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October 9, 2012

Abstract

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How does the pattern of social connections between individuals affect a country's income? Macroeconomists typically overlook findings of sociologists and anthropologists because social characteristics are difficult to observe, to describe formally and to quantify.[1] This paper uses tools from network analysis to explore how different social structures might affect a country's rate of technological progress. The network model also explains why societies might adopt growth-inhibiting structures and allows us to quantify the potential size of these effects. Motivated by the model, we use differences in the prevalence of diseases spread by human contact and the prevalence of other diseases as an instrument to identify an effect of social structure on technology diffusion.

There is a long history of measuring the speed of information or technology diffusion within various kinds of networks (Jackson (2008), Granovetter (2005)). Given these findings, a simple

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[1]There is a small economics literature and a much more extensive sociology literature on the effects of social institutions on income. See e.g. Greif (1994) for economics and Granovetter (2005) for a review of the sociology literature.

way to explain the effect of social structure on GDP is to show that some types of social networks disseminate new technologies more efficiently than others and append a production economy where the average technology level is related to output and income. There are two problems with this explanation. First, social contacts are presumably endogenous. If so, why would a social structure that inhibits growth evolve and persist? Second, this explanation is difficult to quantify or test. How might we determine if its effects are trivial or not? While researchers have mapped social networks in schools or on-line communities (Jackson, 2008), mapping the exact social network structure for an entire economy is not feasible.

Our theory for why some societies have growth-inhibiting social structures revolves around the idea that communicable diseases and technologies spread in similar ways - through human contact. We explore an evolutionary model, where some people favor local "collectivist" social networks and others do not. People who form collectives are friends with each others' friends. The collective has fewer links with the rest of the community. This limited connectivity reduces the risk of an infection entering the collective, allowing the participants to live longer. But it also restricts the group's exposure to new technologies. An individualist social network with fewer mutual friendships speeds the arrival of new technologies, which increases one's expected economic success and favors reproductive success. In countries where communicable diseases are inherently more prevalent, the high risk of infection for individualists makes the individualist trait die out. A collectivist social structure that inhibits the spread of disease and technology will emerge. In countries where communicable diseases are less prevalent, the collectivist types will be less economically and reproductively successful. Greater reproductive success of individualists causes the network to become fully individualist.

The idea that disease prevalence and social structure are related can help to isolate and quantify the effect of social structure on technology diffusion. Isolating this effect is a challenging task because technology diffusion and social structure both affect each other: Technology diffusion is a key determinant of income, which may well affect a country's social structure. To circumvent this problem, we instrument for social structure using disease prevalence data. By itself, disease prevalence would be a poor instrument because it is not likely to be exogenous: higher income levels would likely translate into better health and lower disease levels. Therefore, our instrument uses differences in the prevalence of two types of disease. The first type is diseases that are spread directly from person-to-person. These diseases might plausibly affect social structure because changing one's relationships with others can prevent transmission. The second type of diseases are those transmitted only through animals. Since direct human contact does not affect one's probability of infection, the prevalence of such diseases should not affect social structure. Thus, a main contribution of the paper is to use the difference in prevalence of communicable disease and

animal-transmitted disease as an instrument to measure the effect of social structure on income.

Our model explains why communicable disease might be correlated with social structure, how social structure can influence a country's technology diffusion and average productivity, and why less productive social structures might persist. We isolate one particular aspect of social structure, its degree of individualism versus collectivism, while holding all other aspects of the network fixed. Of course, many of these other aspects of networks may also differ across countries. We isolate collectivism because it is an important determinant of diffusion speed and we have cross-country data measuring it. But measuring other aspects of social networks and understanding their effects on economic growth would be useful topics for further research.

Section 1 begins by considering two exogenous networks, a collectivist and an individualist one. It describes the effect of collectives on disease and technology diffusion. Then, it considers networks that evolve and explores the reverse effects: how technology and disease affect the survival of individualist and collectivist types in the network. Numerical simulations in section 2 illustrate how these forces interact. It shows that higher disease prevalence creates the conditions for collectivist networks to emerge. Collectivist networks slow technology diffusion, which over time, can explain large income differences between collectivist and individualist societies.

Section 3 describes the historical pathogen prevalence data we collected from atlases of infectious disease, the measures of a society's individualism from Hofstede (2001), and the technology diffusion measure from Comin and Mestieri (2012). Section 4 uses this data to test the model's predictions for the relationship between disease prevalence and social structure. This establishes that disease prevalence is a powerful instrument for social structure. The section then goes on to estimate the effect of social structure on technology diffusion, using the difference in communicable and non-communicable diseases as an instrument. A main finding is that a 1-standard-deviation increase in individualism increases productivity by an amount equal to 23% of US productivity.

Related literature The paper contributes to four growing literatures. A closely related literature is one that considers the effects of social structure on economic outcomes. Most of this literature considers particular firms, industries or innovations and how they were affected by the social structure in place (e.g., see Granovetter (2005) or Rauch and Casella (2001)). In contrast, this paper takes a more macro approach and studies the types of social networks that are adopted throughout a country's economy and how those affect technology diffusion economy-wide. Ashraf and Galor (2012) and Spolaore and Wacziarg (2009) also take a macro perspective but measure social distance with genetic distance. Our network theory and findings complement this work by offering an endogenous mechanism to explain the origins of social distance and why it might be related to the diffusion of new ideas.

Thus in its scope, the paper is more related to a second literature, that on technology diffusion. Recent work by Lucas and Moll (2011) and Perla and Tonetti (2011) uses a search model framework where every agent who searches is equally likely to encounter any other agent and acquire their technology. Greenwood, Seshadri, and Yorukoglu (2005) models innovations that are known to all but are adopted when the user's income becomes sufficiently high. What sets this paper apart is its assumption that agents only encounter those in their network. Our insights about why societies adopt networks that do not facilitate the exchange of ideas and our links to empirical measures of social structure arise because of this focus on the network topology.

The third literature, on culture and its effects on national income is similarly macro in scope. Gorodnichenko and Roland (2011) focus on the psychological or preference aspects of collectivism. They use collectivism to proxy for individuals' innovation preferences and consider the effects of these preferences on income. In contrast, we view collectivism as a measure of human relationships and assess the effect of those relationships on income. Similarly, most work on culture and macroeconomics regards culture as an aspect of preferences.[2] Greif (1994) argues that preferences and social structure are intertwined because culture is an important determinant of a society's social structure. While this may be true, we examine a different determinant of social structure that is easily measurable for an entire country, pathogen prevalence. Our evolutionary-sociological approach lends itself better to quantifying the aggregate effects of social structure on economic outcomes.

Finally, our empirical methodology draws much of its inspiration from work on the role of political institutions by Acemoglu, Johnson, and Robinson (2002) and Acemoglu and Johnson (2005) and the role of social infrastructure by Hall and Jones (1999). But instead of examining institutions or infrastructure, which are not about the pattern of social connections between individuals, we study an equally important but distinct type of social organization, the social network structure.

1 A Network Diffusion Model

Our model serves three purposes. First, it is meant to fix ideas. The concept of social structure is a fungible one. We want to pick a particular aspect of social structure, the degree of collectivism in a social network, to anchor our analysis on. In doing this, we do not exclude the possibility that other aspects of social or cultural institutions are important for technology diffusion and income. But we do want to be explicit about what we intend to measure.

[2]See e.g., Tabellini (2010) and Algan and Cahuc (2007) who examine the relationship between cultural characteristics and economic outcomes, and Bisin and Verdier (2001) and Fernández, Fogli, and Olivetti (2004) who examine the transmission of culture. Durlauf and Brock (2006) review work on social influence in macroeconomics, but bemoan the lack of work that incorporates social network interactions.

Second, the model motivates our choice of disease as an instrument for social structure. Specifically, it explains why disease that is spread from human-to-human might influence a society's social network in a persistent way. The disease-based instrumental variable we use is a valid instrument, regardless of the veracity of this theory. The model simply offers one possible explanation for why disease and social structure might have the robust relationship we see in the data.

The third role of the model is that it helps us answer the following question: The richest countries have income and productivity levels that are 100 times higher than the poorest countries. Can differences in social structure plausibly explain such large income disparities? To answer this kind of question requires a model. Section 2 takes up this quantitative exercise.

A key feature of our model linking social structure to technological progress is that technologies spread by human contact. This is not obvious since one might think new ideas could be just as easily spread by print or electronic media. However, at least since Foster and Rosenzweig (1995), a significant subbranch of the growth literature has focused on the role of personal contact in technology diffusion; see Conley and Udry (2010) or Young (2009) for a review. In his 1969 AEA presidential address, Kenneth Arrow remarked,

> "While mass media play a major role in alerting individuals to the possibility of an innovation, it seems to be personal contact that is most relevant in leading to its adoption. Thus, the diffusion of an innovation becomes a process formally akin to the spread of an infectious disease."

