THE PROBLEM OF FRAGMENTATION AND THE PARADOX OF NETWORK INTERVENTION: EVIDENCE
FROM POLICY CHANGE IN HEALTH CARE DELIVERY¹

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The work of organizations increasingly requires bringing together participants with diverse knowledge, experience, and interests. Yet the differences that typically accompany such diversity make building robust relationships difficult. The result is a "problem of fragmentation," according to which collaborative networks exhibit dense, locally connected substructures, but sparse global connectivity. Motivated by these observations, research has begun examining network interventions—deliberate efforts to shape the structure of a network—as an approach for addressing fragmentation. To date, however, studies have produced many conflicting findings. We suggest clarity may come from considering how the inducements offered by network interventions interact with those afforded by the existing social structure of targeted groups (e.g., for connecting with distant contacts). We evaluate these ideas in a study of accountable care organizations (ACOs), using data on 100 million referrals among 1 million providers in 3,000 communities. Our findings suggest a network intervention paradox. The communities that stand to benefit most from network interventions like ACOs are those with greater fragmentation; yet fragmentation is associated with lower engagement and, conditional on engagement, less effectiveness. The result is increasing network inequality; communities with lower fragmentation reduce their fragmentation further, while those with lower fragmentation experience less change.

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INTRODUCTION

In 2001, the Institute of Medicine published a landmark report, *Crossing the Quality Chasm: A New Health System for the 21st Century*, that took aim at the poor quality of care delivered by the U.S. health care system. Coming in at more than 300 pages, the report addresses many potential contributors to the problem, but among those its authors single out as particularly potent is the fragmented nature of modern care delivery, brought about by insufficient and inefficient collaborative relationships among increasingly varied and diverse groups of health care providers. Since the publication of the report, care fragmentation has received considerable attention from policymakers and scholars alike (Bodenheimer, 2008; Kreindler et al., 2012; Hollingsworth et al., 2015; Agha, Frandsen, and Rebitzer, 2019), including organizational researchers (Hilligoss and Cohen, 2013; Kellogg, 2019; Kim et al., 2019).

While the situation in health care is well-known, it is hardly unique, and researchers have observed similar patterns in organizational settings ranging from academia and industrial R&D (Fleming, Mingo, and Chen, 2007; Rosenkopf and Schilling, 2007; Funk, 2014; Tett, 2015; Hidalgo, 2016) to schools and community groups (Moody, 2001; Ingram and Morris, 2007; Currarini, Jackson, and Pin, 2010; Benton, 2016). Indeed, this “problem of fragmentation” reflects an essential tension confronting many modern organizations; their work increasingly requires bringing together participants with diverse knowledge, experience, and interests, but the differences—for example, in specialization—that typically accompany such diversity make building robust relationships among those participants difficult (Hargadon and Sutton, 1997; DiBenigno and Kellogg, 2014; Kaplan, Milde, and Cowan, 2016). While the problem is complex and multifaceted, many studies suggest that the endogenous nature of social network dynamics play a decisive role. Social network structures create inducements—understood broadly here as behavioral stimuli—for those embedded within them to interact with some potential contacts over others. People tend to build relationships more readily with alters who are similar to themselves (homophily, e.g., on shared knowledge, experience, or interests; Kleinbaum, Stuart, and Tushman, 2013; Kossinets and Watts, 2009) or with whom they have previous contact

(reciprocity, repeated ties; Gulati and Gargiulo, 1999; Uzzi and Spiro, 2005) and to meet new contacts through referrals from people they already know (transitivity, assortativity; Davis, 1970; Dahlander and McFarland, 2013), thereby contributing to the formation of networks with dense, locally connected substructures, but sparse (or fragmented) global connectivity (Sorenson and Stuart, 2008; Tatarynowicz, Sytch, and Gulati, 2016). These sorts of network structures have been linked with a variety of potentially undesirable features, including challenging search (e.g., for information or prospective collaborators) and interaction across network substructures (Macy, Christie, and Luce, 1953; Burt and Ronchi, 1990; Ghoshal and Bartlett, 1990; Lazer and Freidman, 2007).

Motivated by these observations, a growing organizational literature has begun to examine network interventions, understood as deliberate efforts to shape or reshape the structure of a network, as an approach for addressing the problem of fragmentation (Chakravarti, Menon, and Winship, 2014; Boudreau et al., 2017; Davidson and Sanyal, 2017; Bapna and Funk, 2020; Hasan and Koning, 2020).² Studies in this area focus on examining whether and how policies, programs, or other stimuli enable (or push), either directly or indirectly, network members to create, disband, or reconfigure relationships (Kleinbaum, 2018; Srivastava, 2015), often in ways that are aimed at transforming the configuration of the network as a whole.³ While scholars have considered a number of interventions, many of them aim to break the endogenous reproduction of fragmented social networks by offering inducements

² Although Valente (2012) offers a valuable review of literature on network interventions, his view of them is generally different from that which we take here. Valente (2012: 49) focuses on networks as vehicles for delivering interventions aimed at changing other, non-network outcomes—i.e., “purposeful efforts to use social network data to generate social influence, accelerate behavior change, improve performance, and/or achieve desirable outcomes among individuals, communities, organizations, or populations.” Our focus is squarely on interventions where the outcome is the network (and its constituent relationships). Thus, our view is consistent with “alteration” type interventions in Valente’s (2012) framework but differs from the other strategies he describes (individuals, segmentation, and induction).

³ We recognize that not all network interventions are aimed at reducing fragmentation. Indeed, one may readily imagine interventions aimed at *increasing* fragmentation (e.g., for disrupting illicit networks) or any number of other network properties. We focus on fragmentation in this paper both because it is a fairly general network property (centering on the degree of connectedness of the network) and is consistent with the focus of much recent organizational research on network interventions.

that supplant those of the existing social structure, thereby making it more attractive to connect with potentially more distant prospective contacts (An, 2015; Vacca et al., 2015; Greco, Grimaldi, and Cricelli, 2017; Graf and Broekel, 2020). Although some interventions create fairly direct inducements for actors to build particular connections (e.g., by sending lists of contact recommendations), researchers and policymakers also commonly design interventions that use more indirect levers and that may also target other objectives (e.g., innovation) in addition to their network aims (La Rocca and Hoholm, 2017; Heß, Jaimovich, and Schündeln, 2020; Sciabolazza, Vacca, and McCarty, 2020; Boudreau and Ganguli, 2020). As an illustration, institutions like the University of Michigan, University of Minnesota, and National Institutes of Health have attempted to increase connectivity across disciplines by creating grant programs that require investigator teams to include researchers from multiple fields. Regardless of their precise form and context, such network interventions are often aimed at prompting targeted actors to engage in purposive networking, particularly the search for new prospective contacts for whom the inducements for connecting are greater than those afforded by the existing social structure.

Although research on network interventions has led to some progress, it has also produced many unanticipated and conflicting findings. Some studies report that providing inducements for actors to form ties is beneficial for stimulating new connections among members of targeted groups (Marmaros and Sacerdote, 2006; Rikakis, 2009; Conti and Doreian, 2010; Ding et al., 2010; Hottenrott and Lopes-Bento, 2014; Srivastava, 2015; Davidson and Sanyal, 2017; Bapna and Funk, 2020). Other studies, however, suggest the opposite, with programs having the effect of consolidating or supporting already well-connected groups of nodes, rather than those targeted by the program designers (Agrawal and Goldfarb, 2008; Carrell, Sacerdote, and West, 2013; Banerjee et al., 2018). In addition, existing studies are inconsistent with respect to whether network interventions produce transient or lasting effects (e.g., Defazio, Lockett, and Wright, 2009; Chakravarti, Menon, and Winship, 2014; Feys and Devos, 2015; Kleinbaum, 2018). These unanticipated and conflicting results are particularly

surprising because theories of network dynamics suggest that inducements are essential for shaping their patterns of tie formation. Thus, while our understanding of how networks change has grown increasingly mature, our ability to shape them remains surprisingly limited.

In this paper, we argue that these unanticipated and conflicting findings result in part from insufficient theorizing on how the inducements offered by various network interventions interact with those afforded by the existing social structure of targeted groups. As noted above, prior work on social networks observes that *search* and *interaction* across network substructures is likely more challenging in networks with greater fragmentation; indeed, it is precisely these undesirable properties that often motivate network intervention. Yet search and interaction are essential to shaping the responsiveness of targeted groups to network interventions. The upshot is that in groups that are characterized by greater network fragmentation (i.e., where there are fewer or longer direct or indirect pathways among group members), members will experience greater constraints in their efforts to form relationships with new contacts, even when provided with inducements to do so. Consequently, we propose that as fragmentation increases, responsiveness to inducements will be lower, both in the sense that members of targeted groups will be less likely to *engage* with (e.g., participate, adopt) the intervention and conditional on engaging, the interventions are less likely to be *effective* at overcoming fragmentation.

We evaluate these ideas in a study of accountable care organizations (ACOs), a new form of health care delivery created by the Affordable Care Act (Fisher et al., 2012; McWilliams et al., 2016). ACOs represent arguably the largest scale network intervention to date, certainly in the U.S. health care industry. ACOs aim to reduce fragmentation in health care by inducing providers to build more robust collaborative relationships across organizational and specialty boundaries. Within this context, we leverage data on 100 million referrals among more than 1 million providers in nearly 3,000 health care communities to examine variation in responsiveness to the Medicare Shared Savings ACO program, both in terms of engagement and effectiveness.

With respect to engagement, we focus on understanding whether fragmentation in local patient sharing networks is associated with regional ACO penetration. To help disentangle causality, we use the staggered rollout of the Medicaid expansion as an instrumental variable. We find a negative association between referral network fragmentation and accountable care organization penetration. Further, we confirm that the salience of financial inducements does not attenuate the challenges of fragmentation. With respect to effectiveness, we focus on understanding whether the relationship between ACO penetration and changes in network fragmentation are contingent upon levels of fragmentation in the preintervention period. To help disentangle causality, we leveraged features of the local political environment and ACO headquarters foundings in proximal regions as instrumental variables. Consistent with our predictions, we observe that the relationship between ACO penetration and changes in network fragmentation is significantly lower in communities with higher preintervention fragmentation.

Taken together, our findings suggest the existence of a network intervention paradox. On the one hand, the communities that stand to benefit most from network interventions like ACOs are those with greater fragmentation. On the other hand, greater fragmentation is associated with lower intervention engagement and, conditional on engagement, less effectiveness. Strikingly, these patterns of responsiveness suggest that network interventions may even contribute to network inequality, resulting in greater variance in fragmentation across communities, such that those with lower fragmentation reduce their fragmentation further, while those with higher fragmentation experience less change.

THEORY DEVELOPMENT

Interest in network interventions can be traced at least to the 1960s, when policymakers and social scientists began creating and evaluating programs for increasing racial integration in schools (Moody, 2001; Feld and Carter, 1998). These programs attempted to rectify connection “failures” by encouraging relationships among students of different races. Although they came in diverse designs—from bussing and pairing to redistricting—these interventions generally

sought to achieve their objectives by creating opportunities for contact between white and black students.

Research in this area provided early hints at the difficulty of designing effective network interventions and highlighted in particular the tendency for such interventions to produce unintended consequences. In his landmark report, *Equality of Educational Opportunity*, written in response to dictates of the 1964 Civil Rights Act, Coleman and colleagues (1966) observed that at some schools, for instance, bussing programs led to “strengthening the identity of the transferred children as foreign visitors,” thereby limiting the ability for integration.

Since the time of Coleman’s writing, the theoretical underpinnings of research on network interventions has witnessed major advances. Researchers are now increasingly aware of the endogenous nature of network dynamics (Rivera, Soderstrom, and Uzzi, 2010). Social structures tend to evolve according to path dependent processes, wherein the connections people form frequently result from various inducements afforded by established social structures. These “endogenous” inducements lead network members to build relationships with others who are similar to themselves (McPherson, Smith-Lovin, and Cook, 2001; Wimmer and Lewis, 2010), with whom they have previous contact (Dahlander and McFarland, 2013), and through referrals from people they already know (Davis, 1970; Granovetter, 1974), thereby contributing to fragmentation in larger, organizational network structures. Thus, the present structure of a social network shapes its future form.

