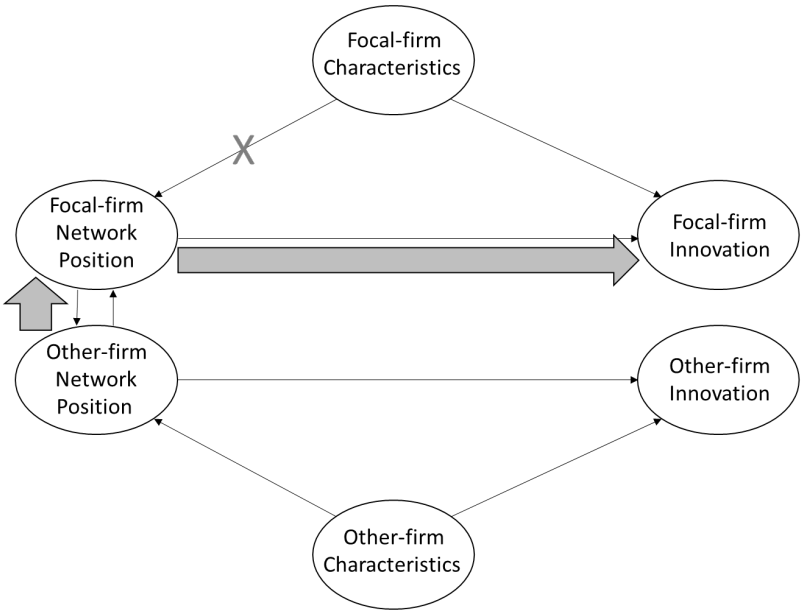

FIGURE 5. Relationships that would invalidate the causal test (best in color)

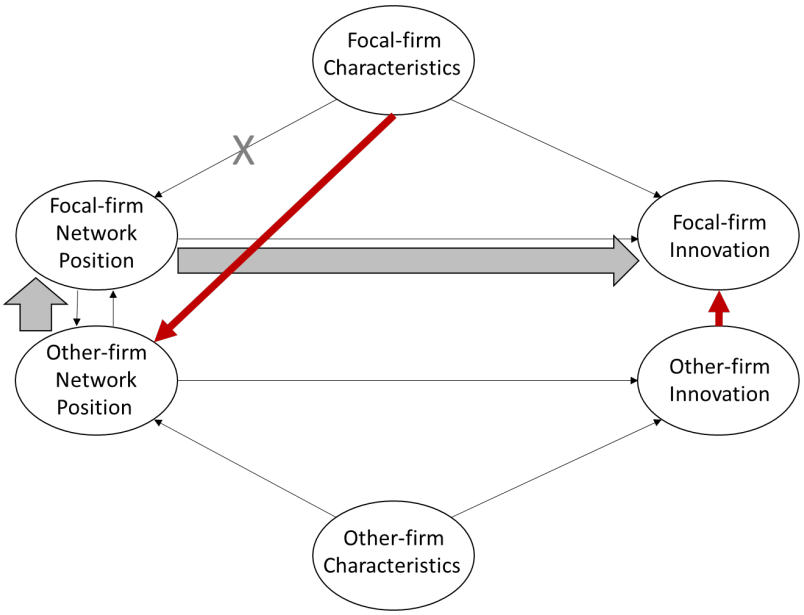

FIGURE 6a. Other-driven network change resulting from other firms' acquisitions and alliances (best in color)

The focal firm, Oxford Molecular, does not initiate any corporate activity during this period, but its network constraint increases because of the actions of its neighbors. Valentis acquires Polymasc Pharmaceuticals, becoming a direct neighbor of Oxford Molecular. Valentis's existing ties with firm #5 and #6 increase the network constraint of Oxford Molecular. On the other hand, Celltech creates an other-driven change to Oxford Molecular through its alliance formation. Celltech forms additional ties with Abbott and University of Washington. Overall, Oxford Molecular experiences an increase in network constraint.