With this description of the process of technological diffusion in mind, we propose the following model.

1.1 Economic Environment

Time, denoted by $t = \{1, \ldots, T\}$, is discrete and finite. At any given time t, there are n agents, indexed by their location $j \epsilon \{1, 2, \ldots, n\}$ on a circle. Each agent produces output with a technology $A_j(t)$:

$$y_j(t) = A_j(t).$$

Social networks Each person i is socially connected to γ other people. If two people have a social network connection, we call them "friends." Let $\eta_{jk} = 1$ if person j and person k are friends and $= 0$ otherwise. To capture the idea that a person cannot infect themselves in the following period, we set all diagonal elements (η_{jj}) to zero. Let the network of all connections be denoted N.

Spread of technology Technological progress occurs when someone improves on an existing technology. To make this improvement, they need to know about the existing technology. Thus, if

a person is producing with technology $A_j(t)$, they will invent the next technology with a Poisson probability λ each period. If they invent the new technology, $\ln(A_j(t+1)) = \ln(A_j(t)) + \delta$. In other words, a new invention results in a $(\delta \cdot 100)\%$ increase in productivity.

People can also learn from others in their network. If person j is friends with person k and $A_k(t) > A_j(t)$, then with probability ϕ, j can produce with k's technology in the following period: $A_j(t+1) = A_k(t)$.

Spread of disease Each infected person transmits the disease to each of their friends with probability π. The transmission to each friend is an independent event. Thus, if m friends are diseased at time $t-1$, the probability of being healthy at time t is $(1-\pi)^m$. If no friends have a disease at time $t-1$, then the probability of contracting the disease at time t is zero.

An agent who catches a disease at time t loses the ability to produce for that period ($A_j(t) = 0$). Let $\psi_j(t) = 1$ if the person in location j is sick in period t and $= 0$ otherwise. An agent who is sick in period t dies at the end of period t. At the start of period $t+1$, they are replaced by a new person in the same location j. That new agent inherits the same social network connections as the parent node. When we discuss network evolution, we will relax this assumption. At the start of period t, the new agent begins with zero productivity and learns the technology of each of his friends with probability ϕ, just like older agents do.

1.2 Two Illustrative Networks

The previous subsection described the economic environment for a given network. Before we add a process of network evolution, it is useful to compare the properties of two fixed networks. The evolutionary process will guide the economy to one of these two networks. They are the unique steady states of the stochastic network process. So, understanding how disease and technologies propagate in these two networks is very informative about the long-run behavior of our economy.

The two steady-state networks are extremes along a particular dimension, their degree of collectivism. This is an aspect of a social structure that has been extensively studied by sociologists. The collectivist network is one with many collectives, mutual friendships or instances of interdependence that are the hallmark of collectivist societies. To measure this interdependence, we can ask: If i is friends with j and with k, how often are j and k also friends? We refer to a structure where i, j and k are all connected to each other as a *collective*. Therefore, a measure of the extent of shared friendships, and thus the degree of collectivism, is the number of such collectives.

To count the number of collectives, we look at all the instances in a given network where one node i is connected to two other nodes j, k. Count that as a triple if j and k are connected. This collectives measure is related to a common measure of network clustering: Divide the number of

collectives by the number of possible collectives in the network to get the *overall clustering* measure (Jackson 2008).

To make our examples concrete, we will fix the number of connections γ to be 4. We explore the possibility of varying the number of connections later.

Network 1 *In the collectivist social network, each individual j is friends with the 4 closest people. In other words, $\eta_{jk} = 1$ for $k = \{j-2, j-1, j+1, j+2\}$ and $\eta_{jk} = 0$ for all other k.*

Network 1 is extreme in its degree of collectivism. The next result shows that there are as many collectives as there are members of the network (n).

Result 1 *In the collectivist network there are n unique collectives.*

The proof of this and all subsequent results are in appendix A.

At the other end of the spectrum, we examine a second network that is identical in every respect, except that it has the lowest possible degree of collectivism. We call that the individualistic network.

Network 2 *In the individualistic social network, each person is friends with the person next to them and the person m positions away from them, on either side. In other words, for any integer $m \in \{3, \ldots, n/2 - 3\}$, the network matrix has entries $\eta_{jk} = 1$ for $k = \{j-m, j-1, j+1, j+m\}$ and $\eta_{jk} = 0$ for all other k.*

Result 2 *In the individualistic network, there are zero collectives.*

These two network structures are particularly informative because of their starkly different numbers of collectives. This stark difference facilitates matching social institution data with one or the other type of network. As we will see, networks with numbers of collectives between 0 and n, are also possible along the transition path. But knowledge of the properties of these two extreme cases provides intuition about the properties of such intermediate cases as well.

Other dimensions along which networks could differ. There is a very large set of possible networks for an economy, too large to analyze completely. Therefore, we restrict attention to one dimension. We choose the prevalence of collectives because it represents the essence of collectivism, which is the sociological feature we have data on. But other dimensions of networks might also be closely related to collectivism. In particular, one might represent individualist societies as having more social linkages or capture the idea of market interactions with a time-varying, random network. We have investigated both of these aspects of networks and found that both more linkages and random networks facilitate the spread of technology and germs. Thus, we could instead base our

analysis on one of these other features and we would still come to the same conclusions: Having an individualist network exposes one to a greater risk of disease and a more productive set of technologies. In fact, preliminary analysis suggests that the quantitative effects of adding more linkages or random networks are even greater than for reducing the number of collectives.

1.3 Theoretical Results: Speed of Diffusion in Each Network

Disease spreads slowly in the collectivist network. The reason is that each contiguous group of friends is connected to at most 4 non-group members. Those are the two people adjacent to the group, on either side. Since there are few links with outsiders, the probability that a disease within the group is passed to someone outside the group is small. Likewise, ideas disseminate slowly. Something invented in one location takes a long time to travel to a far-away location. In the meantime, someone else may have re-invented the same technology level, rather than building on existing knowledge and advancing technology to the next level. Such redundant innovations slow the rate of technological progress and lower average consumption. The following results formalize these ideas.

Diffusion speed in each network The speed at which germs and ideas disseminate can be measured by the number of social connections in the shortest path between any two people. Consider an agent in position 1 and the agent farthest away from him on the circle, agent $n/2+1$. If each person has 2 friends on either side of them, then agent 1 will be friends with agent 3, who will be friends with agent 5, and this person will be friends with agent 7, etc., until we reach $n/2$. Thus, if the network size n is 6, $n/2+1$ is the farthest node. It could be reached in 2 steps: Agent 1 and agent 3 are directly connected and 3 is connected to 4. If $n/2+1$ is 6 ($n=10$), node 6 could be reached in 3 steps: from 1 to 3, 3 to 5 and 5 to 6. In general, the number of steps in this chain will be $(n-1)/4$, if that is an integer, or otherwise the next highest integer. The distance to this farthest person in the network is called the *network diameter*.

Diameter is one measure of diffusion speed because it tells us how many periods a new idea takes to travel to every last person in the network. If each person communicates the idea to each of their friends each period, then in n/γ periods, the farthest person in the network will have learned the idea, along with every other agent. Since disease is spread only probabilistically, from friend to friend, the diameter gives us the smallest number of periods in which every person is infected, with positive probability.[3] Appendix A computes the diameter (as well as the average path length) of our two networks. The diameter of network 1 (collectivist), with n nodes is $(n-1)/4$, if that is an

[3]Our network is symmetric. So, the length of the path to the farthest node is the same, no matter which node one starts at. But in general, the diameter is the maximum path length, over all starting nodes.

integer, or otherwise the next highest integer. Suppose, for example, that network 2 (individualist), has at least 4 nodes ($n > 4$) and $m = 4$ so that each node i is connected to $i-4$, $i-1$, $i+1$, and $i+4$. The diameter of this individualist network is round$(n/8)+1$. For large n, the diameter for the collectivist network is close to $n/4$, while the diameter of the individualist network is close to $n/8$. Therefore, as long as the network is sufficiently large, which for a country, it undoubtedly is, the individualist network will have a smaller diameter.