Consistent with these observations, the design of many network interventions is motivated by the idea that the strategic provisioning of “exogenous” inducements may supplant those afforded by the existing social structure, breaking the endogenous reproduction of established networks by pushing network members to build new connections, including with those who are more distant from one another, and thereby reducing fragmentation (Marmaros and Sacerdote, 2006; Kim and Bak, 20178; Bernstein and Turban, 2018; Funk, Sytch, and Nahm, 2020). Prior research on network interventions has considered a fairly wide range of such exogenous inducements, ranging from recommending contacts and creating artificial social

groups to rewarding shared performance on collaborative tasks (Carrell, Sacerdote, and West, 2013; Vacca et al., 2015; Matous and Todo, 2017; Matous and Wang, 2019; Bapna and Funk, 2020; Hasan and Koning, 2020). Recent interest in using inducements for as a tool for reshaping networks has not however been limited to the confines of organizational network research. The point of commonality among these diverse forms of network intervention is that they all aim in various ways to overcome the inducements offered by established social structures.

Yet despite these theoretical advances in the understanding of network dynamics, findings from the literature on network interventions often continue to echo the frustrations of Coleman and other early investigators, with studies frequently reporting conflicting findings and unintended consequences. To unpack our discussion in the introduction, consider findings from research on proximity-based network interventions. The literature on network dynamics has shown that whether two people will form a relationship is a function of the distance between them; the lower the physical separation, the greater the probability of a connection being created (Allen, 1977; Catalini, 2018). Using these findings, many network interventions aim to facilitate relationship formation by increasing the spatial proximity between targeted actors (e.g., via reducing physical barriers or creating shared spaces). Yet while some studies of proximity-based interventions have shown positive results (e.g., Bapna and Funk, 2020), the findings are far from uniform; other studies have shown either no effect or even effects that are opposite of those intended (e.g., Ingram and Morris, 2007; Bernstein and Turban, 2018). A different approach to the design of network interventions uses findings from research on network dynamics on the importance of shared identity for relationship formation.

Interventions in this stream aim to facilitate connections by placing targeted participants in small groups, and sometimes entail a joint problem-solving component (e.g., Marmaros and Sacerdote, 2006; Conti and Doreian, 2010; Chakravarti, Menon, and Winship, 2014; Argyres, Rios, and Silverman, 2020). Paralleling findings on proximity-based network interventions, some work in this stream reports positive effects; but again, the results are far from uniform, and other work reports limited or transitory benefits. As final illustration, an emerging stream

in the network intervention literature considers whether various incentives may be helpful for facilitating tie formation. As with other approaches, work in this area is grounded in theory and research on network dynamics (Watts, 2001; Jackson and Rogers, 2005; Galeotti, Goyal, and Kamphorst, 2006). Yet here too, there have been inconsistent findings, with some work suggesting promising results (Defazio, Lockett, and Wright, 2008), and other studies pointing to more limited effects.

While network interventions are of course not the first social programs to show unintended consequences, the accumulation of inconsistent findings represents an increasingly thorny problem both for researchers interested in the dynamics of social networks and for policymakers interested in transforming them. Why then have theoretical advances in the understanding of social network dynamics not led to more robust insights on effective interventions?

Network fragmentation and network intervention

We suggest that part of the answer to this question is that existing work has tended to overlook how the inducements offered by network interventions interact with those of the existing social structure. Network interventions aim to reduce fragmentation by providing inducements for targeted actors to build new connections. Thus, search for and interaction with new prospective contacts are essential to shaping the responsiveness of targeted groups to network interventions. Yet, search and interaction across network substructures are likely more challenging in networks with greater fragmentation; indeed, as noted previously, it is precisely these undesirable properties that often motivate network intervention.

First, the search for new contacts is likely more difficult in networks with greater fragmentation. As our previous discussions on the endogenous nature of network dynamics notes, people tend to meet and learn about new contacts through their existing contacts. In networks with greater fragmentation, there are on average more intermediaries (i.e., fewer direct paths) between pairs of randomly selected group members (Watts and Strogatz, 1998), and therefore the ability for actors to navigate through their existing and neighboring contacts

to identify new potential connections decreases. Visually, a useful image is that of a random walker, one who begins a search by approaching an existing contact for information about prospective partners, who then refers the random walker to one of his or her own existing contacts, and so on, manifesting “a network of possible wanderings” (Newell and Simon, 1972: 82). As fragmentation increases, the network offers fewer possible wanderings to our random walker, meaning he or she must wander longer and perhaps more deliberately to reach portions of the network similar to someone who was traversing a less fragmented structure. While exogenous inducements (like network interventions) may prompt individuals to use alternative search strategies (other than referrals from existing contacts, or perhaps contacts of contacts) these efforts are also likely to be more difficult as fragmentation increases because information—including about other network members—tends to circulate more slowly in networks with longer average path lengths (Lazer and Freidman, 2007; Fang, Lee, and Schilling, 2010; Shore, Bernstein, and Lazer, 2015). Thus, targeted individuals are less likely to have even ambient awareness of more distant prospective contacts in the network.

Second, interaction with prospective contacts is likely to be more difficult in groups with higher fragmentation. The slower information flows that are characteristic of networks with greater fragmentation means that it is likely more challenging to learn not only of the identities of new prospective contacts, but also about their character and reliability (Granovetter, 1985; Coleman, 1988; Uzzi, 1997). For similar reasons, members of networks with greater fragmentation are also less able to rely on the assurances of reputational sanctions for opportunistic behavior that are characteristic of more highly connected networks. The upshot here is that in their search for prospective contacts, individuals in relatively more fragmented networks will need to devote more time and energy to evaluating prospective partners; these demands are in turn likely to further constrain the number of potential contacts considered by individuals in such networks. For many network interventions, the higher interaction difficulties that are likely present in more fragmented networks are likely especially problematic

when, as is often true, the inducements encourage joint investments and rewarding joint performance among new collaborators, thereby exacerbating the risks of poor partner choice.

The implication of both factors is that in response to exogenous inducements, individuals in relatively more fragmented networks are likely to cast a smaller net as they look for new prospective contacts. Part of this is due to the greater challenge of search; it is simply more difficult to find prospective partners. Part is due to the challenges of interaction, which require greater investments of time for evaluating potential new contacts. More broadly, these considerations underscore our core argument that the inducements afforded by the existing social structures of target groups are likely critical to understanding the effects of network interventions. The same properties that make fragmented networks ripe for intervention also work in ways that likely limit the ability for interventions to bring about change.

Hypotheses

Building on the theoretical development above, we propose two hypotheses (see Figure 1 for a summary).

First, we suggest that as fragmentation increases, targeted network members will be relatively less responsive to network interventions in that they will have lower engagement (i.e., participation or adoption). Interventions based on inducements work by prompting actors to engage in search for potential contacts. When fragmentation increases, however, the challenges of search are also likely to increase. While the exogenous inducements afforded by a network intervention (e.g., grants given to interdisciplinary teams) may push targeted actors to search longer and harder for prospective new contacts than they may otherwise, they do not directly alter the length or existence of paths along which those searches must take place. Put differently, such inducements do not themselves alter the opportunities for contact. To illustrate this point, imagine we offer inducements for people in two communities, separated by water, and across which there is no bridge, to build connections with one another. Unless our intervention also includes building a bridge (thereby increasing opportunities for contact) it is

unlikely that members of either community will respond to our inducements. Thus, as fragmentation increases, the likelihood of targeted actors engaging with the intervention is likely to be lower. These considerations lead us to our first hypothesis.

Hypothesis 1: As network fragmentation increases, engagement (i.e., participation or adoption) with network interventions will decrease.

Second, we propose that as fragmentation increases, responsiveness to network interventions will also be lower in terms of effectiveness. Put differently, conditional on engagement (e.g., participation, adoption), network interventions are likely to be less transformative in groups with greater fragmentation. Our previous discussions suggest that in response to inducements for creating new connections, actors in groups with relatively greater network fragmentation will form systematically different *kinds* of new relationships from those in groups with relatively lesser fragmentation. Specifically, due to greater challenges of search and interaction, actors in relatively more fragmented networks will be more likely to form connections that are more local in nature, being closer to their existing network neighborhoods or groups. A direct corollary of this argument is that as fragmentation increases, the relationships that actors form in response to exogenous inducements from network interventions will likely be more local and/or to map onto existing patterns of relationships within the network. But the success of network interventions for reducing fragmentation is contingent on actors responding by building connections with distant potential contacts, which allow for smaller path distances between network members. To the extent that actors in relatively more fragmented groups respond by building more local connections, such interventions are likely comparatively less effective at reducing fragmentation, and may conceivably even reinforce the existing fragmentation (by increasingly local connectedness). These considerations lead us to propose a second hypothesis.

Hypothesis 2: As network fragmentation increases, the effectiveness of network interventions for reducing fragmentation will decrease.

Before turning to our data and methods, we note that whether we find support for Hypothesis 1 or Hypothesis 2—or both—would suggest important differences for the understanding and

design of network interventions for reducing fragmentation. Support for Hypothesis 1 but not Hypothesis 2 would suggest that treatment effects are similar across levels of fragmentation; therefore, to the extent that different outcomes are observed between groups with higher or lower fragmentation, these differences may largely be attributed to differential engagement. Thus, under this scenario, we may anticipate that greater equality in outcomes (i.e., similar levels of reduced fragmentation across groups) could be obtained by tailoring the exogenous inducements offered to the levels of fragmentation observed in the group (e.g., giving greater rewards to groups with relatively greater fragmentation).

However, support for both Hypothesis 1 and 2 (or support for Hypothesis 2 alone) would suggest a different mechanism. In particular, evidence of heterogeneous treatment effects may indicate that tailoring inducements across levels of fragmentation would not be enough for achieving greater equality in outcomes across levels. Specifically, support for Hypothesis 2 would suggest the possibility that across levels of fragmentation, the same network intervention may stimulate systematically different behaviors among targeted actors. Thus, similar levels of engagement across groups would not lead to similar outcomes, because the treatment effects are different depending on the levels of fragmentation.

DATA AND METHODS

Research setting

Our research setting is health care delivery in the U.S. Although the U.S. spends far more on health care than comparable countries—17.8% of GDP in 2016—it ranks poorly on many fundamental outcomes, including life expectancy and infant mortality (Papanicolas, Woskie, and Jha, 2018). The challenges facing the U.S. health care system are many and complex, but as noted in our introduction, observers often point to the fragmented nature of care delivery as particularly problematic. Care fragmentation occurs when the delivery of services to patients is spread across multiple, disconnected providers (i.e., providers who do not have established relationships). In settings with greater care fragmentation, communication among providers is

often more difficult and less frequent. Consequently, care fragmentation may contribute to both higher spending (e.g., via greater duplication of services) and lower quality (e.g., via more difficult information sharing) (Romano, Segal, and Pollack, 2015; Funk et al., 2018). At the regional level, care fragmentation is associated with greater utilization (i.e., spending) particularly on services like hospitalizations and emergency department visits, which are often associated with worse outcomes (Agha, Frandsen, and Rebitzer, 2019).

Policymakers have devoted increasing attention to developing programs for reducing care fragmentation in the U.S. health care system. To date, accountable care organizations represent arguably the largest and most ambitious of these initiatives. Accountable care organizations (ACOs) are a new form of health care delivery created by the 2010 Affordable Care Act (Fisher et al., 2012; McWilliams et al., 2016). In essence, an ACO is a group of providers (e.g., physicians, hospitals) who voluntarily come together to form a new organization and an accompanying contract with a particular payer (e.g., Medicare, Medicaid, or a private insurer). According to these contracts, the ACO takes responsibility for the overall health and spending of a defined beneficiary population. Beneficiaries are assigned to an ACO based on where they receive their primary care services. Therefore, the service area of an ACO will typically correspond to a defined geographic community. In return for taking responsibility for their assigned beneficiary population, ACOs have the opportunity to be rewarded with shared savings; ACOs that are able to reduce spending (below a defined benchmark) while also maintaining or improving care quality are given a portion of what would have otherwise gone to the payer.