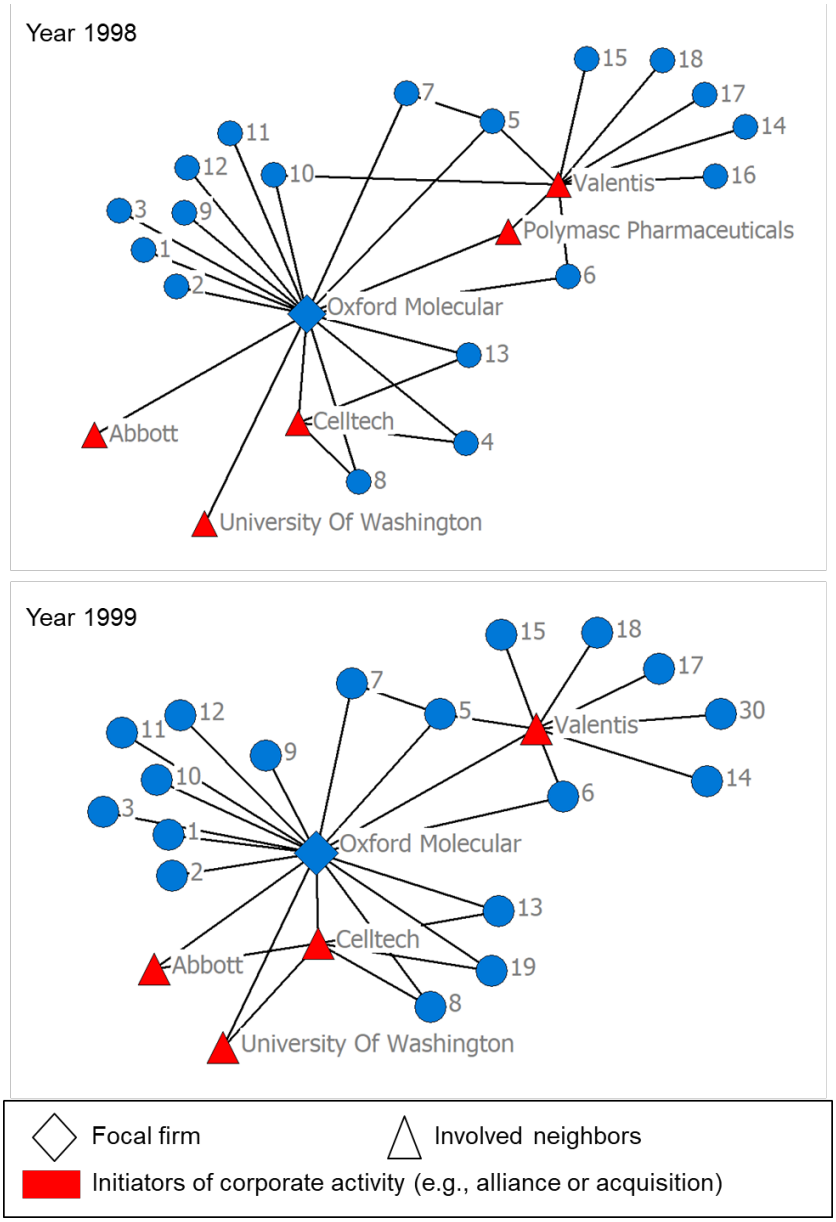

FIGURE 6b. Other-driven network change from other firms' alliances (best in color)

The forcal firm, Lifespan Biosciences, does not initiate any corporate activity during 2002-2004, but its neighbors do. In the first year, Sumitomo Pharma terminates its alliances with Merck and Bristol-Myers Squibb, decreasing the network constraint of Lifespan Biosciences. In the year after that, Merck forms a tie with Sanofi, increasing the focal firm's network constraint.