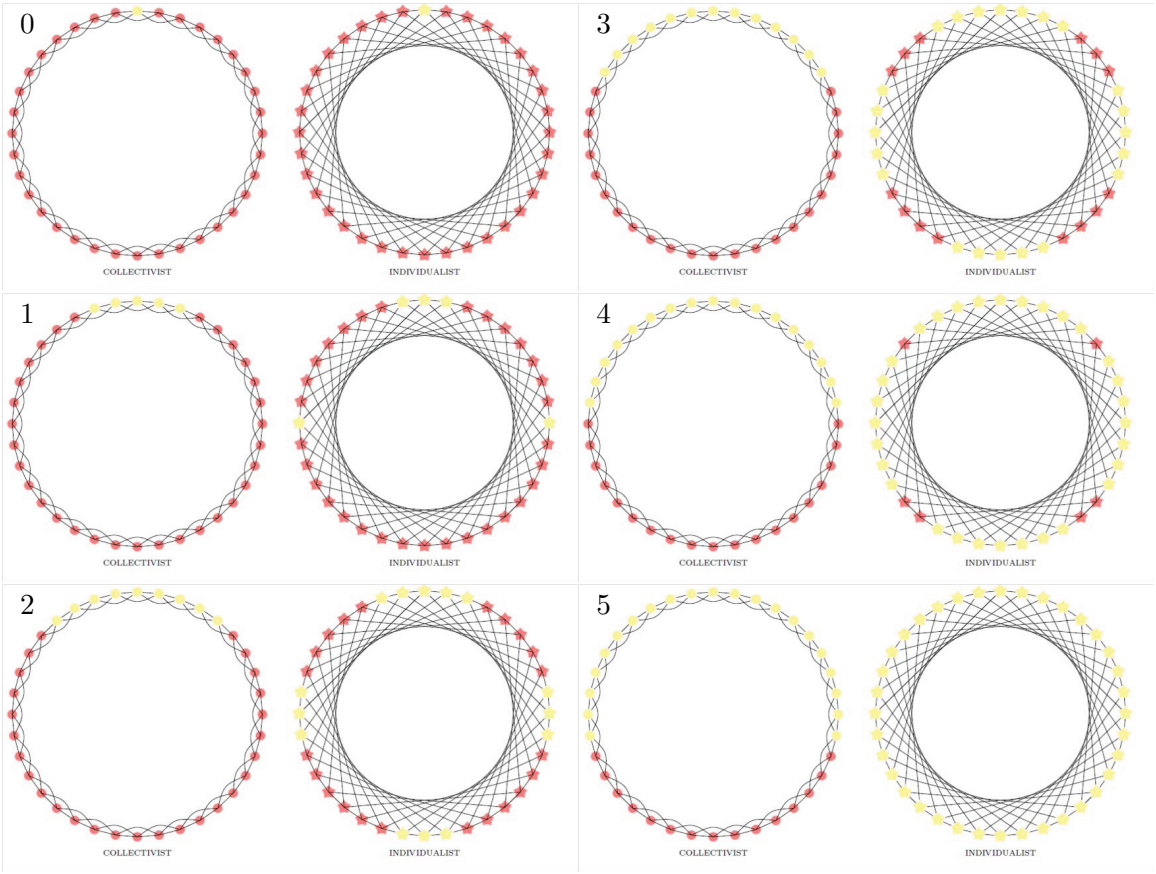
Figure 1 illustrates the smaller diameter and faster diffusion process in individualist networks, in the simple case where the probability of transmission is 1 and $m = 9$.[4] In both cases, a new technology arrives at one node in period 0. The "infected" person transmits that technology to all the individuals she is connected to. In period 1, 4 new people use the new technology, in both networks. But by period 2, there are 9 people using the technology in the collectivist network and 14 using it in the individualist network. In each case, an adopter of the technology transmits the technology to 4 others each period. But in the collectivist network, many of those 4 people already have the technology. The technology transmission is redundant. After 5 iterations, the new technology reaches every node in the individualist network. Thus, the diameter of the individualist network is 5. In contrast, it takes 9 iterations to reach all the nodes in the collectivist network. (The diameter is 9.) In sum, these properties tell us that, on average, ideas and diseases will diffuse more slowly through a collectivist network than an individualist one.

Diffusion speed and the technological frontier The diameter and average path length in a network are important determinants of the speed at which germs and ideas diffuse. In the individualistic network, because the path length between individuals is shorter, diseases and ideas disseminate more quickly. The next result uses the calculations above to characterize the mean and maximum infection times and the mean and maximum discovery time for a new technological innovation. Let $\Psi_j(t)$ be the next period in which the person living in location j at time t gets sick and dies. In other words, $\Psi_j(t) = min\{s : s \geq t, \psi_j(s) = 1\}$. Thus, $\psi_j(0)$ is number of periods that the person living in location j at time 0 will live. Analogously, let $\alpha_j(0)$ be the number of periods it takes for a new idea, introduced in period 0, to reach person j.

Result 3 *Consider two networks, an individualistic network (N2) and a collectivist network (N1). They have equal size $n > 8$, where $n/8$ is an integer, and an equal number of connections per node $\gamma = 4$. If $\pi = 1$ and $\sum_j \psi_j(0) = 1$, then the average lifetime $E_j[\Psi_j(0)]$ and the maximum lifetime $\max_j[\Psi_j(0)]$ are longer in the collectivist network (1) than in the individualist network (2).*

[4] A larger m makes the network connections easier to identify visually. But it also accentuates the difference in diffusion speed.

Figure 1: Slower diffusion in the collectivist network (left) than the individualist network (right).



If a technology is introduced at the top of the network at time 0 and is transmitted with probability one, the light-colored nodes denote the nodes that would adopt the technology in periods 1-5. The individualist network has $m = 9$. The diameter of the individualist network is 5 because it takes 5 iterations to reach the farthest node in the network. It would take another 4 iterations to reach all the nodes in the collectivist network. Therefore the diameter of the collectivist network is 9.

If $\phi = 1$, then the average discovery time $E_j[\alpha_j(0)]$ and the maximum discovery time $\max_j[\alpha_j(0)]$ are slower in the collectivist network (1) than in the individualist network (2).

With a collectivist network, technology invented in one location was transmitted only 2 people further each period. In the individualist network, ideas advance 4 places at a time. But faster diffusion is not the same as faster technological innovation. The reason that diffusion accelerates technology growth is that when idea diffusion is faster, redundant innovations are less frequent. Thus, more of the innovations end up advancing the technological frontier. The following result clarifies the mechanism by which the individualist network achieves a higher rate of growth.

Result 4 *Suppose that at t, a collectivist network (N1) and an individualist network (N2) have the*

same $A_j(t)$ $\forall j$. Then the probability that the next new idea arrival will increase the technological frontier is larger in N2 than N1.

Together, these results explain why ideas and germs spread more quickly in the individualistic network than in the collectivist network, why diffusion might imply a higher level of technology adoption or GDP, and what evolutionary advantages each type of network might offer its adopters.

Could Collectivism Facilitate Technology Diffusion? Perhaps Arrow was not correct and technology diffusion is not a process "formally akin to the spread of infectious disease." Instead, a technology is adopted only when a person comes in contact with multiple other people who have also adopted it. It is theoretically possible that having many mutual friendships makes it more likely that groups of people adopt a technology together. But the adoption complementarity needs to be very strong to overcome the fact that with collectivism, people are less likely to have any exposure to the new technology. Furthermore, such a theory does not help to explain the empirical findings, which will show that collectivism is associated with slower technology diffusion. Ultimately, this model is simply a framework for helping us think about what we find in the data. While other formulations that lead to opposite conclusions are possible, they don't help us to understand the facts at hand.

1.4 Network Evolution Model

So far, we have simply described diffusion properties of two networks. This leaves open the question of why some societies have one type of network or the other. One approach would be to work with a network choice model. But equilibria in such models often do not exist and when they do, they are typically not unique. Instead, we consider an evolutionary model where the network changes as agents die and new ones are born in their place. This evolutionary model also helps to explain why growth-inhibiting social structures might persist long after most diseases have died out.

Preferences, production, endowments and the diffusion processes for technology and disease are the same as in the fixed-network model. In addition, at each date t, each person j can be one of two types: They are either a collectivist $\tau_j(t) = co$ or an individualist $\tau_j(t) = in$. All agents are linked to the two people adjacent to them. In addition, they are linked to at least one other person. Which other people depends on their type and the type of their neighbors. Individualists form links with those adjacent to them and someone four spaces to their right. For example, if the person is in location j, they are linked to $j-1$, $j+1$ and $j+4$. Collectivists form links with those adjacent to them and someone two spaces to their right. For example, if the person is in location j, they are linked to $j-1$, $j+1$ and $j+2$. In addition, a person of either type might be linked

to nodes $j-2$ and/or $j-4$, depending on whether the agents in those locations are individualist or collectivist. In other words, a person's own type governs their links to the right (with indices higher than yours, except near n); others' types govern links to the left.