ACOs as network interventions. Our view of ACOs as network interventions is based on multiple considerations. Perhaps most critically, the promise of shared savings is seen by policymakers as creating a powerful incentive for reducing fragmentation. In describing its Shared Savings ACO program, for example CMS writes, “the Shared Savings Program is designed to provide an incentive for individuals and groups of providers and suppliers to come

together to provide coordinated care for Medicare fee-for-service beneficiaries.”⁴ Elsewhere, Health and Human Services Secretary Kathleen Sebelius and CMS Administrator Donald M. Berwick further underscore the importance of robust connections among providers for ACO success, noting “we do not see how ACO participants and ACO providers/suppliers could achieve the statutory goals of the Shared Savings Program without showing a degree of interaction and interdependence in their joint provision of medical services such that they provide high quality and efficient service delivery.” (Federal Register, 2011) Indeed, reducing fragmentation is so central to the aims of ACOs that in the Medicare Shared Savings Program (discussed below) it is included as one of four fundamental evaluative benchmarks according to which the distribution of shared savings to participants is determined (the other three being patient/care giver experience, preventative health, and attention to at-risk populations).

Beyond the policy domain, our view of ACOs as network interventions is consistent with the perceptions of participants and advocates of ACOs. Echoing official remarks on ACO program design, many suggest that the bulk of savings and quality improvements from ACOs is likely to be attributable to the inducements they create for reducing fragmentation among providers. As Lewis and Fisher (2012: 295) explain in an editorial of the *Journal of the American Medical Association*,

At their core, [ACOs] are social innovations. The explicit intent is to encourage collaboration among physicians, other clinicians, and hospitals to improve care not only for individual patients (by coordinating the care delivered by multiple clinicians across space and time) but also for populations (by developing shared clinical pathways that ensure the best possible care for patients with specific health problems).

Similarly, in a case study of several early ACOs, Kreindler and colleagues (2012: 459) observe, “the ACO appears to be a peculiarly social intervention, using social levers—shared accountability and collective incentives—to encourage new relationships among groups.”

Consistent with comments like these, the rollout of ACOs has witnessed the emergence of a new class of consulting firms that promise to help ACOs build the kind of “high quality referral

⁴ <https://www.cms.gov/Center/Provider-Type/All-Fee-For-Service-Providers/Downloads/FFS-FAQs.pdf>

networks” that will contribute to lower cost, higher quality, and ultimately shared savings. Reports from these firms describe how the ACO program creates “a strong incentive for the ACO to build a referral network,” (Duncan, 2014) warning “in a risk-based world, ACOs...that aren’t taking a proactive approach to referral management put themselves at risk,” (Hefner, 2019) and suggesting that “creating a high-value referral network in the ACO appears to be a crucial step toward maximizing shared savings.” (Williams, 2016)

Finally, features of the ACOs themselves add support to our view of ACOs as network interventions. Perhaps most clearly, many ACOs in the MSSP include the term “network” or related terms (e.g., “alliance”, “connection”, “coordination”) in their names. As an illustration, Appendix 1 plots the prevalence of “network” and related word stems in the names of ACOs. Roughly 10.5% of ACOs in our sample explicitly include the word “network” in their names (e.g., “Arizona Care Network,” “Cape Cod Health Network ACO,” “Northern Michigan Health Network”), while many others use closely related terms. These patterns are particularly compelling given that CMS provides no restrictions or guidelines to ACOs on naming conventions.

Mapping regional care networks with referral pattern data

To allow for systematic evaluation of the dynamics of networks in our setting, we need to first determine the most relevant unit of analysis. We focus on 3,436 communities across the U.S. known as Hospital Service Areas (HSAs). Defined by the Dartmouth Institute for Health Policy and Clinical Practice, HSAs are collections of zip codes that constitute geographic markets for hospital care. In the U.S., health care markets are highly localized; most individuals receive health care services from providers who practice nearby. Thus, geographic regions are substantively meaningful health care communities. HSAs are defined by the Dartmouth Institute empirically, based on actual patterns of care delivery observed in national Medicare claims data. Therefore, HSAs are attractive for our purposes relative to other common geographic boundaries (e.g., states, counties). HSAs have been used extensively in prior

research, including in work on care fragmentation (Agha, Frandsen, and Rebitzer, 2019) and ACO formation (Lewis et al., 2013).

After settling on relevant communities, we need to identify relevant vertexes and edges—the two fundamental building blocks of networks. We considered health care providers (e.g., physicians, nurse practitioners, physician’s assistants) to be the most relevant vertexes. This decision was motivated both by prior work on the importance of provider networks in health care and on the significant role of providers in the implementation of accountable care organizations (Landon et al., 2012; Kim et al., 2019). For each HSA, in each year of our study (described in greater detail below), we identified relevant providers using historical snapshots of the National Plan and Provider Enumeration System (NPPES)⁵, created and made publicly available by the National Bureau of Economic Research. NPPES is a national registry of health care providers in the U.S. Participation in the NPPES data is mandatory for all providers who bill Medicare. In addition to a unique identifier for each provider, known as a national provider identifier (NPI), NPPES reports other general information, including on providers’ practice addresses. Using these addresses, we assigned NPIs to HSAs⁶.

We considered patient sharing relationships to be the relevant edges. This decision was motivated both by the substantive importance of patient sharing relationships in health care delivery and by prior research, which has studied these relationships extensively (Everson et al., 2017; Pollack et al., 2013; Iwashyna et al., 2009). Patient sharing relationships form as a result of patients concurrently receiving care from multiple providers, typically due to referrals from one provider to another. When a patient (or beneficiary, in the Medicare context) needs specialized treatment or has complex needs, more than one provider is typically involved in the provision of services. Primary care providers (PCPs), for example, who are usually the first point of

⁵ <https://www.nber.org/research/data/national-plan-and-provider-enumeration-system-nppes>

⁶ To minimize heterogeneity and ensure that our networks included only relevant providers, we excluded organizational and deactivated NPIs. Consulting with physician and health services researcher colleagues, we also excluded providers (e.g., radiologists) who are not directly involved with patient care. Appendix 2 lists the provider taxonomy codes that were included in the networks.

contact for patients in the health care system, refer patients to specialists when more in-depth treatment is needed. Providing care for common patients requires communication regarding health history, treatment plans, and other topics. Consequently, shared patients correspond closely to actual provider relationships, and surveys of physicians find that shared patients are strongly predictive of self-reported professional ties (Barnett et al., 2011).

For each HSA, for each year of our study, we map patient sharing relationships using data from CMS that were made publicly available following a Freedom of Information Act Request⁷. These data are formatted as edge lists that record pairs of providers (i.e., NPI × NPI entries) who billed for services provided to the same Medicare beneficiary within a defined time window (e.g., 30, 60, 90, or 180 days), on an annual basis. For our study, we use the relatively conservative 60-day window, based on the likelihood that providers are more likely to communicate about patients that they see within relatively more proximate time periods.

Due to privacy regulations, only dyads that share more than 11 patients in a year are reported in the data. Although this may be considered a limitation, previous research suggests that dyads consisting of fewer shared patients are unlikely to represent meaningful relationships (Barnett et al., 2011). Thus, by limiting our focus to providers who share a minimum of 11 patients within a 60-day window, our network measures should be less biased by false positives—i.e., inclusion of relationships where none exist. Because our focus is on regional networks, we only include patient sharing relationships among providers who both practice within a focal HSA. However, our models include controls for referral relationships that span regional boundaries.

Finally, we focus our study on the period between 2011 and 2015. The first MSSP ACOs started operation in 2012. Therefore, by beginning our study in 2011, we are able to observe the structure of regional patient sharing networks in the period immediately before the program

⁷ <https://www.cms.gov/Regulations-and-Guidance/Legislation/FOIA/Referral-Data-FAQs>

began. Our decision to end the study in 2015 was driven primarily by the availability of data on patient sharing relationships from CMS.

Measures

Hypothesis 1 predicted that as network fragmentation increases, engagement (i.e., participation or adoption) with network interventions would decrease. Hypothesis 2 predicted that as network fragmentation increases, the effectiveness of network interventions for reducing fragmentation would decrease. Thus, engagement is our dependent variable in Hypothesis 1 and explanatory variable in Hypothesis 2.

To evaluate engagement, we identified, for each HSA, in each (post 2011) year of our study, the membership of local providers in MSSP ACOs, which we obtained through the MSSP ACO Research Identifiable File (RIF). This RIF lists the NPIs of participating providers for all MSSP ACOs. With these data in hand, we defined *Providers joining ACOs* as the number of providers who joined an MSSP ACO, for each region $\times$ year observation. To help facilitate interpretation, we log-transformed the resulting values. Higher values on this measure indicate greater responsiveness in terms of engagement.

[Insert Figure 2 about here]

Figure 2 shows the rate of providers joining ACOs in each HSA in 2015. The figure suggests that although there is variation in adoption across regions, adoption patterns are not obviously correlated with basic regional traits (e.g., West Coast, East Coast; urban, rural).

To evaluate effectiveness, we consider the overall (i.e., whole network) structure of patient sharing networks among providers within HSAs. Specifically, following Borgatti (2006), we define *Fragmentation* as

$$D_f = 1 - \left(\frac{2 \sum_{i>j} \frac{1}{d_{ij}}}{n(n-1)} \right)$$

where n is the number of providers in the network and d_{ij} is the path distance (i.e., number of intermediaries) between providers i and j . We measure *Fragmentation* for each HSA in each year.

To facilitate interpretation, we multiply D_f by 100. Consequently, the measure ranges from 0 to 100, with larger values indicating greater fragmentation.

In Figure 3, we map the level of fragmentation in each HSA in 2011. As in Figure 2, regions vary in the level of fragmentation; however, we do not observe any readily discernable concentration of high, low, or moderate D_f across broad regions of the U.S. (e.g., North, South, East, West).

After computing D_f , we conducted several supplemental analyses to evaluate its validity as a measure of fragmentation. To the extent that D_f is a valid measure, we should observe that it correlates with variables related to fragmentation (e.g., its antecedents and consequences) as identified by prior work. Appendix 3 presents the results of these analyses. Consistent with our expectations, we generally find positive and significant associations between our measure of fragmentation various indicators, including healthcare costs (e.g., reimbursement percentile) and inverse and statistically significant associations between fragmentation and health care quality (e.g., readmission rate), including after adjustments for other network properties. Appendix 3 also reports on the convergent validity of our measure. We observe that D_f has reasonable associations with other network properties that are likely indicative of fragmentation (e.g., density, clustering). Overall, these evaluations help to support our measure of fragmentation as a meaningful one in our research setting.

[Insert Figure 3 about here]

Control variables. Our models include several control variables.

We control for the (logged) number of *Providers* in the HSA at time t , which is likely related to both fragmentation (e.g., path distances are likely to be larger in larger networks) and the responsiveness of providers to the ACO program (e.g., more local providers means that more providers are available to join ACOs).

In addition, as mentioned previously, when constructing regional patient sharing networks, we limited our attention to ties among providers within HSAs. We believe this

decision is justified given the conceptual focus of our study and the local nature of health care delivery. However, not all patient sharing relationships occur among collocated providers. Higher levels of patient sharing with providers beyond regional boundaries may be indicative of HSAs that map less closely onto meaningful health care communities, which may in turn influence both the level of responsiveness to the MSSP ACO program and the degree of fragmentation observed in the local patient sharing network. We therefore control for *External referral relationships (weighted)*, which we define as the number of patient-sharing ties that span the HSA boundary, weighted by the total number of patient-sharing relationships involving local providers.

We also control for several market characteristics.

First, we adjust our models for the local quality of care, which may be associated with both local patient-sharing network structure and responsiveness to the MSSP ACO program. For each HSA, in each year of the study window, we control for the *Readmission rate*, defined as the percentage of Medicare beneficiaries within the region who required an emergency room visit within 30 days of discharge following a medical admission to the hospital. This rate, which was obtained from the Dartmouth Institute, is adjusted for several risk factors, including age, sex and race.