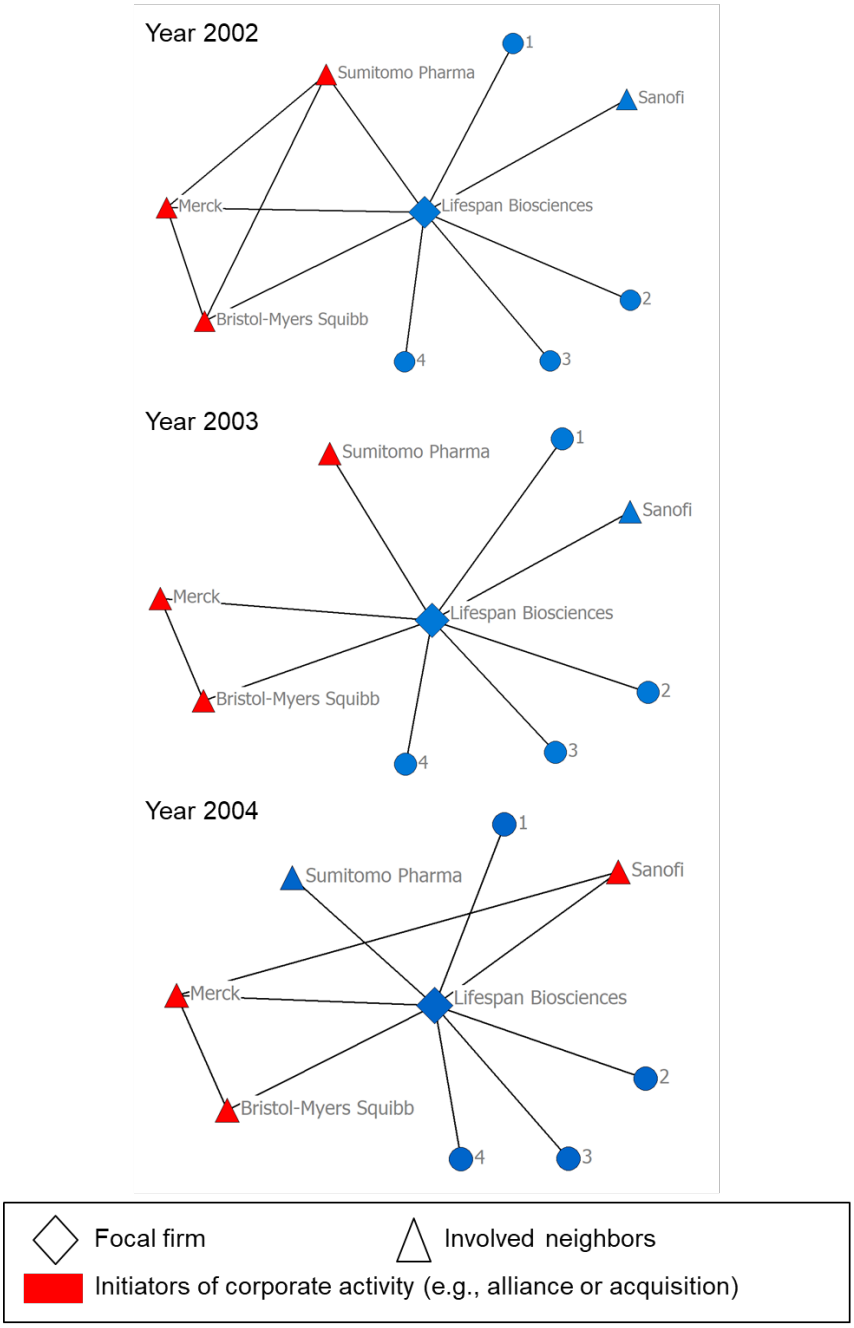

FIGURE 6c. Self-driven network change from the firm's own alliances and acquisitions (best in color)

The forcal firm, Watson Pharmaceuticals, initiates two acquisitions (Oclassen Pharmaceuticals and Royce Laboratories) and two alliances (with Rhone-Poulenc Rorer and Rorer). Before its corporate activities, Watson Pharmaceuticals is constrainted by firm #45. Afterwards, Watson Pharmaceuticals becomes a broker by spanning multiple structural holes, decreasing it network constaint as a result.