A person's type is fixed throughout their lifetime. The network structure only changes when someone dies. There are two reasons an individual can die. First, they can acquire the disease. Someone who acquires the disease at time t has zero output in period t. At the end of period t, they die. Second, agents can die stochastically, for non-disease related reasons (accident, old age, etc.). With probability ξ, each person has an accident and dies at the end of each period. This probability is independent across time and individuals. When someone at node j dies in period t, then at the start of period $t+1$, a new person inhabits that node. The reason we introduce this second cause of death is to allow the network to evolve, even after the disease has died out.

A newborn person inherits the best technology from the set of people that the parent was socially connected to. He also inherits the type of the person with that best technology. In other words, if the person at node j is socially connected to nodes $\{k : \eta_{jk}(t) = 1\}$ and dies at time t, the new person at node j at time $t+1$ will start with technology $\max_{\{k:\eta_{jk}(t)=1\}} A_{kt}$. Let k^* be the argument that maximizes this expression (i.e. the friend with the highest time-t technology), then the time-$(t+1)$ type of the person is the same as the time-t type of person k^*: $\tau_j(t+1) = \tau_{k^*}(t)$.

The idea behind this process is that evolutionary models often have the feature that more "successful" types are passed on more frequently. At the same time, we want to retain the network-based idea that one's traits are shaped by one's community. Therefore, in the model, the process by which one inherits the collectivist or individualist trait is shaped by one's community, the social network, and by the relative success (relative income) of the people in that network.

1.5 Theoretical Results: Network Evolution

The question we want this model to answer is: Why do some societies end up with a collectivist network even though it inhibits growth? What features might influence the long-run network equilibrium? These results describe the long-run properties of networks and disease. Understanding the stochastic process that governs disease and network type provides intuition for the numerical results in the next section, which will show that a higher initial prevalence of disease makes it more likely that a society will end up with a collectivist network, like that in network 1.

The first set of results show that eventually, the economy always converges to either the fully collectivist network (1) or the fully individualist one (2).

Result 5 *With probability 1, the network becomes homogeneous:* $\exists T$ *s.t.* $\tau_j(t) = \tau_k(t)$ $\forall k$ *and* $\forall t > T$.

In other words, after some date T, everyone will have the same type forever after. They might all be individualist or all be collectivist. But everyone will be the same. The reason for this is that since traits are inherited from neighbors, when a trait dies out, it never returns. The state where all individuals have the same trait is an absorbing state. Since there are a finite number of states, and whenever there exists a j, k such that $\tau_j(t) \neq \tau_k(t)$, every state can be reached with positive probability in a finite number of steps, then with probability one, at some finite time, an absorbing state is reached and the economy stays there forever after.

Similarly, having zero infected people is an absorbing state. Since that state is always reachable from any other state, with positive probability, it is the unique steady state.

Result 6 *With probability 1, the disease dies out:* $\exists T$ *s.t.* $\psi_j(t) = 0 \quad \forall j$ *and* $\forall t > T$.

What these results tell us is that which network type will prevail is largely dependent on which dies out first, the individualist trait, or the disease. When there is a positive probability of infection, people with individualist networks have shorter lifetimes, on average. If disease is very prevalent, it kills all the individualists and the society is left with a collectivist network forever after. If disease is not very prevalent, its transmission rate is low, or by good luck, it just dies out quickly, individualists will survive. Since they are more economically successful, they are more likely to pass on their individualist trait. So, the economy is more likely to converge to an individualist network. This is not a certain outcome because of exogenous random death. It is always possible that all individualists die, even if the disease itself is no longer present. The main take away is that networks can persist long after the conditions to which they were adapted have changed.

2 Numerical Results

We use a calibrated model simulation to accomplish three objectives. First, we use the simulations to illustrate and clarify the model's mechanics. Second, we check whether differences in networks can potentially explain the magnitude of the large differences in incomes across countries. Third, we establish that societies with higher initial disease prevalence are more likely to become collectivist. The model is not rich enough to produce predicted growth rates or disease rates that are accurate. Rather, the objective here is simply to confirm the direction of the model's predictions and gauge whether the predicted effects are trivial or not.

2.1 Parameter Choice

To evaluate magnitudes, we need to choose some realistic parameter values for our model. The key parameters are the probabilities of disease and technology transmission, the initial pathogen

Table 1: Parameters and their empirical counterparts

	Parameter	Value	Target
Initial disease prevalence	$\text{Prob}(\psi_j(0) = 1)$	0.5%	TB death rate in China
Disease transmission probability	π	32%	Disease disappears in 150 years (indiv country avg)
Innovation productivity increase	δ	30%	2.6% growth rate in individualist country
Technology transfer probability	ϕ	50%	Half-diffusion in 20 years (Comin et. al. '06)
Technology arrival rate	λ	0.25%	1 arrival every 2 years (Comin et. al. '06)
Exogenous death rate	ξ	1/70	average lifespan

prevalence rate and the rate of arrival of new technologies. These parameters are summarized in Table 1.

For the initial pathogen prevalence rate, we use the annual tuberculosis death rate in China, a country where the disease was endemic. Tuberculosis is the most common cause of death in our sample. Note that this is a mortality rate, not an infection rate. Since individuals who get sick in the model die, this is the relevant comparison. Also, it is a conservative calibration because it uses only one disease and it would be easier to get large effects out of a higher disease prevalence rate. One would like to choose the probability of disease transmission to target a steady state rate of infection. But, as we've shown, the only steady state infection rate is zero. Thus, we set the transmission rate so that, on average, the disease disappears in 150 years. This average masks large heterogeneity. In many economies, the disease will disappear after 2 periods. In others, it will persist for hundreds of years. Thus, the economy starts with a given fraction of the population being sick and each sick person represents an independent 32% risk (π) of passing the disease on to everyone that person is friends with.

Everyone starts with a technology level of 1. But each period, there is a chance that any given person may discover a new technology that raises their productivity. The rate of arrival of new technologies is calibrated so that a new technology arrives in the economy every 2 years, on average. This corresponds to the average rate of adoption of technologies in the (Comin, Hobijn, and Rovito, 2006) data set. The magnitude of the increase in productivity from adopting a new technology is calibrated so that the individualistic network economy (more likely to be the developed economy in the data) grows at a rate of 2.2% per year. The probability of transmitting a new technology to each friend (λ) is chosen to explain the fact that for the average technology, the time between invention and when half the population has adopted the technology is approximately 20 years

(Comin, Hobijn, and Rovito, 2006).

Finally, in the evolutionary model, there is a probability of exogenous death. We choose this probability to match an average lifespan in a low-disease economy of approximately 70 years.

The economy consists of 200 people, each with 4 friends. We average the results from 200 independent runs.

2.2 An Illustrative Numerical Example

To illustrate the mechanics of technology and disease diffusion, we first describe a small-scale illustrative example. Here, we hand-pick some of the parameter values (in particular, the rate of technology arrival) to make it easier to visualize diffusion taking place. Figure 2 illustrates the diffusion of technology and disease. Each box represents a person/date combination. Time is on the horizontal axis. People are lined up on the vertical axis according to their location. In the first period (first column of boxes on the left), everyone starts with the same technology level. But there are a few agents who have a disease (the darkest boxes).

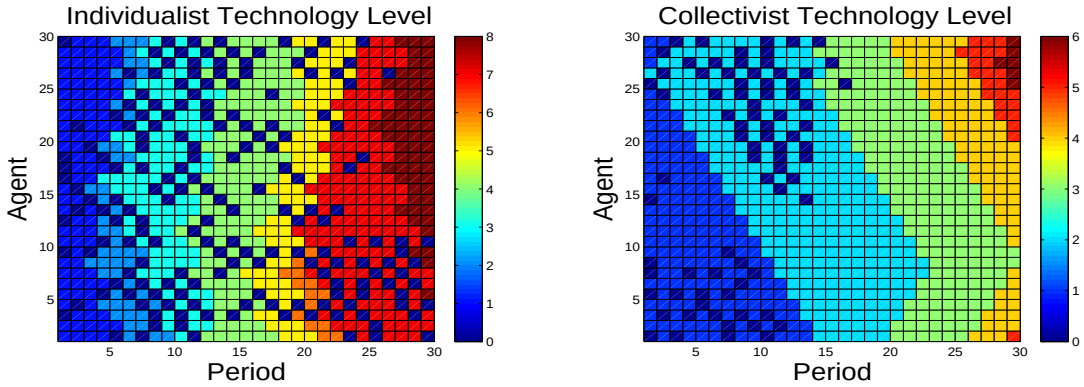


Figure 2: How disease and technology spread through networks. The darkest boxes indicate individuals who acquired the disease in period t and therefore have zero time-t productivity. Warmer colors indicate higher levels of technology.