Second, we control for healthcare capacity by including a measure of the *Primary care coverage rate*. This measure, also from the Dartmouth Institute, is defined as the percentage of patients visiting a primary care clinician within 14 days of discharge following medical admission; like *Readmission rate*, values of the measure are risk-adjusted. In addition, we include measures that capture the presence of hospitals within the focal region. For each HSA, in each year, we define two dummy variables that indicate the presence of *Multiple hospitals* or *No hospitals*. Given the important anchoring role of hospitals in local health care communities, their presence is likely to be associated with both responsiveness to the MSSP ACO program and local patient sharing network structure. The data underpinning these measures were obtained from the Dartmouth Institute.

Analysis

Statistical models. The dependent variables used to evaluate both hypotheses—*Providers joining ACOs (log)* (Hypothesis 1) and *Fragmentation* (Hypothesis 2)—are continuous, and our data are formatted as a panel, with HSA $\times$ year observations. We therefore estimate linear regressions with HSA and year fixed effects. Specifically, to evaluate Hypothesis 1, we estimate models of the form

$$y_{it+1} = X_{it} \cdot \beta + c_i + \mu_t + \epsilon_{it}, i = 1, \dots, N, t = 2011, 2012, 2013, 2014$$

where i indexes HSAs, t indexes years, y is the dependent variable, X is a matrix of time and HSA varying independent and control variables, c is a fixed effect for each MSA, μ is a fixed effect for each study year, and ϵ is an error term. These models assume that *Providers joining ACOs (log)* is a function of four things:

- μ_t encompasses factors at each time period t that affect all HSAs equally
- c_i captures all time *invariant* heterogeneity among HSAs
- x_{it} records time-varying factors that differ for each HSA at each year t
- ϵ_{it} encompasses the idiosyncratic, time-varying error term for each HSA $\times$ year observation

We measure the dependent variable—*Providers joining ACOs (log)*—at time $t+1$ to help account for administrative lags in the process of ACO formation; if factors present in a particular HSA at time t increase (or decrease) the likelihood of providers joining ACOs, the associated changes in ACO membership will only be observed the following year.

To evaluate Hypothesis 2, we use a split sample approach. Specifically, we divide HSAs into two groups (with a “low” group for those below the median and a “high” group for those above) based on their observed levels of *Fragmentation* in 2011, the year immediately preceding the start of the MSSP ACO program. We then estimate separate regression equations for each group, of the form

$$y_{it} = x_{it} \cdot \beta + c_i + \mu_t + \epsilon_{it}, i = 1, \dots, N, t = 2012, 2013, 2014, 2015$$

In addition to the split sample approach, the main difference between our modeling strategy for Hypothesis 1 and 2 is that in the latter, we measure the outcome (*Fragmentation*) at

time t . Our rationale for this lag structure is that ACO membership is most likely to influence patient sharing patterns in the focal year, rather than adjacent years.

Instrumental variables. When evaluating our hypotheses, a potential concern is that the relationship between fragmentation and responsiveness to the MSSP ACO program is endogenous. In particular, our estimates may be subject to a simultaneity problem. Hypothesis 1 predicts that engagement will be a function of the existing network structure within a community. Yet, if MSSP ACOs are effective as network interventions, as predicted by Hypothesis 2, then fragmentation is likely to decrease as engagement with the MSSP ACO program increases. Consequently, over time, the causal arrow may run in both directions.

To help address this issue, we estimate two-stage least-squares instrumental variables models in our assessments of both hypotheses. Beginning with Hypothesis 1, we use the Affordable Care Act's Medicaid expansion as an instrument for *Fragmentation* (Courtemanche et al., 2017; Kaestner et al., 2017). In contrast to Medicare, which provides insurance primarily to the elderly, Medicaid focuses on insuring low-income individuals (i.e., individuals with incomes around or below the federal poverty line). Thus, the Medicare and Medicaid programs (generally) target distinctive patient populations. The Affordable Care Act included provisions for expanding Medicaid (making the program available to more people) at the state level. The law intended the expansion to take place in 2014 but allowed states to expand early. On Jun 28, 2012, the Supreme Court ordered that states could not be forced to expand Medicaid. Therefore, the Medicaid rollout is highly staggered, both across states and time.

Our rationale for using the Medicaid expansion as an instrument for fragmentation is as follows. Local expansion of the Medicaid program should lead to a sudden increase in the population of individuals with insurance coverage. As this population increases, so too should visits to local health care providers. Consequently, these providers are likely to encounter an influx of new patients, which may both constrain their capacity and increase their likelihood of encountering patients with previously unseen conditions and needs. Together, these factors are likely to lead providers to refer patients to a broader range of other providers than normal. In

addition, newly insured patients, coming from a distinctive patient population, also seem likely to seek care from sets of providers that deviate from those previously established. Thus, we anticipate that the Medicaid expansion will lead to new patient sharing relationships among local providers. Although these dynamics will be driven by patients in the Medicaid population, we anticipate that there will be spillover, such that there will be corresponding changes to patient sharing networks serving Medicare beneficiaries. In other words, exogenous variation of Medicaid expansion should influence fragmentation levels among providers for Medicare patients. Yet, because our sample focuses on MSSP (i.e., Medicare) ACOs, the Medicaid expansion is unlikely to be directly related to local engagement with MSSP ACOs.

Notwithstanding these attractive features of the Medicaid expansion, a challenge with this approach is that the Medicaid expansion is a state level variable, while our units of observation are smaller regions (HSAs). In wealthy areas, the number of newly insured individuals resulting from the expansion is likely to be small, and to have little association with local patient sharing network structure. To address this issue, we collected data from the American Community Survey, for each study year, for all zip code tabulation areas in the U.S., on the number of individuals living below the federal poverty line, as a proxy for Medicaid eligibility. After collecting these data, we instrument for *Fragmentation* using a time varying indicator for state-level Medicaid expansions, which we interact with our measure of individuals living below the poverty line.

Turning to Hypothesis 2, we use two instruments for *Providers in ACOs (log)*. Our first instrument is the local political environment. Recall that Hypothesis 2 predicts that as network fragmentation increases, the effectiveness of network interventions for reducing fragmentation will decrease; thus, the causal arrow for Hypothesis 2 effectively runs in the opposite direction of Hypothesis 1. Prior work suggests that state governments play an important role in the local implementation of ACOs, particularly as providers of shared resources like databases and other information infrastructures (e.g., multipayer databases, health information exchange) (Purington et al., 2011). State governments are also influential for convening stakeholders to

discuss the possibility of ACO formation. Thus, engagement with the MSSP ACO program will likely be higher in regions with more supportive local political environments. At the same time, we have little reason to believe that the state political environment (particularly party control, which, as we discuss next, we use as our proxy) will be associated with referral patterns.

To measure the supportiveness of the local political environment for ACO formation, we defined measures of the political party tendency (Republican or Democrat) of each HSA, in each study year. Historically, Republicans have been less receptive to government-led health care reforms, particularly the Affordable Care Act, the repeal of which has been a party priority since the law was first passed (Pacheco, Haselswerdt, and Michener, 2020). We therefore propose that the higher the proportion of Republicans political leaders in a region, the less likely that the region will present a favorable environment for ACO formation. When defining our political party tendency measures, we focus on state congresses and governors, which are more likely to be involved with local politics than national-level leaders (e.g., members of the U.S. Senate).

We collected data on state-level congressional party composition from National Conference of State Legislatures.⁸ These data provide biannual information on whether the two legislative chambers of state government (house and senate) are dominated by Democrats, Republican, or neither party. The raw variable takes value of 0, 1, and 0.5, respectively. For state governors, we hand-collected data on the political party of the governor in each state in each year. The raw variable takes value of 0 if the governor is Democratic and 1 if Republican. Because both the state congress and governor are at state level, we match HSAs to states and apply state level raw variables to each HSA. Subsequently, we calculate the proportion of Republicans for each study year in each HSA, separately for state-level congresses and governors.⁹ Thus, our political instrument consists of two separate variables (*State congress*

⁸ <http://www.ncsl.org/research/about-state-legislatures/partisan-composition.aspx>

⁹ The raw state-level variables can take values of 0, 1 (congressional and gubernatorial control) or 0.5 (congressional control only). However, because some HSAs span multiple states (and we calculate

political tendency and *Governor political tendency*), each of which ranges from 0 to 1. Further, to help account for potential differences for unified and divided (legislative and executive branch) party control, we include the interaction of these two measures.

Our second instrument for *Providers in ACOs (log)* is counts of ACO headquarters foundings in proximal regions. We anticipate that the establishment of an ACO headquarters is likely to draw attention to the opportunities afforded by the ACO program (e.g., via news reports, word of mouth), thereby marginally increasing the likelihood of local providers joining an ACO. Consistent with this idea, more than half of physicians with ACO membership had their practice in a different region than their ACO headquarters (see Appendix 4). Our measurement of this instrument focuses on geographically proximal regions (not the focal region) in the period before the focal observation (i.e., at $t-1$).¹⁰ We have little *a priori* reason to anticipate that administrative offices (rather than practice locations) for ACOs outside the focal region and time period would directly influence patient sharing patterns among providers within the focal region.

RESULTS

Table 1 shows descriptive statistics and bivariate correlations for variables in our analyses. Due to differences in the lag structure used to evaluate Hypothesis 1 and 2, the included years (and therefore sample sizes) vary across models. For descriptive statistics and correlations, we report the years 2012 to 2014, which are included in all models.

[Insert Table 1 about here]

Hypothesis 1 is evaluated in Table 2. Model 1 includes only control variables. Model 2 includes only our main independent variable for Hypothesis 1, *Fragmentation*. Model 3 is our fully specified model and includes both the independent and control variables. In Models 2 and 3, the coefficient for *Fragmentation* is negative and significant ($p < 0.01$), consistent with

proportions for each HSA accordingly), both the state-level congress and governor variables sometimes assume a broader range of values between 0 and 1.

¹⁰ Specifically, we use ACO headquarters foundings in the 3rd, 4th, and 5th closest HSAs. Our results, however, are robust to alternative definitions of proximal regions.

Hypothesis 1; as fragmentation increases, fewer providers join ACOs. Specifically, holding all other variables at their means, a one standard deviation increase in *Fragmentation* above the mean is associated with an 8.78% decrease in *Providers joining ACOs (log)* (based on Model 3). Model 4 presents the results of our two-stage least-squares instrumental variables regression. Results using the Medicaid expansion as an instrument for *Fragmentation* are consistent with our baseline model (i.e., Model 3).¹¹

[Insert Tables 2 and 3 about here]

We evaluate Hypothesis 2 in Table 3. Model 5 includes only control variables. Model 6 includes only our main independent variables, *Fragmentation_{t-1}* and *Providers in ACOs (log)*. Model 7 includes both the independent variables and control variables. In both Models 6 and 7, *Fragmentation_{t-1}* is positive and significant ($p < 0.01$) and *Providers in ACOs (log)* is negative and significant ($p < 0.01$). Fragmentation in the previous year is positively associated with fragmentation in the next year and (consistent with the results in Table 2,) engagement with the MSSP ACO program is associated with lower fragmentation.

Models 8-11 evaluate Hypothesis 2 using the split sample approach discussed above, where splits are made based on the level of fragmentation before the introduction of ACO program (i.e., in 2011). Models 10 and 11 are similar to Models 8 and 9, but the former use instrumental variables.¹² Models 8 and 10 include HSAs that had fragmentation below the

¹¹ We report first stage regressions for M4 in Appendix 5-1. The interaction between the Medicaid expansion and poverty is negatively associated with fragmentation ($p < 0.05$). This is consistent with our prediction that by prompting new referrals, the Medicaid expansion would be associated with lower fragmentation in areas with higher numbers of individuals below the poverty line. Appendix 5-2 reports tests of validity for the Medicaid expansion instrument in models; we find little evidence of underidentification, weak identification, or overidentification.