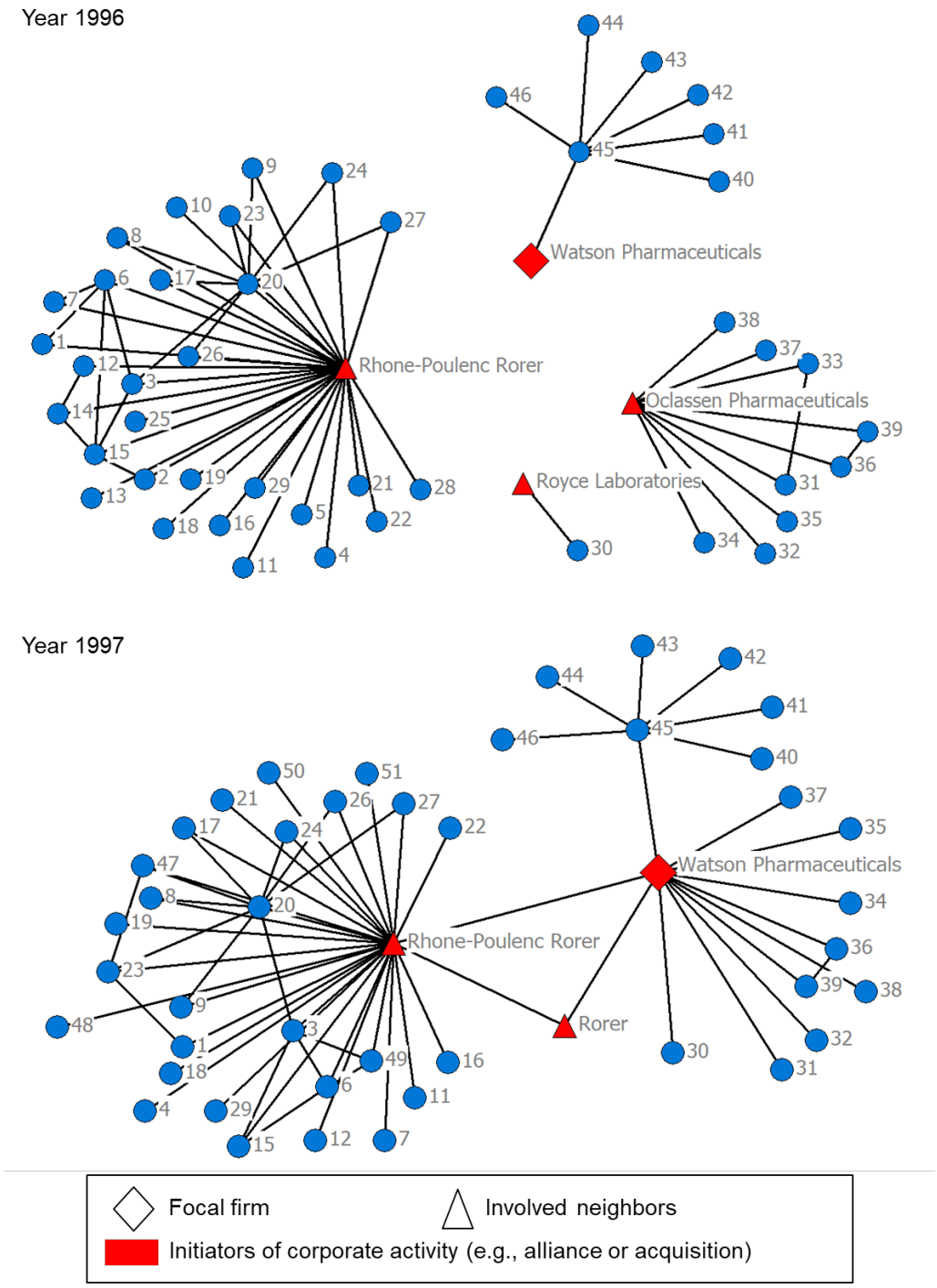

FIGURE 7. Distribution of actual changes in network constraint for *(a) self-driven-only change* and *(b) other-driven-only change*

(a) (b)

TABLE 1. Distribution of self-driven and other-driven network changes. We define self-driven change as changes driven by self-initiated alliances or acquisitions, and other-driven changes as changes driven by alter-initiated or alliances or acquisitions.

		Self-driven network change from self-initiated alliance or acquisition		
		No	Yes	Total
Other-driven change from third-party's alliance or acquisition	No	3,886 (No change)	699 (Self-driven-only change)	4,585
	Yes	14,012 (Other-driven-only change)	12,951 (Simultaneously self and other-driven change)	26,963
	Total	17,898	13,650	31,548

TABLE 2. Changes in network constraint across self-driven and other-driven conditions

Conditions	Observations	Mean	Standard deviation	Min	Max
Potential Change (zero + non-zero changes)					
Other-driven-only change	14,012	0.00075	0.034755	-0.625	0.625
Self-driven-only change	699	-0.45389	0.199167	-0.938	0.125
Simultaneously other and self-driven change	12,951	-0.04148	0.25980	-1	0.975
Actual Change (non-zero changes only)					
Other-driven-only change	282	0.03725	0.243	-0.625	0.625
Self-driven-only change	684	-0.46385	0.190	-0.938	0.125
Simultaneously other and self-driven change	11,715	-0.04586	0.273	-1	0.975

TABLE 3. Comparison of first-difference (model 1 and 3) and fixed effects (model 2 and 4) estimation of the effect of structural holes on patent counts and citation-weighted patent counts.

Dependent variables:	**Patent count**		**Citation-weighted count**	
	(1)	(2)	(3)	(4)
VARIABLES	First-Difference	Fixed effects	First-Difference	Fixed effects
	Δ Patent count	Patent count	Δ Citation-weighted count	Citation-weighted count
Δ Network constraint	-0.536***		-2.599***	
	(0.133)		(0.897)	
Network constraint		-2.629***		-6.866*
		(0.894)		(3.727)
Year fixed effects	Y	Y	Y	Y
Firm fixed effects	.	Y	.	Y
Observations	31,548	39,736	31,548	39,736
R-squared	0.004	0.006	0.005	0.022
Number of firms		7,784		7,784

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

TABLE 4. First-difference estimation of the effect Δ*network constraint* on Δpatent counts and Δcitation-weighted patent counts. Δ*network constraint* is partitioned into two mutually exclusive network change categories: other-driven-only and not-other-driven-only

Model #	(1)	(2)	(3)	(4)	(5)	(6)
Dependent Variable	ΔPatent Count			ΔCitation-Weighted Patent Count		
Δ Network constraint other-driven-only	0.486	0.478		-0.891	-0.930	
	(0.710)	(0.710)		(2.177)	(2.176)	
Δ Network constraint not-other-driven-only	-0.553***		-0.553***	-2.627***		-2.628***
	(0.135)		(0.135)	(0.911)		(0.911)
Year fixed effects	Y	Y	Y	Y	Y	Y
Observations	31,548	31,548	31,548	31,548	31,548	31,548
R-squared	0.004	0.004	0.004	0.005	0.005	0.005

*** p<0.01, ** p<0.05, * p<0.1 (robust standard errors in parentheses)

TABLE 5. First-difference estimation of the effect of Δ*network constraint* on Δpatent counts and Δcitation-weighted patent counts. Δ*network constraint* is partitioned into three mutually exclusive network change categories: other-driven-only, self-driven-only, simultaneously-other-driven-self-driven. Cases in which no network change was observed are dropped because they exhibit no variance in network change.