By the second period, new ideas start to arrive. In the second column of boxes, there are a couple of lighter-colored boxes that indicate that these agents have reached the next technology level. In the collectivist network (left figure), some agents who are 1-2 places away from agents that were sick in period 1 are now sick. In the individualistic network (right figure), some agents who are 1 or 4 places away from agents that were sick in period 1 are now sick. In period 3, the new ideas that arrived in period 2 start to diffuse to nearby locations. In the collectivist network, some individuals are still using the initial technology level in period 8. In the individualistic network, all the healthy agents have adopted the second technology level after period 5.

After 30 periods, the most technologically advanced agents in the collectivist network only realize 7 steps in the quality ladder. In the individualistic network, some agents operate at 9 steps. If each innovation represents a 5% productivity increase, being two steps further represents a 10% higher degree of productivity.

This example is meant to illustrate how an individualistic network spreads ideas more efficiently, and how it also spreads germs more efficiently. Of course, this is just an example. It is a comparison of the maximum level of technology from a small number of agents. To get a sense of the aggregate effect, the following simulation uses the calibrated parameters and averages the results over many agents and many simulations.

2.3 How Much Effect Might Networks Have on Output?

A potential concern about using this model to explain income differences across countries is the worry that its predicted effect is trivial, compared to the vast differences in incomes across countries. What our calibration exercise shows is that changing a society's social network structure has a small effect on the annual diffusion rate. But over time, small effects cumulate. The result is large differences in productivity levels in the long run. Thus, changes in networks produce differences in technology diffusion rates which could explain a significant part of the disparity in countries' incomes. While idea transmission facilitates reaching higher levels of productivity, disease prevalence diminishes productivity. To see the net effect of these two forces, we simulate the model many times and examine the average outcomes.

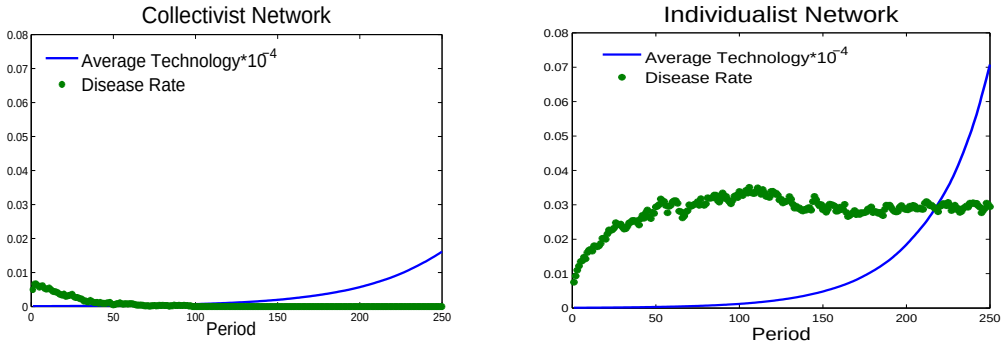


Figure 3: Average disease prevalence and productivity

Figure 3 plots the average disease prevalence and the average technology level for the whole population over 250 years. The fraction of the population infected with disease is significantly higher in the individualistic network society. In fact, the collectivist networks inhibit the spread of disease so much that it quickly becomes extinct in most simulation runs.

However, having an individualistic network results in technology that grows at 2.6% per year.

This is true by construction because it was one of the calibration targets. But the economy with the collectivist network grows at only 2.0% per year. While the difference in growth rates is small, in time, it produces large level differences. After 250 years, the average level of technology is 476% higher in the individualistic network than in the collectivist network. This simple example makes the point that a difference in network structure can create a small friction in technology diffusion. When cumulated over a longer time horizons, this small friction has the potential to explain larger differences in countries' incomes.

Of course, this also tells us that social structure is not likely to explain the nearly 100-fold difference between incomes in the poorest and richest countries. We know that corruption, war and distorted incentives explain the worst growth disasters. At the same time, 476% of national income is a large difference between seemingly similar countries. It is an extreme result in the sense that we compared a purely individualist network to a purely collectivist one. Most societies will lie somewhere in between. But it gives us an idea of the potential size of the effect. The actual effect is an empirical matter that we take up in the following sections.

2.4 Network Evolution

What we ultimately want to know from the evolutionary model is: Are high-disease societies more likely to evolve toward collectivist networks? One might wonder whether societies that start out as high-disease and adopt collectivist social structures might end up with lower disease rates in the long-run. That turns out not to be the case.

We would like to calculate the probability of arriving at each steady state (where all agents have the same type) analytically and see how that probability changes in response to changes in disease prevalence. However, to characterize the probability of a single stochastic process crossing one boundary before another is a difficult problem. Here, there are two interacting stochastic processes, one for disease and one for network types. Both have absorbing states. That added complexity makes characterizing the crossing probabilities an intractable problem.

What we can do is examine the probability of each network steady-state in the context of our numerical example. We use the same parameters as before. (See Table 1.) We set the initial fraction of individualists to 10% and simulate the economy for 250 periods 200 times. To see how the initial disease prevalence rate affects the network steady state, we consider two initial disease prevalence conditions: One is 5% (the calibration target of the original model) and the other is twice that level. Thus, there is a low disease economy, with 5% of agents infected, and a high disease economy that starts with 10% of agents infected.

Figure 4 shows the fraction of economies that have converged to a zero-disease steady-state or a purely collectivist steady-state by each date. This can also be interpreted as the probability

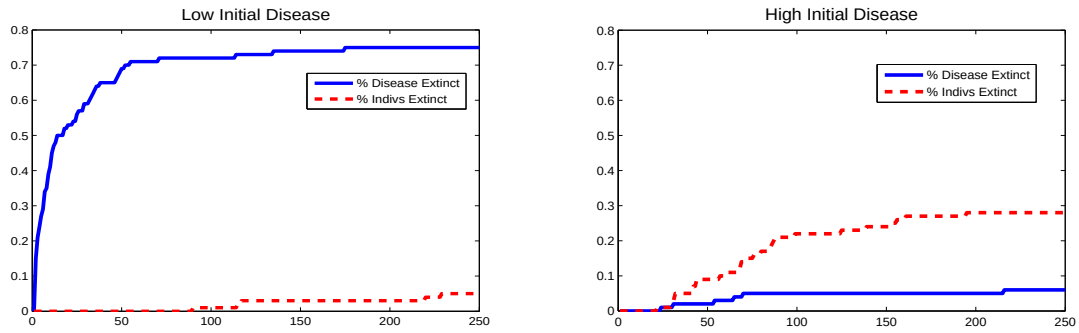


Figure 4: Simulation of the evolutionary model. The economy on the right differs only because it began with a (2×) higher rate of disease prevalence. High initial disease makes the probability of converging to a zero-individualist (purely collectivist) state more likely.

that a given economy will converge to that steady state by that date. In the low-disease economy, much of the time, the disease dies out within a few periods. Only in a few runs does the disease persist and infect a large fraction of the population. In the high-disease economy, the disease rarely dies out within 250 periods. Conversely, individualists flourish in the low-disease economy. In the high-disease economy, after 100 periods, there is a 25% chance that all individualists have died and that economy will forever remain collectivist.

Thus, the prediction of the model is clear: *Low-disease societies are more likely to be individualist and high-disease societies are more likely to be collectivist.* The secondary effect whereby collectivism reduces disease is always dominated by the primary effect that disease disproportionately kills individualists. To see why, consider the contrary: If high-disease societies were more individualist, the disease would systematically kill the individualist types and transform the society to a collectivist one. It is simply not a stable outcome.

3 Data

Our theory is about the relationship between pathogen prevalence, social structure, and technology diffusion. We have assembled a data set that contains all 3 variables for 62 countries. This section describes how these three variables are measured. Additional details, maps and summary statistics are in the appendix.

3.1 Measuring Pathogen Prevalence

We measure the presence of deadly pathogens in two ways. The first approach recognizes that disease conditions may take a long time to affect social networks and therefore it is desirable to use historical data. At the same time, because our identification strategy relies on differences in disease prevalence, our data must be available for many different diseases, across many countries. One can

go back to the colonial period (as in Acemoglu, Johnson, and Robinson (2001)), but the different kinds of diseases that we need to implement our identification strategy are not present in that data. A variety of diseases is present in disease atlases from the 1930's. Using these atlases, we compiled a data set of historical prevalence of 9 different pathogens in 75 geopolitical regions.[5] This data does appear to capture some long-run features of the epidemiological environment because they are remarkably consistent with the colonial data (see Appendix). Our second approach recognizes that, if we want to uses differences in diseases as an instrument, it is useful to have a large number of each type of disease. Therefore, we also use more recent data with the prevalence of 34 diseases in 78 geopolitical regions.