¹² We report first-stage regressions for M10 and M11 in Appendix 5-1. Consistent with our predictions, in both first-stage models, the interaction between state-level congressional and governor political party tendency is negative (*Providers in ACOs [log]*) ($p < 0.05$ and $p < 0.1$, respectively), suggesting that a more Republican political environment depresses ACO engagement. Proximal ACO headquarters are positively associated with ACO engagement (*Providers in ACOs*) ($p < 0.01$) in both first-stage models (M10 and M11). The coefficients for the instruments in the first stage of M10 are not significantly different from the corresponding coefficients in the first stage of M11, consistent with our expectation that the local political environment is not directly relevant for fragmentation. Appendix 5-2 reports tests of validity for our local political environment and ACO headquarters instruments; we do not find evidence of underidentification, weak identification, or overidentification.

median in 2011; Models 9 and 11 include HSAs that had fragmentation above the median. The coefficient on *Providers in ACOs (log)* is negative in all four models and significant ($p < 0.05$) in all but Model 11 (i.e., the instrumental variable model for the high fragmentation group). The coefficient on *Providers in ACOs (log)* is larger in magnitude in Model 8 than in Model 9 and the difference is significant ($p < 0.01$). Similarly, the coefficient on *Providers in ACOs (log)* is larger in magnitude in Model 10 than in Model 11 and the difference is significant ($p < 0.1$). Based on Model 8, holding all other variables at their means, a one standard deviation increase in *Providers in ACOs (log)* is associated with a 1.04% decrease in *Fragmentation* in HSAs with low 2011 fragmentation; based on Model 9, by contrast, one standard deviation increase in *Providers in ACOs (log)* is associated with a much smaller 0.19% decrease in *Fragmentation* for HSAs with high 2011 fragmentation. To complement these results, we turn to Model 12, which uses the full sample, and adds the interaction between $Fragmentation_{t-1}$ and *Providers in ACOs (log)*. We observe that the coefficient on the interaction term ($Fragmentation_{t-1} \times Providers\ in\ ACOs\ (log)$) is negative and significant ($p < 0.01$). Taken collectively, these results provide support for Hypothesis 2; as network fragmentation increases, network interventions like the MSSP ACO program are less effective for reducing fragmentation.

Robustness checks

We conducted several additional analyses to evaluate the robustness of our findings to alternative model specifications.¹³

[Insert Table 4 about here]

Models 13 and 14 replicate Model 4 but using different measures of engagement with the MSSP ACO program. The outcome in Model 13 counts—for each HSA, in each study year—the number of new ACO headquarters offices formed at time $t+1$. In contrast to our main models, we do not find a statistically significant relationship with *Fragmentation*. Although open

¹³ For models reported in this subsection, where the Medicaid expansion was used as instrument (Model 13-16 and 18), we report tests of validity in Appendix 2. Overall, we do not find evidence of underidentification, weak identification, or overidentification.

to interpretation, this finding is may be seen as a useful placebo check. There is no mandate requiring that ACO headquarters be located near the practice locations of their participants; given the complex accounting capabilities they require, ACO headquarters locations may be driven more by the local availability of these capabilities than practice locations. Consistent with our conjecture, we find that more than half of physicians who are ACO members are located in a different region than headquarters of ACO that they belong to. We report these statistics in Appendix 4.

In Model 14, the outcome variable is the (logged) number of unique MSSP ACOs joined by providers at time $t+1$ in the focal region (recall that our main dependent variable captures the number of providers joining ACOs, regardless of the specific ACOs that they join). Consistent with our main results, we find that there is a negative association between *Fragmentation* and the number of unique ACOs joined by local providers ($p < 0.01$).

Model 15 replicates our main model (Model 4) but using an alternative proxy for fragmentation. Our primary proxy was based on the average path distances between providers in the network. The alternative measure in Model 15, by contrast, records, for each HSA in each study year, the fraction of providers in the main component of the provider network, where “main component” refers to the largest set of nodes that are directly or indirectly (i.e., through intermediaries) connected. Defined in this way, higher values on this measure indicate *less* fragmentation. In support of our findings, we find that the coefficient for *Main component* is positive and statistically significant ($p < 0.01$).

As discussed previously, our models are potentially subject to simultaneity bias; the causal arrow between *Fragmentation* and responsiveness to the MSSP ACO program may, at least over time, run in both directions. Previously, we addressed this concern using an instrumental variable approach. Models 16 and 17 consider two additional approaches for ruling out simultaneity. Model 16 is identical to Model 4 (our main model) but introduces a control for the cumulative number of local providers who had joined the MSSP ACO program up to and including the focal year. Our rationale for this approach is that prior responsiveness

to the MSSP ACO program should be associated with both future responsiveness and with contemporary levels of *Fragmentation*. Findings using this approach are consistent with our main models. In Model 17, we proxy for fragmentation using only the value of *Fragmentation* observed in 2011, the year immediately preceding the start of the MSSP ACO program. This approach rules out simultaneity because the MSSP ACO program cannot influence levels of *Fragmentation* prior to its existence. Once again, the findings are similar to our main models.

Finally, Model 18 reports results from a placebo test where, for each HSA in each study year, we replace the observed value of *Fragmentation* with one selected randomly from another HSA in the same state and same year as the focal HSA. If local network structure is indeed driving the observed association between *Fragmentation* and responsiveness to the MSSP ACO program, then we should expect to see no predictive value of *Fragmentation* observed in geographically proximate regions. In support of our results, this is exactly what we find.

Supplementary analysis

We conducted several analyses to explore the mechanisms underpinning the observed relationships between fragmentation and responsiveness to the MSSP ACO program (and vice versa).

[Insert Table 5 about here]

Engagement. Models 19 tests whether the negative association we observe between fragmentation and engagement with (i.e., adoption of) the MSSP ACO program may be attenuated in places where the potential payoff of the MSSP ACO program for providers is greater. Specifically, given the designs of the MSSP ACO program, the potential for shared savings may be greater in places where, after adjusting for risk factors, spending on health care is higher. Here, our intuition is that in communities with higher Medicare spending, the exogenous inducements afforded by the MSSP ACO program would be more attractive because there is more room for costs savings. Using data from Dartmouth Institute, we evaluated this possibility by interacting *Fragmentation* with a risk adjusted measure of Medicare spending per beneficiary, reimbursement percentile, a standard measure in the health services literature.

Although the coefficient on the interaction term is in the expected direction, it is not statistically significant. This result suggests that financial inducements alone may be insufficient to overcome the challenges of fragmentation for network intervention engagement.

Models 20 and 21 offer an additional assessment of our proposed mechanism. In our theory development, we suggested that in networks with greater fragmentation, engagement would likely be lower because such networks create fewer opportunities for search and interaction among network members. If this explanation holds, then in communities where there are alternative (i.e., nonnetwork) mechanisms to facilitate search and interaction among local actors, the negative association we observe between fragmentation and engagement with the MSSP ACO program should attenuate. To evaluate our prediction on search, Model 20 interacts *Fragmentation* with *Hospitals (2011)* (i.e., the number of Hospitals in the focal HSA [using Dartmouth Institute data])¹⁴. Hospitals are important shared social contexts in health care communities, bringing together providers and administrators from diverse (even competing) organizations, thereby potentially creating greater local awareness of prospective contacts (and facilitating search). Because our models include HSA fixed effects, we are only able to identify the coefficient for *Fragmentation* $\times$ *Hospitals (2011)* (i.e., the main effect of *Hospitals [2011]* is not identified). Model 21 interacts *Fragmentation* with *Social capital*, as proxied by counts of local establishments in industries that have been previously identified as relevant to community social capital (Putnam, 2000). We used County Business Patterns data from the U.S. Census to define community-level social capital for each HSA in each study year¹⁵. Our logic for this moderator is that greater social capital in the community is likely to make interaction among providers and health care administrators (due to the greater likelihood of common social

¹⁴ Within HSAs, the number of hospitals is highly stable during our study window. Therefore, to facilitate identification, we measure this variable as of 2011.

¹⁵ Following Rupasingha, Goetz, and Freshwater (2006), we included religious organizations, bowling centers, fitness and recreational sports centers, golf courses and country clubs, sports teams and clubs, civic and social associations, business associations, political organizations, professional organizations, and labor organization. The sum of the number of establishments listed here is divided by the population in each HSA.

contacts or affiliations) easier, but relative to the presence of local hospitals, is less likely to facilitate search. Consistent with our theoretical expectations, we find positive and significant coefficients for both interaction terms ($p < 0.01$ and $p < 0.05$, respectively).

Effectiveness. We supplement our findings on effectiveness (i.e., Hypothesis 2) by exploring whether there are qualitative differences in the nature of responsiveness among communities. In our theory development, we suggested that identifying and interacting with potential partners who are relatively more distant in a network from a focal actor would be more challenging than identifying and interacting with those who are relatively more proximate. Consequently, the resulting *patterns* of responsiveness to the MSSP ACO program may also differ according to levels of fragmentation within a community. Understanding these patterns is important because the effectiveness of a particular network intervention likely depends not just whether on targeted actors respond, but also *how* they respond (e.g., the kinds of new connections they make). Models 22, 23, and 24 evaluate patterns of responsiveness by considering several alternative dependent variables.¹⁶

First, the outcome in Model 22 is ACO diversity, which we measure, for each HSA in each time period, as the Herfindahl-Hirschman index (HHI) (subtracted from 1 so that the variable represents diversity), over the distribution ACOs, of providers joining ACOs. HSAs in which all providers who join an ACO join the same ACO will have lower values on this measure than those in which providers join different ACOs in equal proportions. In Model 22, we find that the coefficient for *Fragmentation* is positive and significant ($p < 0.01$), which suggests that increasing fragmentation is associated with increases in ACO diversity.

Second, Model 23 examines the relationship between fragmentation and cross-ACO edges, where the latter is defined, for each HSA in each time period, as the number of patient-sharing relationships that span ACO boundaries, weighted by the total number of patient-

¹⁶ We report tests of instrument validity of Medicaid expansion in these models in Appendix 2. We do not find evidence of underidentification, weak identification, or overidentification.

sharing relationships. The result of this analysis is a negative and statistically significant ($p < 0.01$) association between fragmentation and cross-ACO edges. Put differently, increases in fragmentation tend to be associated with fewer patient-sharing relationships that span ACO boundaries.

Third, Model 24 is similar to Model 23, but instead of cross-ACO edges, considers the relationship between fragmentation and ACO-nonACO edges, which we define, for each HSA in each study year, as the number of patient-sharing relationships between providers who are and who are not members of MSSP ACOs, again weighted by the total number of patient-sharing relationships. Here, we also find a negative and statistically significant relationship ($p < 0.05$), which suggests that increases in fragmentation are associated with fewer connections between ACO and nonACO providers.

Finally, Model 25 examines the relationship between fragmentation and the correspondence between the existing care network and regional provider ACO memberships. Specifically, we define a new dependent variable, measured (at time $t+1$), as the normalized mutual information between provider ACO memberships and provider network community memberships (based on a clustering of the referral network using the Louvain community detection algorithm). Our intuition is that as fragmentation increases, the associated challenges of search and interaction may lead ACOs to form in ways that map closer onto existing network structures. The positive and significant ($p < 0.05$) coefficient on *Fragmentation* suggests that ACOs formed in communities with higher fragmentation map more closely to the existing care network than communities with lower fragmentation.

In summary, while our main analyses show that the degree of engagement with the MSSP ACO program varies with fragmentation, these supplemental results suggest so too does the *nature* of engagement. As fragmentation goes up, the diversity of ACO memberships and the correspondence between ACO memberships and the existing network structure also increases, while referrals that span ACO boundaries (either cross ACO, or from ACO to nonACO) decrease. Together, these findings suggest the striking possibility of an association

between fragmentation—the target of network intervention—and patterns of responsiveness that further exacerbate fragmentation.

Paradox. For our final analysis, we present visualizations of the process that we argue, over time, leads to a network intervention paradox; in groups that stand to benefit most from network intervention, members face the greatest constraints in their efforts to form relationships with new contacts, even when provided with inducements to do so. This analysis helps to demonstrate the implications of our finding of support for Hypotheses 1 (engagement) and Hypothesis 2 (effectiveness). The left panel of Figure 4 plots raw (i.e., unadjusted) values of *Providers joining ACOs (log)* (i.e., the main dependent variable under Hypothesis 1) annually, from 2011 to 2015, separately for HSAs with high/low (above/below the median) levels of normalized pre-intervention fragmentation¹⁷. Consistent with Hypothesis 1 and with the results of our models, we see dramatic differences in engagement with the MSSP ACO program between HSAs that had high/low fragmentation before the start of the program; providers in HSAs with higher pre-fragmentation are less likely to join MSSP ACOs, a gap that appears to *widen* over time.