Model #	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Dependent Variable	ΔPatent Count				ΔCitation-Weighted Patent Count			
Δ Network constraint other-driven-only	0.516	0.505			-0.770	-0.840		
	(0.711)	(0.712)			(2.188)	(2.186)		
Δ Network constraint self-driven-only	-0.793***		-0.772***		-6.884**		-6.803**	
	(0.234)		(0.233)		(2.866)		(2.865)	
Δ Network constraint simultaneously-other-and-self-driven	-0.541***			-0.535***	-2.112**			-2.062**
	(0.153)			(0.153)	(0.940)			(0.940)
Year fixed effects	Y	Y	Y	Y	Y	Y	Y	Y
Observations	27,662	27,662	27,662	27,662	27,662	27,662	27,662	27,662
R-squared	0.004	0.004	0.004	0.004	0.006	0.005	0.006	0.005

*** p<0.01, ** p<0.05, * p<0.1 (robust standard errors in parentheses)

APPENDIX A. REPLICATION AND EXTENSION OF PRIOR APPROACHES

Replication

To properly ground our study, we first test the relationship between structural holes and innovation using the empirical specification in most previous papers. This consists of regressing innovation on network constraint, with firm and year fixed effects. Here the network is constructed by only relying on alliance formation and dissolution as the building blocks of the network. We do not yet account for the impact of acquisitions and divestitures on the network. This is the typical setup in prior research, although some studies remove acquired firms from the data altogether.

TABLE A1. Effect of structural holes on patent counts and citation-weighted patent counts. (a) Alliance only (model 1 and 2) assumes an alliance network comprised of only alliance deals. (b) M&A & Divestiture included (model 3 and 4) assumes an alliance network affected by M&As and divestitures on top of the alliance deals.

	(a) Alliance only		**(b) M&A & Divestiture included**	
	(1)	(2)	(3)	(4)
VARIABLES	Patent count	Citation-weighted	Patent count	Citation-weighted
Network constraint	-2.746***	-8.091***	-2.629***	-6.866*
	(0.554)	(3.004)	(0.894)	(3.727)
Year fixed effects	Y	Y	Y	Y
Firm fixed effects	Y	Y	Y	Y
Observations	42,242	42,242	39,736	39,736
R-squared	0.014	0.033	0.006	0.022
Number of firms	7,910	7,910	7,784	7,784

Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1

Models 1 and 2 in Table A1 present the results. Model 1 assess the effect of structural holes on patent counts. Model 2 assess the effect on citation-weighted patent counts. We find that a one-unit decline in network constraint is associated with an increase of about 2.7 patents and 8.1 citation-weighted patents. Both effects are statistically significant. These findings

support the canonical theory (Burt, 1992, 2004) and replicate prior empirical findings showing that structural holes increase patenting.

Incorporating Acquisitions and Divestitures into the Network

As a second step, we account for acquisitions and divestitures as events that modify the structure of the alliance network. This improves the precision with which we measure network structure. To reflect the impact of acquisitions on the alliance network, we generate a list of all the acquisitions made by firms in this industry during the relevant period as recorded in *SDC Platinum*. We identify 1,387 acquisition deals during 1991-2007. With that information, we "regenerate" the biotechnology alliance network in each period by reassigning the alliances of the target firm to the acquiring firm for the remaining life of each alliance during the post-acquisition period (see Hernandez and Shaver, 2019). The target firm thus disappears from the network, but its alliances get reassigned to the acquirer.[4] After regenerating the alliance network at the beginning of each year, we calculate network constraint for every firm in the sample. Acquisitions can modify the ego networks of the acquirer directly and, via alliance-network externalities, the ego networks of other firms in the network neighborhood of the acquirer or target. In any given year, many acquisitions reshape the structure of the industry network. Thus, we are not able to attribute the structural change experienced by a focal firm to a specific deal—we can only capture the aggregate impact of all deals affecting a focal firm on its network position in any given year. This also happens when firms establish or end multiple alliances in the same year—the change in structural position cannot be attributed to a single tie change.

[4] This procedure assumes that all alliances remain post-acquisition. It could be that an acquisition causes a subset of the alliances of a target firm to dissolve. Hernandez and Shaver (2019) find no evidence of post-acquisition loss of alliances in a smaller sample of deals from the same industry (life sciences) as in this study. Our anecdotal exploration of firms' press releases suggests that many times firms have strong incentives to keep target's alliances because they are a source of synergy in acquisitions (e.g. PR Newswire, 2004). If any loss of alliances caused by acquisitions were randomly distributed throughout the industry, this would create noise but not bias in empirical estimates. If such a loss were systematically related to certain types of deals, this could imply bias in our estimates. However, the lack of information on the fate of alliances post-acquisition makes it hard to know how many alliances are lost or what may predict that loss.

Divestitures represent a distinct form of network change, where one node splits into two nodes and a fraction of the parent firm's alliances may get reassigned to the newly created firm (see Hernandez and Menon, 2021). Reflecting divestitures in the alliance network is impossible in our case because we are not able to observe how the ties are re-allocated between the split nodes. For this reason, we drop firms that experienced a divestiture to lower the chance of measurement error. Doing so does not substantially modify the network because divestitures are rare in the life sciences industry during our time frame. Between 1995 and 2007, only 34 firms experienced divestitures (see table A2 below). If a firm divested more than once, we drop it in the year of its first divestiture. This results in a relatively small loss of 100 firm-year observations.