Historical pathogen data. To assess the historical prevalence of disease, we study 9 pathogens: leishmanias, leprosy, trypanosomes, malaria, schistosomes, filariae, dengue, typhus and tuberculosis. We choose these diseases because we have good worldwide data on their incidence, and they are serious, potentially life-threatening diseases that people would go to great length to avoid. The data come primarily from Murray and Schaller (2010). They are based on old atlases of infectious diseases and information originally collected by the U.S. military in the 1930's. They used a 4-point coding scheme: 0 = completely absent or never reported, 1 = rarely reported, 2 = sporadically or moderately reported, 3 = present at severe levels or epidemic levels at least once. The countries with the highest pathogen prevalence are Brazil, India, China, Nigeria and Ghana. Countries with the lowest prevalence include Canada, Switzerland, Luxembourg, Hungary and Sweden. Figure 7 shows the historical world-wide distribution of pathogens according to the overall index. This is the data we use for most of our analysis, including our IV estimations.

Contemporary data For comparison, we used the same method to create an alternative measure of pathogen prevalence based explicitly on contemporary information. The data come from GIDEON (Global Infectious Diseases and Epidemiology Network) and use a 3-point coding scheme to report the 2011 prevalence of 34 of the most common infectious diseases. For many of these diseases, the scheme is coded directly by GIDEON; in these cases, a value of "1" means "not endemic" (cases do not originate in this country), a value of "2" means "sporadic" ($<$ 1 case per million people, per year), and a value of "3" means "endemic" (an ongoing presence). The complete list of diseases we use, along with characteristics of each disease, is reported in table 8.

[5]The data on pathogen prevalence from the 1930's line up quite well the data on mortality rates from the colonial period (see Figure 8 in the appendix) for the subset of countries we have in common, showing that our data captures the same long run differences in the epidemiological environment.

Disease reservoirs To identify the effect of disease on social structure, we will use the difference in the prevalence of various types of diseases. Epidemiologists often classify infectious diseases by reservoir.[6] The reservoir is any person, animal, plant, soil or substance in which an infectious agent normally lives and multiplies. The reservoir serves as a source from which other individuals can be infected. The infectious agent depends on the reservoir for survival. It is from the reservoir that the disease is transmitted to humans. Animals often serve as reservoirs for diseases. There are also nonliving reservoirs, such as soil, which is a reservoir for fungi and tetanus. Figure 8 summarizes the properties and classification of all the pathogens that we collected data on.

Human-specific Many diseases have only human reservoirs, even though they historically may have arisen in other species, such as measles which originated in cattle. Such diseases may be spread with the help of an animal (called a vector), such as a mosquito that injects one person's blood in another person. But it is in the human, not in the mosquito, where the disease flourishes. Human-specific diseases in our data set include Diptheria, Filaria, Measles and Smallpox.

Zoonotic Other diseases, although they infect and kill humans, develop, mature, and reproduce entirely in non-human hosts. These are zoonotic diseases. Humans are a dead-end host for infectious agents in this group. Our zoonotic diseases include anthrax, rabies, schistosomiasis (SCH), tetanus, and typhus (TYP).

Multi-host Some infectious agents can use both human and non-human hosts to complete their lifecycle. We call these "multi-host" pathogens. Our multi-host diseases include leishmaniasis (LEI), leprosy (LEP), trypanosomes (TRY), malaria (MAL), dengue (DEN) and tuberculosis (TB).

Since multi-host and human-specific pathogens can reside in humans, they have the potential to affect the relative benefits of a social network. Zoonotic pathogens are not carried by people, only by other animals. Their prevalence is less likely to affect the benefits of any particular social structure. Therefore, for the purposes of our analysis, we will group human-specific and multi-host diseases together. For example, using the 1930's data, we define $G_h \equiv FIL + LEI + LEP + TRY + MAL + DEN + TB$. We compare the effects of these human- and multi-reservoir diseases to those of zoonotic diseases. In the historical data, the two zoonotic diseases are $G_z \equiv SCH + TYP$. With contemporaneous data, we construct similar sums. The variable G_h is the sum of 22 human and multi-host diseases and G_z is the sum of 12 diseases.

[6] See e.g., Smith, Sax, Gaines, Guernier, and Gugan (2007) or Thornhill, Fincher, Murray, and Schaller (2010).

Using the disease prevalence data from each era separately, we construct the following two differences to use as instrumental variables:

$$\text{diff\_germ} \equiv G_h - G_z \tag{1}$$

$$\text{diff\_germ\_std} \equiv \frac{G_h}{std(G_h)} - \frac{G_z}{std(G_z)} \tag{2}$$

3.2 Measuring Collectivism

In our model, collectivism is defined as a social pattern of closely linked or interdependent individuals. What distinguishes collectives from sets of people with random ties to each other is that in collectives, it is common that two friends have a third friend in common. This is the sense in which they are interdependent.

The ideal data to measure collectivism would be each country's complete social network. We would look for a high prevalence of social collectives. There are a handful of studies that map out partial social networks, but only for small geographic areas, across eight countries. (See Fischer and Shavit (1995) for a review.) Therefore, we use data from Hofstede (2001) that is available for 72 countries. In 1970, he surveyed IBM employees worldwide to find national differences in cultural values. Hofstede performed a factor analysis of the survey responses, and found two factors that together can explain 46% of the variance in survey responses. He labels one factor "Collectivism vs Individualism", and uses it to construct an index of individualism that ranges from between 0 (strongly collectivist) to 100 (strongly individualist). Hofstede describes collectivist and individualist societies as follows: "on the individualist side we find societies in which the ties between individuals are loose... On the collectivist side, we find societies in which people from birth onwards are integrated into strong, cohesive in-groups, often extended families..." This description reflects two views of a collectivist society: one where ties are strong, and one where ties are shared.

In a widely cited paper, Granovetter (1973) provides the bridge between shared ties and strong ones; he argues, "the stronger the tie between A and B, the larger the proportion of individuals [that either of them knows] to whom they will both be tied." Granovetter goes on to give three theoretical reasons to believe this is true: (1) Time. If A and B have strong ties, they will spend a lot of time together. If A and C also have strong ties, they will also spend a lot of time together. If these events are independent or positively correlated, this necessarily implies B and C will spend a lot of time together, giving them a chance to form a strong tie. (2) The tendency of an individual to interact with others like himself. If A and B have strong ties, chances are good that they are similar; the same holds for A and C. Transitivity implies B and C will be similar, and will therefore get along. (3) The theory of cognitive balance. If A is good friends with B and C, then

B will want to develop a good relationship with C, in order to maintain his relationship with A. Thus, Granovetter's theory explains why Hofstede's survey questions, many of which are about the strength of social ties, are informative about the prevalence of collectives, as defined in the model.

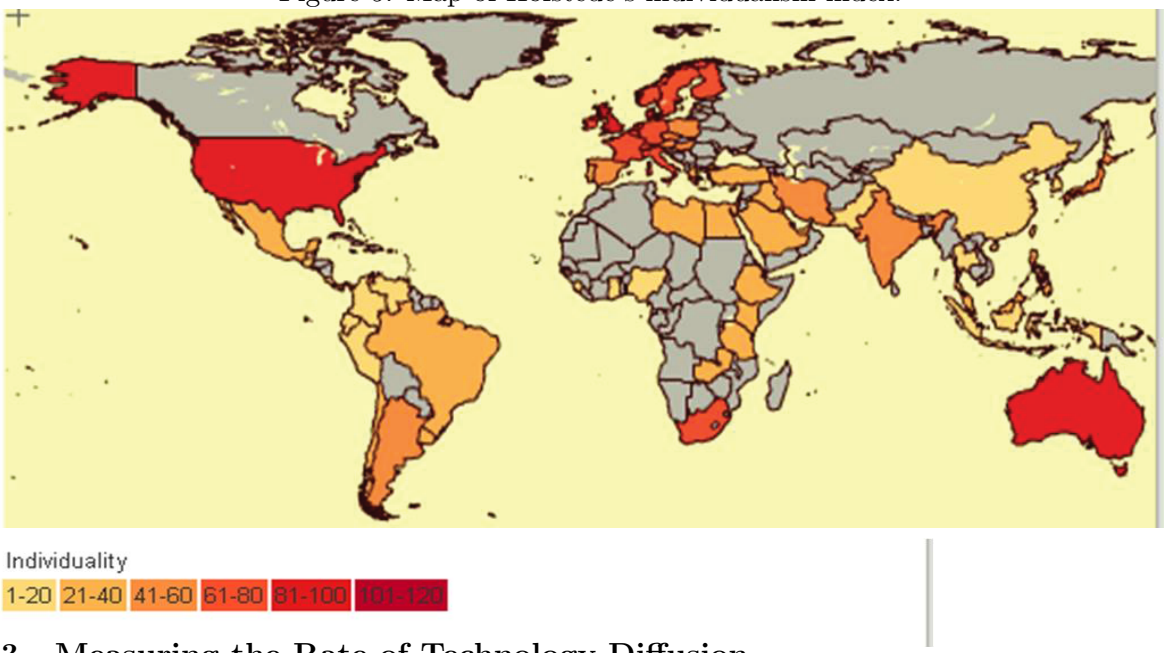
Other questions in Hofstede's survey assess the strength of cooperation, social influence and individuals' weight on social objectives. One example of such a question is " How important is it to you to work with people who cooperate well with each other?" Coleman (1988) explains why cooperative behavior is also linked to the presence of network collectives. He shows that effective norms depend on the presence of collectives because people enforce norms through collective punishments of deviators. If j observes i deviating from a social norm, then j can directly contact other friends of i to enact some joint retribution for the misdeed. When collective punishments are implementable, cooperation and conforming behavior is easier to sustain than if punishments must be implemented in an uncoordinated way.