The center panel of Figure 4 plots annual changes in *Fragmentation*, again from 2011 to 2015, and with separate lines for HSAs with high/low (above/below the median) levels of normalized pre-intervention fragmentation. As may be anticipated given our predictions in Hypothesis 2 and the findings from our previous inferential models, we observe dramatic differences across regions with high/low pre-intervention fragmentation. The patterns suggest that following the introduction of the MSSP ACO programs, HSAs with higher fragmentation became more fragmented, while those with lower fragmentation became less fragmented, a pattern reminiscent of the Matthew Effect in science.

¹⁷ We normalized *Fragmentation* by dividing by the number of nodes in the community. *Fragmentation* is highly correlated with the number of nodes (0.53). We then used normalized fragmentation level in 2011 as normalized pre-intervention fragmentation to divide HSAs into two groups. The same measure is used for both the left panel and the center panel of Figure 4.

Finally, the right panel of Figure 4 complements the left and center panels by plotting predicted values of a regression model (see Model 26 of Table 5) that examines differences in the relationship between *Providers in ACOs (log)* and *Fragmentation*, over time, thereby allowing us to visualize differences in the effectiveness of MSSP ACOs as network interventions, while also adjusting for confounding covariates¹⁸. Although not directly comparable to the left and center panels of Figure 4 (due to different axes and groups), the overall conclusion is consistent. We observe widening gaps in *Fragmentation* over time, such that *Fragmentation* does not change much for HSAs with comparatively higher ACO adoption, but increases for those in which ACO adoption is comparatively lower. Further, the apparent gap between HSAs with high versus low adoption observed in 2012 (the first year of the MSSP ACO program) is consistent with the left panel of Figure 4 (and with Hypothesis 1), such that regions with higher *Fragmentation* are less likely to engage with the MSSP ACO program.

[Insert Figure 4 about here]

DISCUSSION

Over the past few decades, scholars have increasingly come to understand the dynamics of social networks as an endogenous process. People tend build relationships more easily with people who are similar to themselves or with whom they have previous contact and to meet new contacts through their existing contacts. Thus, the current structure and composition of a network shapes its future form. Many of these endogenous mechanisms work in ways that lead the resulting networks to have dense, locally connected substructures, but sparse global connectivity. While the implications of any particular network structure are often dependent upon the context, networks with greater fragmentation have been linked with a number of undesirable properties. This is especially true for many modern, knowledge intensive organizations, which increasingly depend on bringing together participants with diverse knowledge, experience, and interests, and for whom the endogenous nature of network

¹⁸ To evaluate these trends, we include *Year* as continuous variable (and its interaction with *Providers in ACOs [log]*). The results are consistent when we include dummy variables for each year (and their associated interactions).

dynamics often creates a “problem of fragmentation.” Against this backdrop, researchers have devoted growing attention to network interventions, understood as deliberate efforts to shape a network according to some criterion of the intervention designer, often through the provision of exogenous inducements that aim to overcome those endogenously afforded by the existing social structure. Yet research in this area has also produced many conflicting findings, and to date, our collective knowledge of the natural dynamics of social networks has far outpaced our ability to shape them.

In this article, we attempted to overcome these limitations of prior work by more systematically theorizing and empirically evaluating how the inducements offered by various network interventions interact with those afforded by the social structure of targeted groups. We observed that as fragmentation increases, the search for and interaction with prospective contacts—particularly those who are more distant in the network—is more challenging. While various inducements offered by the designers of network interventions may encourage network members look for new contacts, these inducements must contend with those of the existing social structure. In networks with greater fragmentation, these inducements are more likely to push against those of the intervention, with the latter probably encouraging network members to form new, more distant connections, and the former pushing them to look in more locally and familiar places. These considerations point to potentially important heterogeneity in the effects network interventions, such that as fragmentation increases, responsiveness is likely to be lower, with members of targeted groups being less likely to engage with (e.g., participate, adopt) the intervention and, conditional on engagement, for the intervention to be effective for reducing fragmentation.

We evaluated these ideas empirically in the context of ACOs, a new form of health care delivery created by the Affordable Care Act. ACOs generally, and the MSSP specifically (our empirical focus), represent arguably the largest scale network intervention implemented to date. The results of our empirical analyses offered support for our theoretical development.

First, we found that increasing fragmentation in local patient sharing networks was associated with decreases in engagement with the MSSP ACO program, i.e., the number of local providers joining MSSP ACOs. Our results on social capital, the presence (or absence) of local hospitals, and health care spending suggest that this relationship may have more to do with the challenges of searching for and interacting with potential partners than with the financial inducements for connecting.

Second, our results suggest that in communities where preintervention fragmentation levels were relatively higher, engagement with the MSSP ACO program was less effective for reducing subsequent fragmentation. Our supplemental analyses suggest that this relationship is likely attributable at least in part to how levels of fragmentation in prior periods influence patterns of engagement. In particular, we found that as fragmentation increases, local providers tended to join a more diverse set of ACOs and tended to have fewer connections with other providers outside their ACO. We also found that increasing fragmentation was associated with providers joining ACOs in ways that mapped more closely onto the existing network structures of their communities. Thus, as fragmentation increases, providers appear to engage with the MSSP ACO program in ways that may not only make reducing fragmentation more challenging, but that may even work to further exacerbate fragmentation within the community.

Taken together, these findings suggest the existence of a network intervention paradox. Consistent with the objectives of many network interventions, a central aim of the MSSP ACO program has been to decrease health care spending and increase quality by transforming health care delivery networks in ways that reduce fragmentation. Our finding that fragmentation is inversely associated with engagement and effectiveness suggests, however, that the program may be least transformative in the communities that its designers would presumably most like to reach. The paradox emerges, then, when we observe that network interventions may increase the gaps between communities with relatively high and low fragmentation (i.e., before the intervention). This would happen in particular if an intervention led to relatively lower

fragmentation in communities that already had lower fragmentation but not in communities with higher fragmentation.

Our study is not without limitations, which should be considered when interpreting our findings. First, our empirical analysis relies on observational data. Although we have attempted to address endogeneity concerns using statistical methods, caution should be used when giving our results a causal interpretation. Second, our methodology assumes that HSAs correspond to meaningful health care delivery communities, an approach that is consistent with prior research. However, to the extent that this assumption is not met, interpretation of our results becomes more challenging. Finally, our network maps rely on administrative claims data. On the one hand, this approach is attractive because it allows us to observe networks in hundreds of communities, across the U.S., over time, which would be virtually impossible using other means (e.g., surveys). In addition, prior work has conducted extensive validation exercises to suggest that shared patients in Medicare claims correspond to meaningful relationships (Barnett et al., 2011; Everson et al., 2017; Funk et al., 2018). On the other hand, our reliance on administrative data necessarily means that we are unlikely to capture all potentially relevant relationships.

Notwithstanding these limitations, we believe our study has several implications for future research. First, more work needs to be done on the design of network interventions. In particular, our results suggest that widening gaps between the most and the least connected groups may be potentially unintended consequence of network interventions that work by providing exogenous inducements for connecting. An important question then is whether and how the design of such interventions may be adapted in ways that minimize these consequences and therefore lead to potentially more egalitarian outcomes. For example, one approach may be to limit the participation of groups that already have relatively robust network configurations. Regardless of the precise approach, future work that identifies and evaluates alternatives and articulates the general principles underpinning the effective design of network interventions would represent a major contribution.

The focus of our study was on a particular category of network interventions, specifically those that aim to reduce fragmentation by providing exogenous inducements. While interventions of this sort are common in both the literature and in practice, they represent only one approach for shaping social networks. Policymakers and managers often work to facilitate connections by introducing shared social contexts—break rooms, workspaces, neighborhood parks. Future research may benefit from examining whether the introduction of shared social contexts entails similar challenges to those we observe in this study.

Finally, future work may also benefit from more explicitly attempting to reconcile literature on network dynamics with growing work on network interventions. Thus far, findings from empirical research suggest the need for caution in applying insights from the literature on network dynamics to the study of interventions. Although we know, for example, from work on network dynamics, that *ceteris paribus*, people are more likely to form a connection when the benefits (or costs) for both parties are greater (or lower), when these relative benefits (or costs) are artificially manipulated (e.g., via an intervention) we may not necessarily observe the same behavior. Part of the challenge may be that in more natural settings, the relative benefits of two people forming a connection are a function of many different (potentially contingent) factors that are difficult or impossible to wrap up in a single intervention, thereby making intervention design and modeling difficult. Nevertheless, there are clearly many ways in which the literature on network dynamics may inform work on network interventions (and vice versa); research that attempt to clarify the phenomenological underpinning of the two approaches would therefore be particularly valuable.

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Table 1. Descriptive statistics and correlations

Variable	Mean	SD	Min	Max	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
(1) Providers joining ACOs (log)	1.27	1.42	0.00	7.72	1.00								
(2) Providers in ACOs (log)	1.58	1.59	0.00	8.06	0.94	1.00							
(3) Fragmentation	79.46	12.73	0.00	100.00	0.37	0.38	1.00						
(4) Providers (log)	4.67	1.45	1.10	9.89	0.71	0.72	0.53	1.00					
(5) External referral relationships (weighted)	74.60	17.72	19.79	100.00	-0.43	-0.43	-0.05	-0.69	1.00				
(6) Readmission rate (adjusted)	20.08	2.91	4.25	36.99	-0.05	-0.05	0.07	-0.04	0.11	1.00			
(7) Primary care coverage rate (adjusted)	48.00	9.13	7.53	81.15	-0.10	-0.11	-0.19	-0.10	-0.11	-0.06	1.00		
(8) Multiple hospitals (1 = yes)	0.21	0.41	0.00	1.00	0.45	0.44	0.24	0.60	-0.52	-0.08	-0.07	1.00	
(9) No hospitals (1 = yes)	0.06	0.23	0.00	1.00	-0.06	-0.06	0.14	-0.12	0.24	0.01	-0.04	-0.12	1.00

Notes: N=8,382. Correlations reported in this table are based on data from the years 2012 through 2014. Although some of our analyses use additional years, we report correlations for 2012 through 2014 because data from these years are included in all models.

Table 2. Regression models of providers joining ACOs

	M1 [†]	M2 [†]	M3 [†]	M4 [‡]
Estimator	OLS	OLS	OLS	IV
Network characteristics				
Fragmentation		-0.01*** (0.00)	-0.01*** (0.00)	-0.11*** (0.02)
Providers (log)	0.57*** (0.13)		0.67*** (0.14)	1.76*** (0.34)
External referral relationships (weighted)	0.01** (0.00)		0.01*** (0.00)	0.06*** (0.01)
Market characteristics				
Readmission rate (adjusted)	0.00 (0.00)		0.00 (0.00)	0.00 (0.01)
Primary care coverage rate (adjusted)	-0.00 (0.00)		-0.00 (0.00)	-0.00 (0.00)
Multiple hospitals (1 = yes)	0.04 (0.10)		0.03 (0.10)	-0.09 (0.13)
No hospitals (1 = yes)	-0.15 (0.11)		-0.14 (0.11)	0.06 (0.11)
Year fixed effects	Yes	Yes	Yes	Yes
HSA fixed effects	Yes	Yes	Yes	Yes
N	11192	11192	11192	11192
R2	0.73	0.73	0.73	0.68

Note: Dependent variables for all models in this table are measured at time t+1.

[†] Robust standard errors in parentheses.

[‡] Cluster-robust standard errors (on state) in parentheses.