TABLE A2. Divestitures per year

Year divested	Freq.	Percent
1995	3	8.82
1996	4	11.76
1998	1	2.94
1999	2	5.88
2000	1	2.94
2002	3	8.82
2003	3	8.82
2004	6	17.65
2005	5	14.71
2006	3	8.82
2007	3	8.82
Total	34	

After accounting for acquisitions and divestitures, we estimate the relationship between structural holes and innovation using the typical fixed effects specification. Models 3 and 4 in Table A1 present the results, with model 3 showing the effect on patent counts and 4 showing the effect on citation-weighted counts. The results are similar to those in Models 1-2. A one-unit decline in network constraint is associated with an increase of about 2.6 patents and 6.9 citation-weighted patents. With more precise network measurement, the coefficients are slightly

smaller than before, but the effects remain statistically significant. The effect size on patenting reduces by about 4 percent and of citation-weighed patents by about 15 percent.

Reincorporating firms involved in divestitures

Dropping the 34 firms involved in divestitures does not materially alter our primary results reported in Table 4. Table A3, below, shows that we reach the same conclusions if we reincorporate those 34 firms into our analysis.

TABLE A3. First-difference estimation, including firms that engaged in divestitures

Model #	(1)	(2)	(3)	(4)	(5)	(6)
Dependent Variable	ΔPatent Count			ΔCitation-Weighted Patent Count		
Δ Network constraint other-driven-only	0.504	0.495		-0.847	-0.887	
	(0.708)	(0.708)		(2.175)	(2.175)	
Δ Network constraint not-other-driven-only	-0.550***		-0.550***	-2.616***		-2.616***
	(0.135)		(0.135)	(0.909)		(0.909)
Year fixed effects	Y	Y	Y	Y	Y	Y
Observations	31,648	31,648	31,648	31,648	31,648	31,648
R-squared	0.004	0.004	0.004	0.005	0.005	0.005

*** p<0.01, ** p<0.05, * p<0.1 (robust standard errors in parentheses)

APPENDIX B. ROBUSTNESS TESTS

TABLE B1. Robustness check with alternative dependent variable time windows

We used a five-year window for patent application in the main analysis. Below are the results for (a) four-year windows (T+1 to T+4) and (b) three-year windows (T+1 to T+3).

(a) Four-year DV windows

Model #	(1)	(2)	(3)	(4)	(5)	(6)
Dependent Variable	ΔPatent Count			ΔCitation-Weighted Patent Count		
Δ Network constraint other-driven-only	0.852	0.845		-1.560	-1.590	
	(0.741)	(0.741)		(2.705)	(2.704)	
Δ Network constraint not-other-driven-only	-0.447***		-0.447***	-2.033**		-2.034**
	(0.133)		(0.133)	(0.921)		(0.921)
Year fixed effects	Y	Y	Y	Y	Y	Y
Observations	31,548	31,548	31,548	31,548	31,548	31,548
R-squared	0.005	0.005	0.005	0.005	0.005	0.005

*** p<0.01, ** p<0.05, * p<0.1 (robust standard errors in parentheses)

(b) Three-year DV windows

Model #	(1)	(2)	(3)	(4)	(5)	(6)
Dependent Variable	ΔPatent Count			ΔCitation-Weighted Patent Count		
Δ Network constraint other-driven-only	0.423	0.417		-1.096	-1.134	
	(0.744)	(0.745)		(2.117)	(2.116)	
Δ Network constraint not-other-driven-only	-0.417***		-0.417***	-2.504***		-2.504***
	(0.118)		(0.118)	(0.761)		(0.761)
Year fixed effects	Y	Y	Y	Y	Y	Y
Observations	31,548	31,548	31,548	31,548	31,548	31,548
R-squared	0.005	0.005	0.005	0.004	0.004	0.004

*** p<0.01, ** p<0.05, * p<0.1 (robust standard errors in parentheses)

TABLE B2. First-difference estimation with control variables

Model #	(1)	(2)	(3)	(4)	(5)	(6)
Dependent Variable	ΔPatent Count			ΔCitation-Weighted Patent Count		
Δ Network constraint other-driven-only	0.372	0.364		-1.516	-1.563	
	(0.701)	(0.701)		(2.108)	(2.113)	
Δ Network constraint not-other-driven	-0.646***		-0.646***	-3.919***		-3.920***
	(0.162)		(0.162)	(1.024)		(1.024)
Δ Focal firm technological base	-0.0298	-0.0298	-0.0298	-0.170**	-0.170**	-0.170**
	(0.0196)	(0.0196)	(0.0196)	(0.0829)	(0.0830)	(0.0829)
Δ Partner technological base	-7.20e-05	-0.000136	-7.20e-05	-0.000336	-0.000728	-0.000336
	(0.000195)	(0.000193)	(0.000195)	(0.000823)	(0.000811)	(0.000822)
Δ Focal firm technological diversity	1.767***	1.818***	1.768***	3.138	3.444	3.135
	(0.540)	(0.542)	(0.540)	(2.191)	(2.203)	(2.191)
Δ Partner technological diversity	-0.184	0.103	-0.184	-1.398	0.348	-1.398
	(0.197)	(0.177)	(0.197)	(1.163)	(1.068)	(1.163)
Δ Technological similarity	0.217	0.268	0.217	0.153	0.459	0.154
	(0.275)	(0.274)	(0.275)	(1.213)	(1.207)	(1.213)
Δ Alliance experience	-0.319***	-0.319***	-0.319***	-1.364***	-1.366***	-1.364***
	(0.109)	(0.109)	(0.109)	(0.295)	(0.295)	(0.295)
Δ Alliance age	0.129**	0.110*	0.129**	1.451***	1.338***	1.451***
	(0.0616)	(0.0606)	(0.0616)	(0.279)	(0.275)	(0.279)
Δ Repeated alliances	-0.265	-0.316	-0.265	1.183	0.876	1.183
	(0.722)	(0.721)	(0.722)	(2.698)	(2.688)	(2.698)
Year fixed effects	Y	Y	Y	Y	Y	Y
Observations	31,548	31,548	31,548	31,548	31,548	31,548
R-squared	0.020	0.020	0.020	0.030	0.030	0.030