A third category of questions in Hofstede's survey are about mobility, specifically one's willingness to move or change jobs. The essence of strong social ties is that the people involved are averse to breaking those ties. Thus an unwillingness to change one's social environment is indicative of strong social network ties. In the survey, the individualism index loads positively on one's willingness to move, which is consistent with the interpretations of individualism as a society with fewer collective and thus weak ties.

Thus, while Hofstede's survey asks questions that are not directly about the pattern of social relationships, there is a body of sociological theory and evidence that supports the connection between the behaviors that Hofstede asks about and the pattern of network collectives as described in our model. This connection is bolstered by the findings of the studies that do explicitly map out social networks among a subset of the population in local areas. Table 6 in the Appendix shows that highly individualist countries have lower network interdependence than more collectivist ones. Finally, other variants of the model that capture other aspects of collectivism, such as strong ties or fixed versus random networks, deliver the same effects. Networks with many weak ties, with random link formation, or with mobility, all have that ability to disseminate information or diseases more efficiently than their collectivist network counterparts (see Jackson (2008)). Appendix B contains more details about these alternative models and about the survey questions and other correlated social survey measures that shed light on the interpretation of Hofstede's index.

Figure 5 summarizes the findings of Hofstede's survey in a color-coded map. The most individualist countries (with an index between 80 to 91) are the Netherlands, Canada, Hungary, the United Kingdom, Australia and the United States. The most collectivist countries (with an index between 6 and 14) are Guatemala, Ecuador, Panama, Venezuela, Colombia, Pakistan, and Indonesia.

Figure 5: Map of Hofstede's individualism index.



3.3 Measuring the Rate of Technology Diffusion

We use a technology diffusion measure that is derived from the cross-country historical adoption of technology data set developed by Comin, Hobijn, and Rovito (2006). The data covers the diffusion of about 115 technologies in over 150 countries during the last 200 years. At a country level, there are two margins of technology adoption: the "extensive" margin (whether or not a technology is adopted at all) and the "intensive" margin (how quickly a technology diffuses, given that it is adopted.) A country can be behind in a technology even though it is adopting it quickly, if the technology was introduced to the country late.

Since our model speaks only to the diffusion rate of a technology, i.e. its intensive margin of adoption, we need to filter the extensive margin from the data. We do this with the results from Comin and Mestieri (2012), where attention is restricted to 15 technologies. Technical details are in that paper, but the idea is the following: For a given country, plotting the normalized level of a given technology (e.g. log telephone usage minus log country income) over time yields an increasing curve. For a given technology, these curves look similar across countries, except for horizontal and vertical shifts. The horizontal shifts correspond to the extensive margin of technology adoption; if country A adopts telephones in exactly the same way as country B, only twenty years later, its curve will be identical to that of B except shifted twenty years to the right. However, if country A adopts telephones less vigorously but at the same time, its curve will be below that of B's. This diffusion rate of technology is what we are interested in, so it is what we focus on. Specifically, Comin and Mestieri (2012) estimate the slope of a non-linear diffusion curve. A higher slope parameter m_{ij}

indicates a faster diffusion rate of technology j in country i.

Our ideal measure of the technological level of a country would be its average diffusion rate from all 15 technologies. A complication is that the data set is unbalanced; if data for a country is only available for slowly-spreading technologies, it might artificially appear technologically backward. To control for this problem, we estimate $m_{ij} = \alpha_j + e_{ij}$, where α_j is a technology-specific fixed effect. Our measure of technology diffusion for a given country is the average residual diffusion$_i = \sum_j e_{ij}$.

4 Empirical Results: How Much Do Networks Affect Technology?

Our objective is to better understand how social structure affects technology diffusion and economic development. The difficulty is that economic development also can potentially change the social structure. The challenge is to isolate each of these two effects. To do this, we consider the following structural model:

$$A = \beta_1 + \beta_2 S + \epsilon \tag{3}$$

where A is the speed of technology diffusion, S is social structure (individualism), as measured by the Hofstede index, the β's are unknown coefficients and ϵ is a mean-zero residual orthogonal to S. Social structure is

$$S = \gamma_1 + \gamma_2 A + \gamma_3 G_h + \gamma_4 G_z + \eta, \tag{4}$$

where the γ's are unknown coefficients, G_h and G_z are human and zoonotic disease prevalences, and η is a mean-zero residual orthogonal to A, G_h and G_z. The coefficient of interest is β_2, which measures the effect of social structure S on technology diffusion A.

This model recognizes the endogeneity problem inherent in estimating the relationship between A and S. It incorporates our main hypothesis, that social structure S matters for technology A, but it also reflects the idea that perhaps technology (and income) can cause social structure to change as well. Because A depends on S and S depends on A, an OLS estimate would be biased.

Our theory suggests that an instrument with power to predict social structure S is disease prevalence $G_h + G_z$. But, this is not likely to be a valid instrument both because technology affects disease (vaccines are a technology, for example) and because poor health reduces productivity and diminishes one's capacity for invention. We capture the correlation between disease prevalence and technology, from both directions of causality, in the following relationship, which says that, after controlling for social structure, there is a residual correlation between technology and disease:

$$\epsilon = \delta_1 + \delta_2(G_h + G_z) + \xi. \tag{5}$$

If $E[\epsilon(G_h + G_z)] \neq 0$, in other words, if $\delta_2 \neq 0$, then disease prevalence is an invalid instrument.

To resolve this problem, we use the difference in human disease prevalence and zoonotic disease prevalence $(G_h - G_z)$ as our instrument. When $var(G_h) = var(G_z)$, the difference $(G_h - G_z)$ is orthogonal to the sum $(G_h + G_z)$. Therefore, in our final exercise, we scale G_z to give it the same variance as G_h to ensure that the orthogonality holds. Thus, our identifying assumption is

$$E[\xi(G_h - G_z)] = 0.$$

Since in Equation 5 we restrict the coefficients on G_h and G_z to be the same, we assume that human disease prevalence and zoonotic disease prevalence have the same effect on technology. Hence the total effect on technology is determined by the sum $G_h + G_z$. This is orthogonal to the composition of the effect between the two types of disease, $G_h - G_z$, which has no direct effect on A. But as long as $\gamma_3 \neq \gamma_4$ in (4), then human and zoonotic diseases have different effects on social structure S. Therefore, since the diseases have different effects on social structure S and similar effects on the speed of technology diffusion A, the instrument $(G_h - G_z)$ can be a powerful and valid instrument.

Finally, note that we do not need to know all the determinants of social structure. Rather, any subset of the determining variables can serve as valid instruments for S. Similarly, we do not need to observe S exactly. A proxy variable with random measurement noise is sufficient for an unbiased instrumental variables estimate of the coefficient β_2.