*p<0.1; **p<0.05; ***p<0.01

Table 3. Regression models of fragmentation

	M5 [†]	M6 [†]	M7 [†]	M8 [†]	M9 [†]	M10 [†]	M11 [†]	M12 [†]
Estimator	OLS	OLS	OLS	OLS	OLS	IV	IV	OLS
Sample	All HSAs	All HSAs	All HSAs	Low preintervention (2011) fragmentation HSAs	High preintervention (2011) fragmentation HSAs	Low preintervention (2011) fragmentation HSAs	High preintervention (2011) fragmentation HSAs	All HSAs
Network characteristics								
Fragmentation _{t-1} [§]		0.25*** (0.03)	0.23*** (0.03)					0.17*** (0.03)
Providers in ACOs (log) [§]		-0.36*** (0.07)	-0.44*** (0.05)	-0.59*** (0.09)	-0.09** (0.04)	-2.17** (1.04)	-0.46 (0.33)	0.10 (0.07)
Fragmentation _{t-1} × Providers in ACOs (log) [§]								-0.09*** (0.01)
Providers (log)	9.15*** (1.06)		9.29*** (1.04)	16.51*** (1.64)	5.21*** (0.95)	18.22*** (1.72)	5.68*** (1.26)	9.83*** (1.05)
External referral relationships (weighted)	0.36*** (0.02)		0.34*** (0.02)	0.57*** (0.03)	0.26*** (0.02)	0.58*** (0.05)	0.26*** (0.03)	0.38*** (0.02)
Market characteristics								
Readmission rate (adjusted)	0.03 (0.02)		0.02 (0.02)	-0.01 (0.04)	0.04** (0.02)	0.01 (0.04)	0.05* (0.02)	0.01 (0.02)
Primary care coverage rate (adjusted)	-0.01 (0.01)		-0.01 (0.01)	-0.02 (0.02)	-0.01 (0.01)	-0.03** (0.02)	-0.01 (0.01)	-0.02* (0.01)
Multiple hospitals (1 = yes)	2.33*** (0.15)		1.86*** (0.14)	1.77*** (0.26)	0.90*** (0.11)	1.25** (0.48)	0.79*** (0.17)	1.40*** (0.13)
No hospitals (1 = yes)	2.12*** (0.42)		2.15*** (0.38)	-0.22 (1.22)	1.12*** (0.27)	0.04 (1.04)	1.15*** (0.19)	1.68*** (0.38)
Year fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
HSA fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
N	11088	11088	11088	5009	6079	4944	6015	11088
R2	0.94	0.94	0.94	0.91	0.93	0.90	0.93	0.95

Note: Dependent variables for all models in this table are measured at time t.

[†]Robust standard errors in parentheses.

^{*}Cluster-robust standard errors (on state) in parentheses.

[§]Mean centered when model includes an interaction term.

*p<0.1; **p<0.05; ***p<0.01

Table 4. Robustness analyses

	M13 [†]	M14 [†]	M15 [†]	M16 [†]	M17 [‡]	M18 [‡]
Description of analysis	ACO HQs	Unique ACOs	MC membership	Lagged exposure	2011 only	Placebo test
Estimator	IV	IV	IV	IV	OLS	IV
Dependent variable	Number of new ACO headquarter offices _{t+1}	Number of unique ACOs formed (log) _{t+1}	Providers joining ACOs (log) _{t+1}			
Network characteristics						
Fragmentation	-0.02 (0.02)	-0.05*** (0.01)		-0.18*** (0.03)	-0.01*** (0.00)	-0.12*** (0.03)
Main component (fraction)			13.89*** (2.92)			
Providers (log)	0.17 (0.26)	0.81*** (0.15)	1.54*** (0.36)	2.61*** (0.51)	0.85*** (0.02)	0.54** (0.24)
External referral relationships (weighted)	0.01 (0.01)	0.03*** (0.00)	0.08*** (0.02)	0.10*** (0.02)	0.01*** (0.00)	0.01** (0.01)
Market characteristics						
Readmission rate (adjusted)	0.00 (0.00)	0.00 (0.00)	0.01 (0.01)	0.01 (0.01)	-0.01*** (0.00)	0.02* (0.01)
Primary care coverage rate (adjusted)	-0.00 (0.00)	-0.001 (0.00)	-0.00 (0.00)	0.00 (0.00)	-0.00 (0.00)	-0.00 (0.00)
Multiple hospitals (1 = yes)	-0.03 (0.04)	0.01 (0.05)	-0.09 (0.13)	-0.19 (0.12)	0.17*** (0.04)	0.17 (0.17)
No hospitals (1 = yes)	0.01 (0.05)	0.02 (0.07)	0.24* (0.14)	0.09 (0.18)	0.06 (0.05)	0.55* (0.31)
Other						
Lagged exposure				-0.00*** (0.00)		
Year fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
HSA fixed effects	Yes	Yes	Yes	Yes	No	Yes
N	11192	11192	11192	11192	11192	11144
R2	0.37	0.73	0.60	0.59	0.55	0.46

Note: Dependent variables for all models in this table are measured at time t+1.

[†]Cluster-robust standard errors (on state) in parentheses.

^{*}Robust standard errors in parentheses.

*p<0.1; **p<0.05; ***p<0.01

Table 5. Supplemental analyses

	M19 [†]	M20 [†]	M21 [†]	M22 [‡]	M23 [‡]	M24 [‡]	M25 [‡]	M26 [‡]
Description of analysis	Adjusted reimbursements (percentile)	Hospitals (2011)	Social capital	ACO diversity	Cross-ACO edges	ACO-nonACO edges	ACO/ network correspondence	Year interactions
Estimator	OLS	OLS	OLS	IV	IV	IV	OLS	OLS
Dependent variable	Providers joining ACOs (log) _{t+1}			HHI index (subtracted from 1) of providers over the distribution of ACOs _{t+1}	Number of patient-sharing relationships spanning ACO boundaries _{t+1}	Number of patient-sharing relationships between ACO member and non-ACO physician _{t+1}	Mutual information between provider ACO memberships and network _{t+1} (norm alized)	Fragmentation
Network characteristics								
Fragmentation [§]	-0.01*** (0.00)	-0.03*** (0.01)	-0.01*** (0.00)	0.03*** (0.01)	-0.00** (0.00)	-0.01*** (0.00)	0.00** (0.00)	
Providers in ACOs (log)							0.00*** (0.00)	572.485*** (39.85)
Providers (log)	0.67*** (0.14)	0.66*** (0.14)	0.69*** (0.14)	-0.11 (0.10)	0.03*** (0.01)	0.126*** (0.0396)	0.05** (0.02)	12.14*** (1.33)
External referral relationships (weighted)	0.01*** (0.00)	0.01*** (0.00)	0.01** (0.00)	-0.01*** (0.00)	0.00** (0.00)	0.01*** (0.00)	0.00 (0.00)	0.54*** (0.02)
Market characteristics								
Readmission rate (adjusted)	0.00 (0.00)	0.00 (0.00)	0.00 (0.00)	-0.00 (0.00)	0.00 (0.00)	-0.00 (0.00)	0.00* (0.00)	0.02 (0.03)
Primary care coverage rate (adjusted)	-0.00 (0.00)	-0.00 (0.00)	-0.00 (0.00)	-0.00 (0.00)	-0.00 (0.00)	-0.00 (0.00)	0.00 (0.00)	-0.01 (0.01)
Multiple hospitals (1 = yes)	0.03 (0.10)			0.04** (0.02)	0.00 (0.00)	-0.00 (0.01)	0.00 (0.01)	-0.76*** (0.19)
No hospitals (1 = yes)	-0.14 (0.11)			-0.01 (0.04)	-0.00 (0.00)	-0.02 (0.02)	-0.01 (0.01)	1.18 (0.74)
Adjusted reimbursements [§] (percentile)	0.00 (0.00)							
Social capital [§] (mean centered)			-0.79 (2.49)					
Year								1.09*** (0.08)
Interactions								
Fragmentation [§] × Hospitals [§] (2011)		0.02*** (0.01)						
Fragmentation [§] × Adjusted reimbursements [§] (percentile)	0.00 (0.00)							
Fragmentation [§] × Social capital [§]			0.16** (0.07)					
Providers in ACOs (log) × Year								-0.28*** (0.02)
Year fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
N	11192	11192	11192	8005	11180	11180	8004	11192
R2	0.73	0.73	0.73	0.66	0.51	0.67	0.71	0.94

[†]Robust standard errors in parentheses.

[‡]Cluster-robust standard errors (on state) in parentheses.

[§]Mean centered when model includes an interaction term.

*p<0.1; **p<0.05; ***p<0.01

Figure 1. Hypothesized relationships. This figure summarizes the predictions of our hypotheses. Hypothesis 1 predicts that as fragmentation increases, communities will be less likely to engage with network interventions. Hypothesis 2 predicts that in communities with greater preintervention fragmentation, network interventions will be less effective for reducing future fragmentation.

Figure 2. Rate of physicians joining ACOs (per 100) as of 2015. This figure shows the total proportion of physicians in MSSP ACOs (per 100 physicians) in hospital service areas across the United States for the year 2015 (the final year of our study). Physician ACO membership was determined using the 2015 ACO Provider RIF from CMS; the local physician population was determined using practice addresses from the NPPES registry. The labels “Low,” “Moderate,” and “High” correspond to terciles of the proportion of physicians in MSSP ACOs, with “High” indicating the greatest rate of ACO penetration.

Figure 3. Fragmentation by hospital service area in 2011. This figure shows regional variation in our measure of network fragmentation for hospital service areas across the United States, in the year immediately before the start of the Medicare Shared Savings Program. Regional networks were mapped using data from Medicare claims on shared patients among local physicians. The labels “Low,” “Moderate,” and “High” correspond to terciles of fragmentation, with “High” indicating more fragmented (i.e., less connected) regional networks.

Figure 4. Network intervention paradox In Panel (A), we split communities into two groups based on their preintervention fragmentation (as of 2011). We use normalized preintervention fragmentation, fragmentation divided by the size of community (i.e., number of providers), to split communities. *Low fragmentation* corresponds to communities below median, and high fragmentation group corresponds to those above median. In the graph, the two lines represent the average number of providers joining ACOs in each year of the study. Consistent with our regression results, we see that communities with low preintervention fragmentation engage more with the intervention (i.e., adopt more) than those with high preintervention fragmentation, and this difference in engagement *widens* over time. In Panel (B), for each year, we plot changes in (normalized) fragmentation relative to the preintervention period (i.e., 2011) for Low fragmentation and High fragmentation communities (using the same definitions of low and high fragmentation as Panel [A]). Consistent with our regression results, we see that in the postintervention period *Low fragmentation* communities become *less* fragmented while *High fragmentation* communities become more fragmented. In Panel (C), we plot predicted values from Model 26 of Table 5, which predicts fragmentation as a function of ACO adoption, separately for communities with *High* and *Low ACO adoption*, where top 5% level of adoption refers to *High ACO adoption* and top 95% level of adoption refers to *Low ACO adoption*. Consistent with what we see in Panel (A) and Panel (B), differences in fragmentation between two groups *increases* over time.

Supplementary Appendix

Appendix 1. Distribution of “network” related terms in ACO names

Appendix 2. Taxonomy codes included in the networks

Appendix 3. Assessments of the validity of the network fragmentation measure

Appendix 4. Locations of physician and her ACO headquarters

Appendix 5-1. First stage models

Appendix 5-2. Assessments of the instrumental variables

Appendix 1. Distribution of “network” related terms in ACO names

Notes: This figure helps to support our view of ACOs as network interventions by showing that many ACOs in the MSSP use “network” and/or related word stems (e.g., “allianc”, “coalit”, “collabor”) in their names. Based on the commonality of such terms, we can infer that many ACO founders appear to view building and strengthening collaborative relationships as an important objective of the program.

Appendix 2. Taxonomy codes included in networks

Taxonomy code	Specialty
193	Group [†]
204	Electrodiagnostic Medicine
	Neuromusculoskeletal Medicine & OMM
	Neuromusculoskeletal Medicine, Sports Medicine
	Oral & Maxillofacial Surgery
	Transplant Surgery
207K	Allergy & Immunology
207L	Anesthesiology
207N	Dermatology
207P	Emergency Medicine
207Q	Family Medicine
208U	Clinical Pharmacology
208D	General Practice
208M	Hospitalist
208C	Colon & Rectal Surgery
207R	Internal Medicine
207S	Medical Genetics
207T	Neurological Surgery
207V	Obstetrics & Gynecology
207W	Ophthalmology
207Y	Otolaryngology
208V	Pain Medicine
2080	Pediatrics
2081	Physical Medicine & Rehabilitation
2082	Plastic Surgery
2083	Preventive Medicine
2084	Psychiatry & Neurology
2086	Surgery
208G	Thoracic Surgery (Cardiothoracic Vascular Surgery)
2088	Urology

[†]A business entity under which one or more individuals' practice.