*** p<0.01, ** p<0.05, * p<0.1 (robust standard errors in parentheses)

We control for various firm and the alliance-level factors commonly included in studies of alliance networks and innovation. The focal firm's technological base, a proxy for absorptive capacity, is calculated as the cumulative number of patents up to the year of observation. Partner firms' technological base is calculated in the same way but takes the mean of the portfolio of alliance partners. To account for the scope of the firm's innovativeness, we calculate focal firm's technological diversity. We use the formula 1- $\sum_i (pat_i/N)^2$ (Blau, 1977), where pat_i is the number of patents filed in class i and N is the total number of patents filed by the firm (Vasudeva, Zaheer, and Hernandez, 2013; Kumar and Zaheer, 2019). A perfect heterogeneity will result in a value of 1 and a perfect concentration will result in a value of 0. Partners' technological diversity is calculated in the same way based on the patenting activity of all the partners in the portfolio. To capture the firm's track record of alliance participation, alliance experience is calculated as the total number of alliances a firm has initiated up to the year of observation (Anand and Khanna, 2000). We also control for the age of the firm's alliances using the average age of the alliances in the portfolio (Soda, Usai, and Zaheer, 2004). The ratio of repeated alliances was calculated as proportion of alliances that had been formed at least once before the focal year (Gulati 1995). Lastly, technological similarity among the focal firm and the alliance partners was calculated based on the cosine similarity of the patent classes filed. We construct a k-dimensional vector l containing the percentage of patents filed in each class by the focal firm i and the partner portfolio j, then we calculate the cosine similarity using the formula $cos_{ijt} = l_{it}l'_{jt}/\sqrt{(l_{it}l'_{it})(l_{jt}l'_{jt})}$ (Jaffe, 1986; Kumar and Zaheer, 2019).

APPENDIX C. TEMPORAL PATTERN OF NETWORK CHANGE CATEGORIES

In any given year firms can experience (1) no change, (2) other-driven-only change, (3) self-driven-only change, or (4) simultaneous other-driven and self-driven change. Here we explore temporal patterns to assess if there are any systematic sequences of changes across categories. The tables below report the number of consecutive years in which firms remain in one of the four conditions. The clear pattern is that firms switch conditions frequently. The most common pattern is that firms stay in a category for only one year. No firms remain in the same condition for the entire duration of our sample, except for 106 firms that experience *both endogenous and exogenous* changes every year.

No change

Consecutive years	Frequency	Percent	Cumulative
1	1,388	61.69	61.69
2	473	21.02	82.71
3	220	9.78	92.49
4	169	7.51	100
Total	2,250	100	

Self-driven -only

Consecutive years	Frequency	Percent	Cumulative
1	608	93.97	93.97
2	33	5.1	99.07
3	5	0.77	99.85
4	1	0.15	100
Total	647	100	

Other-driven -only

Consecutive years	Frequency	Percent	Cumulative
1	2,073	37.88	37.88
2	1,378	25.18	63.07
3	856	15.64	78.71
4	1,165	21.29	100
Total	5,472	100	

Simultaneously -other-driven -self-driven

Consecutive years	Frequency	Percent	Cumulative
1	1,137	36.47	36.47
2	608	19.5	55.97
3	354	11.35	67.32
4	303	9.72	77.04
5	116	3.72	80.76
6	100	3.21	83.96
7	92	2.95	86.91
8	81	2.6	89.51
9	80	2.57	92.08
10	63	2.02	94.1
11	44	1.41	95.51
12	34	1.09	96.6
13	106	3.4	100
Total	3,118	100	

The next two tables provide a highly detailed illustration of the temporal patterns. A “1” represents a firm being in a certain category in a given year and “*” represents a firm being absent. For example, in the first row of the *other-driven-only* condition, if the pattern is “**********1” it means that 547 firms are present in the last year of observation and not in any other year. The clear conclusion is that there is no systematic or stable sequence of changes from one category of network change to another.

Other-driven-only				**Self-driven-only**		
Frequency	Percent	Pattern		Frequency	Percent	Pattern
547	10.0	1		82	12.67	1
275	5.03	11		56	8.66	.....1.
215	3.93	111		49	7.57	.....1..
194	3.55	.....1.		49	7.57	.....1....
163	2.98	.....1111...		49	7.57	.....1.....
148	2.70	.....1111..		47	7.26	.....1...
147	2.69	1111		42	6.49	.....1.....
139	2.54	.....1111.		40	6.18	.....1.....
92	1.68	.....1111....		37	5.72	....1.....
92	1.68	....1111.....		36	5.56	.1.....
88	1.61	.....1..		32	4.95	..1.....
88	1.61	.....1...		28	4.33	1.....
87	1.59	1.....		20	3.09	...1.....
79	1.44	.....111..		6	0.93	11
77	1.41	.....1.....		5	0.77	.....1.1.
76	1.39	.....11.		4	0.62	.....11....
76	1.39	...1111.....		3	0.46	.....11.
69	1.26	.....1....		3	0.46	.....11..
64	1.17	.....111.		3	0.46	.....11.....
61	1.11	.....1.1		3	0.46	...1.1.....
61	1.11	.....111...		3	0.46	..11.....
61	1.11	.1111.....		2	0.31	.....1..1..
61	1.11	1111.....		2	0.31	.....11.....
60	1.10	..1111.....		2	0.31	.....1.1.....
59	1.08	.....11...		2	0.31	....111.....
57	1.04	.....11..		2	0.31	...11.....
55	1.01	111.....		2	0.31	.1.1.....
52	0.95	....1.....		2	0.31	1.....1...
52	0.95	.1.....		2	0.31	11.....
52	0.95	11.....		34	5.18	(other patterns)
51	0.93	..1.....			Total: 647	
49	0.90	.....1.....				
49	0.90	...1.....				
46	0.84	.....111....				
45	0.82	....111.....				
42	0.77	.....11....				
40	0.73	.....111.....				
39	0.71	.....1.1..				
38	0.69	.....1.11				
37	0.68	.....1.....				
35	0.64	..111.....				
1654	30.22	(other patterns)				
Total: 5,472						

No change				**Simultaneously-other-driven-self-driven**		
Frequency	Percent	Pattern		Frequency	Percent	Pattern
315	14.00	1		277	8.88	1
129	5.73	.....1.....		153	4.91	.....1.
113	5.02	.....1..		107	3.43	11
111	4.93	11		106	3.40	1111111111111
97	4.31	.....1.		68	2.18	.....1..
90	4.00	.....1....		59	1.89	111
77	3.42	1.....		55	1.76	.....11.
71	3.16	.....1...		42	1.35	1111
64	2.84	111		41	1.31	.....1...
62	2.76	.....1.....		36	1.15	11111
60	2.67	.....11....		31	0.99	111111
55	2.44	.....11...		31	0.99	1111111
54	2.40	...1.....		31	0.99	11111111
51	2.27	....1.....		29	0.93	.....1.1
51	2.27	..1.....		27	0.87	.....1..1
49	2.18	.1.....		27	0.87	.1.....
45	2.00	.....1.....		26	0.83	.....1.1.
40	1.78	.....1111..		26	0.83	.....1....
26	1.16	.....111...		25	0.80	111111111
25	1.11	.....11.		25	0.80	..11111111111
25	1.11	.....1111...		25	0.80	1.....
25	1.11	11.....		24	0.77	.....11..
22	0.98	.....11..		24	0.77	.....1...1..
20	0.89	.....1.1...		23	0.74	.....1.11
20	0.89	.....11.....		23	0.74	.....1...1
19	0.84	.....1.1.....		22	0.71	.....11.1
17	0.76	.....111..		22	0.71	...1111111111
16	0.71	.....1.1		22	0.71	..1.....
16	0.71	..111.....		20	0.64	.....1...1.
15	0.67	1111		20	0.64	.....1.....
15	0.67	.111.....		20	0.64	0.111111111
14	0.62	...111.....		19	0.61	.....1.....
13	0.58	.....1111.		19	0.61	...1.....
13	0.58	..11.....		18	0.58	1111.....
12	0.53	.....1.1..		18	0.58	111111111....
12	0.53	.....111....		17	0.55	.....1.111
12	0.53	..1111.....		17	0.55	.....1.....
12	0.53	111.....		16	0.51	.....111.1
12	0.53	1111.....		15	0.48	....1...1....
11	0.49	.....11.....		15	0.48	.11.....
11	0.49	....111.....		15	0.48	11.....
11	0.49	....1111.....		14	0.45	.....1.1..
11	0.49	...1111.....		14	0.45	1.11111E+11
10	0.44	.....111.		13	0.42	.....1..1..
10	0.44	.....111.		13	0.42	.....11..11
301	13.37	(other patterns)		1428	45.8	(other patterns)
Total: 2,250				Total: 3,118		

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