Appendix 3. Assessments of the validity of the network fragmentation measure

We conducted several supplemental analyses to help establish our measure of fragmentation as a meaningful one in our research setting. These assessments consisted of three categories: (1) comparisons with alternative measures of fragmentation, (2) associations between fragmentation and other network properties, and (3), associations between fragmentation and substantive variables identified by prior work as being related to fragmentation. The results of all three categories of assessments are shown in the below table, which is organized as follows. Rows correspond to variables interest and are grouped into several logical categories, including alternative measures of fragmentation, other relevant network measures, healthcare cost, healthcare quality, and healthcare capacity.

With respect to alternative measures of fragmentation, we considered several possibilities. The measure *Fragmentation* (without non-referring isolates) is a variation on our main *Fragmentation* measure but excludes from the network providers who do not make referrals in the focal region and year. *Main component* (fraction) is an alternative approach to fragmentation that captures the proportion of nodes in the largest connected component (i.e., set of nodes that are reachable through a direct or indirect path) of the regional network; thus, higher values on this measure indicate less fragmentation. In our main analyses, we focused on relationships among providers within the same HSA; *Fragmentation* (extended network) is an alternative measure that also adds the direct ties between local providers and providers in other HSAs to the network. Finally, while our main analyses measure fragmentation at the level of the hospital service area (HSA), we also computed an alternative proxy at the level of the larger, hospital referral region (HRR).

For each variable (i.e., the rows in the table), we report basic descriptive statistics and correlations with fragmentation. The first column is correlation with fragmentation. Columns (2)-(4) report correlations among demeaned variables, each by region, year, and both. Columns (5) and (6) report partial correlations with fragmentation, after controlling for nodes and isolates, respectively. Last, in columns (7) and (8), we report mean values of each variable for two groups, regions that had fragmentation below the median of each year and regions that had fragmentation above the median of each year. The unit of analysis in the table is the HSA-year, and we use the same samples as our main analyses. In what follows, we give an overview of the results for each of the three categories of analyses.

To begin, raw correlations without adjustments (column 1) are generally as expected. Our primary measure of *Fragmentation* is generally highly (and significantly) correlated with alternative proxies for fragmentation. This result is reassuring, as it suggests our findings are unlikely driven by measurement choices. We also observe that higher fragmentation is associated with higher cost and lower quality. Correlations with other network properties are also consistent with our expectations.¹⁹ Finally, higher fragmentation also appears to be associated with lower health care capacity (as evidenced by the negative correlation between *Fragmentation* and *Primary care coverage rate (adjusted)* [$r=-0.19$; $p<0.05$] and the positive correlation between *Fragmentation* and *No hospital* [$r=0.15$; $p<0.05$]). Interestingly, we also observe a positive association between *Fragmentation* and *Multiple hospitals* ($r=0.23$; $p<0.05$). While clearly regions with multiple hospitals have greater healthcare capacity, this association seems reasonable to the extent that multiple hospitals may be indicative of greater market competition. In sum, even in the absence of statistical adjustments, assessments of face validity

¹⁹ The correlation between fragmentation and (11) *Path-length* is relatively low ($r=0.05$; $p<0.05$). This may be because in our calculation of average path length, we set infinite path lengths (which occur when pairs of nodes are in disconnected components) equal to the number of nodes in the network. This approach has the effect of inflating our measure of path length.

suggest our measure of *Fragmentation* performs well in our setting. After adjusting for various confounding factors in columns (2)-(6), fragmentation is generally less correlated with each variable, but the patterns of association are still generally significant and in the expected directions. Finally, differences in the mean values of each variable for high/low fragmentation regions (shown in columns 8 and 9, respectively) are consistent with the pattern of correlations we observe in column (1).

Assessments of the validity of the network fragmentation measure[†]

					Correlation with fragmentation			Partial correlation with fragmentation		Mean values [§]		
					Demeaned HSA	Demeaned year	Demeaned HSA and year	Control for nodes	Control for isolates	Low frag.	High frag.	
Category		Variable	Mean	S.D.	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Fragmentation	(1)	Fragmentation [†]	0.76	0.18							0.64	0.88
	(2)	Fragmentation (without non-referring isolates)	0.54	0.19	0.82*	0.68*	0.82*	0.67*	0.81*	0.81*	0.43	0.66
	(3)	Main component (fraction) [‡]	0.56	0.17	-0.90*	-0.87*	-0.90*	-0.86*	-0.89*	-0.89*	0.69	0.44
	(4)	Fragmentation (extended network)	0.72	0.13	0.58*	0.32*	0.58*	0.29*	0.55*	0.55*	0.67	0.77
	(5)	Fragmentation (HRR)	0.89	0.05	0.30*	0.26*	0.29*	0.07*	0.27*	0.27*	0.87	0.90
	(6)	Fragmentation (HRR, without non-referring isolates)	0.69	0.06	0.23*	0.17*	0.23*	0.02	0.20*	0.20*	0.68	0.71
Network Measures	(7)	Providers [‡]	295.99	940.18	0.23*	0.03*	0.23*	-0.06*	0.23*	0.06*	73.89	520.96
	(8)	Density	0.16	0.18	-0.96*	-0.93*	-0.96*	-0.92*	-0.96*	-0.96*	0.26	0.05
	(9)	Clustering coefficient	0.61	0.21	-0.45*	-0.16*	-0.45*	-0.14*	-0.42*	-0.43*	0.69	0.52
	(10)	Components	161.55	585.40	0.23*	0.06*	0.23*	-0.03*	-0.02*	0.11*	26.24	297.77
	(11)	Path-length	16.06	323.28	0.05*	0.01	0.05*	0.00	0.05*	0.04*	3.24	29.15
	(12)	Isolates	158.66	579.59	0.23*	0.06*	0.23*	-0.03*	-0.03*	0.23*	24.99	293.22
	(13)	Non-referring isolates	139.08	532.41	0.22*	0.04*	0.22*	-0.04*	-0.07*	-0.25*	19.74	258.94
	(14)	External referral relationships (weighted) [‡]	76.96	17.44	-0.09*	0.38*	-0.09*	0.35*	-0.00	-0.02	77.55	76.29
Healthcare Cost	(15)	Adjusted reimbursements (percentile) [‡]	9532.10	1548.20	0.02*	-0.01	0.02*	-0.01	0.02*	0.02*	9470.67	9597.29
Healthcare Quality	(16)	Readmission rate (adjusted) [‡]	20.08	2.91	0.08*	0.03*	0.07*	0.00	0.10*	0.10*	20.06	20.09
Healthcare Capacity	(17)	Primary care coverage rate (adjusted) [‡]	48.04	9.55	-0.19*	-0.12*	-0.18*	-0.04*	-0.17*	-0.17*	49.96	46.28
	(18)	Multiple hospitals (1 = yes) [‡]	0.17	0.38	0.23*	0.03*	0.23*	-0.02	0.15*	0.16*	0.09	0.26
	(19)	No hospitals (1 = yes) [‡]	0.06	0.24	0.15*	0.02	0.15*	0.02	0.17*	0.16*	0.02	0.09

*p<0.05

[†]Samples are restricted to observations from 2012 to 2014 that are included in all models. Descriptive statistics differ from those in Table 1 in our main text because there were 6 observations for which the Path-length variable (row 11) was undefined; these 6 observations were included in our main analyses.

[‡]Variable appears in the main analyses; the raw fragmentation measure is not multiplied by 100 here (row 1), and the number of providers is not log-transformed (row 7).

[§]Mean values of all variables in the table for observations that have high fragmentation (column 8) are significantly different (p<0.05) from observations that have low fragmentation (column 7), except for *Adjusted reimbursements (percentile)* (row 15).

Appendix 4. Locations of physician and her ACO headquarters

Notes: This figure shows that more than half of physicians who are affiliated with ACOs have their practices located in a different region from their ACO headquarters. This pattern supports the exclusion restriction in our models that use ACO headquarters foundings in proximal regions as an instrumental variable. Second, this figure helps explaining statistically insignificant findings in Model 13, where we use the number of ACO headquarters foundings as a measure of engagement. Since location of physicians and their ACO headquarters may not coincide, fragmentation in an area is not directly related to engagement in the network intervention program (i.e., ACO program).

Appendix 5-1. First stage models

Main Model	FM4 [†]	FM10 [†]	FM11 [†]
Estimator	OLS	OLS	OLS
Sample	All HSAs (2011 – 2015)	HSAs with low preintervention fragmentation (2012 – 2015)	HSAs with high preintervention fragmentation (2012 – 2015)
Dependent variable	Fragmentation	Providers in ACOs (log) _{t+1}	Providers in ACOs (log) _{t+1}
Network characteristics			
Providers (log)	10.28*** (1.17)	0.88*** (0.17)	1.25*** (0.26)
External referral relationships (weighted)	0.41*** (0.03)	0.01 (0.00)	0.01 (0.00)
Market characteristics			
Readmission rate (adjusted)	0.05* (0.03)	0.00 (0.00)	-0.00 (0.01)
Primary care coverage rate (adjusted)	0.01 (0.01)	-0.00* (0.00)	-0.01** (0.00)
Multiple hospitals (1 = yes)	2.33*** (0.22)	-0.29*** (0.07)	-0.25*** (0.04)
No hospitals (1 = yes)	2.34*** (0.42)	0.11 (0.10)	0.16** (0.07)
Instruments			
Medicaid expansion	-0.69 (0.45)		
Poverty	-0.00*** (0.00)		
Medicaid expansion × Poverty	-0.00** (0.00)		
State congress political tendency _{t-1}		-0.08 (0.13)	-0.11 (0.15)
Governor political tendency _{t-1}		0.08 (0.13)	-0.33* (0.17)
State congress political tendency _{t-1} × Governor political tendency _{t-1}		-0.36** (0.18)	-0.56* (0.28)
Nearby ACO headquarters _{t-1}		0.14*** (0.04)	0.12*** (0.03)
Year fixed effects	Yes	Yes	Yes
HSA fixed effects	Yes	Yes	Yes
N	13965	4944	6015
R2	0.92	0.80	0.89

Note: Dependent variables in FM10 and FM11 are measured at time t+1.

[†] Cluster-robust standard errors (on state) in parentheses.

*p<0.1; **p<0.05; ***p<0.01

Appendix 5-2. Assessments of the instrumental variables

Main model	M4 [†]	M10 [†]	M11 [†]	M13 [†]	M14 [†]	M15 [†]	M16 [†]	M18 [†]	M23 [†]	M24 [†]	M25 [†]
Underidentification test											
Kleibergen-Paap rk LM statistic	19.40	9.26	14.47	19.40	19.40	20.08	17.49	6.34	18.37	19.41	19.41
Chi-sq(3) P-val =	0.00	0.06	0.01	0.00	0.00	0.00	0.00	0.10	0.00	0.00	0.00
Weak identification test											
Cragg-Donald Wald F statistic	63.30	13.96	34.89	63.30	63.30	24.25	44.60	3.97	55.55	63.28	63.28
Kleibergen-Paap rk Wald F statistic	18.80	4.48	5.29	18.80	18.80	13.59	16.28	3.12	16.59	18.75	18.75
Stock-Yogo critical values	13.91	16.85	16.85	13.91	13.91	13.91	13.91	13.91	13.91	13.91	13.91
Overidentification test											
Hansen J statistic	4.64	5.04	4.76	1.49	0.62	4.73	0.19	1.77	0.86	3.51	4.67
Chi-sq(2) P-val =	0.10	0.17	0.19	0.47	0.73	0.09	0.91	0.41	0.65	0.17	0.10

[†]Instrument variable: Medicaid expansion

[‡]Instrument variable: nearby ACO headquarters and local political environments