4.1 First-Stage Regressions: Disease and Social Institutions

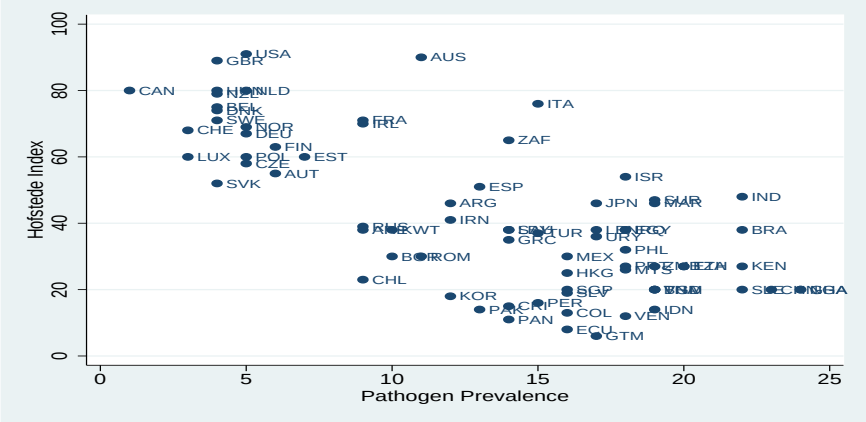
We begin by investigating the relationship between our instruments and our measure of social structure. There are two key findings: First, the instruments are powerful predictors of social structure. Second, disease $(G_h + G_z)$ is negatively correlated with individualism. Although this effect is not identified, the correlation is consistent with one key prediction of the evolutionary network model.

To illustrate the robustness of these results, we explore a handful of instrumental variable specifications. Most of the specifications have multiple instruments because that allows us to evaluate the validity of our instruments by testing the orthogonality of each instrument with the residual in equation (3). Following Hall and Jones (1999), we use two language-based variables as additional instruments to test the validity of our own disease-based instruments. The variable pronoun is a dummy variable that is equal to 1 if it is conventional to omit first- and second-person pronouns in a country's dominant spoken language (Kashima and Kashima, 1998). For example, English and German typically do not omit pronouns, while Spanish does. In addition, we use a

variable for the fraction of the population speaking English as a first language.[7] Including English specifically contributes additional explanatory power. Because these variables are language-based, they are a product of the country's distant past and possibly its colonial heritage. As Hall and Jones (1999) argue, they are unlikely to be affected by current income or technology.

We begin by exploring the data on individualism and disease prevalence. Figure 6 illustrates the clear, negative relationship between our measure of social structure, the Hofstede index, and the sum of the prevalence of all nine pathogens in our historical disease data set. The negative

Figure 6: Hofstede's individualism index plotted against total pathogen prevalence. Total pathogen prevalence is *human* + *zoonotic*. This is a sum of the prevalence of all nine diseases described in section 3.



relationship is consistent with our theory, in which greater disease prevalence favors the emergence of a collectivist social structure. Even though collectivism itself inhibits the spread of disease, the net effect in the model is that high pathogen prevalence is correlated with collectivism (see figure 4). Since collectivist societies are ones with a low Hofstede index, the model and data both generate a negative relationship between pathogens and individualism.

Table 2 quantifies this relationship. Column 1 shows that pathogen prevalence and individualism are negatively related in a statistically significant way. The explanatory power of pathogens is large; the R^2 of the regression is over 50%. The economic magnitudes are also large. A one-unit increase in our historical pathogen measure corresponds to one disease being endemic instead of sporadic. Having one more socially transmittable human disease consistently prevalent corresponds to an individualism index that is 3.46 points lower (14% of a standard deviation).

In addition, both our language variables and the difference in disease prevalence are highly-significant predictors of social structure (table 2, column 3). Disease difference is a powerful instrument because the average correlation of individualism with each disease carried by humans is

[7]The English variable is available from the Penn World Tables, Mark 5.6.

Table 2: **First-stage regressions of pathogen prevalence variables on individualism index**

Dependent variable	Hofstede's individualism index (S)							
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Historical				Contemporary			
Total pathogens	−2.73				−2.72			
	(0.31)				(0.32)			
Human - zoonotic		-3.46	-2.15			-3.77	-2.38	
pathogens (diff_germ)		(0.44)	(0.45)			(0.57)	(0.48)	
Human - zoonotic				-5.26				-7.12
pathogens (diff_germ_std)				(2.04)				(2.90)
English			25.33	28.48			23.05	24.90
			(7.50)	(8.58)			(7.42)	(8.43)
Pronoun			-19.17	-28.33			-23.14	-30.02
			(4.83)	(4.70)			(4.35)	(4.57)
Constant	77.10	67.53	69.71	59.86	77.90	127.0	220.5	69.46
R^2	0.52	0.47	0.71	0.64	0.51	0.38	0.72	0.64
Observations	72	72	62	62	71	72	62	62

The table reports OLS estimates of the γ coefficients in $S = \gamma_1 + \gamma_2 A + \gamma_3 G_h + \gamma_4 G_z + \eta$. The variables diff_germ and diff_germ_std are defined in equations (1) and (2). Columns (1)-(4) use historical disease prevalence data from the 1930s. Columns (5) -(8) use a more extensive set of diseases, measured in 2005. The other instruments are pronoun drop and whether is English spoken (see appendix B). Standard errors are in parentheses. All coefficients are significant at the 5% level.

much larger in magnitude than the average correlation with each of the zoonotic diseases (-0.53 vs. -0.29). The fact that the correlations are negative tells us again that higher disease prevalence is associated with more collectivist societies.

These results are important for the next stage, identifying an effect of institutions on technology diffusion. But they are also interesting on their own because they are consistent with one reason why countries may have adopted different social institutions. Perhaps social structures have evolved, in part, as a defense against the spread of directly-communicable diseases. But further statistical work needs to be done to say conclusively that disease prevalence is part of the reason why some societies have adopted social structures that inhibit technological diffusion and growth.

4.2 Concerns about instrument exogeneity

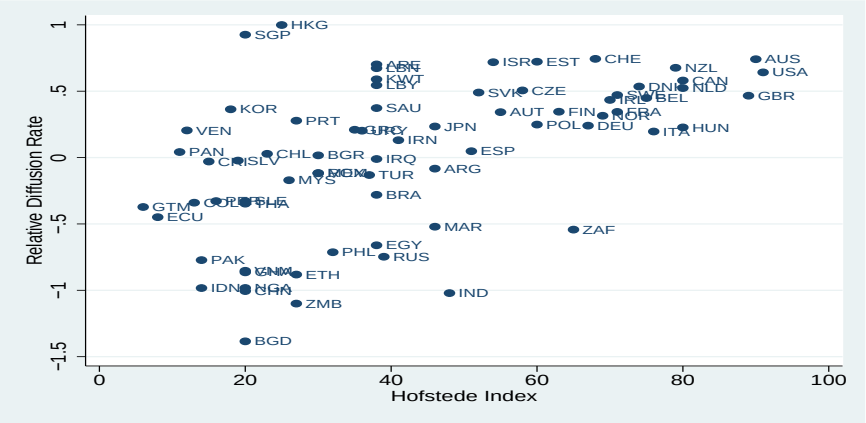
Our identifying assumption is that while technology diffusion and GDP may affect disease prevalence, even 40 years prior, it affects many diseases similarly. Thus, the difference in the prevalence of two types of disease is exogenous with respect to GDP. The difference we consider is the difference between diseases that reside in humans (human-specific plus multi-host) and diseases that reside exclusively in non-human animals (zoonotic diseases).

Unequal variance One concern with this instrument might be that the difference between disease prevalence rates might not be orthogonal to the sum. For example, if zoonotic disease had (hypothetically) been eradicated in every country in our sample, then *diff_germ*$= G_h - G_z = G_h$. Since the prevalence of disease is likely to be correlated with income and technology diffusion, this situation would render *diff_germ* an invalid instrument. For two variables x and y, $(x+y)$ is uncorrelated with $(x-y)$ when x and y have equal variances. Our human and zoonotic disease variables do not have exactly the same variance. To ameliorate this concern, we also use *diff_germ_std* as an instrument in table 2 and find that it produces estimates of the importance of social structure that are even larger than the initial estimates.

Uneven effects of technology. Our empirical strategy is based on the assumptions that G_h and G_z have the same relationship with A but different relationships with S. One may think this relationship does not necessarily hold. For example, perhaps clean water initiatives are one of the first public health measures adopted when income rises. If this were the case, then there would be a negative correlation between zoonotic illness and technology diffusion, and therefore a positive correlation between (human - zoonotic) diseases x and shocks to technology diffusion ϵ. If $E[\epsilon x] > 0$, how would this bias the results? A positive shock to income (high ϵ) would increase the difference in disease (x), which would decrease individualism S (since we estimate $\gamma_3 < 0$). This would induce negative correlation between A and S, which would lower the estimated coefficient β_2 in equation 3. So β_2 would be downward biased. Thus, if the instrument is invalid because economic development primarily reduces water-borne illnesses, then the true size of social structure's effect on technology diffusion is even larger than what we estimate.

Social structure affects disease. The other hypothetical cause for concern might be that faster technology diffusion and the accompanying higher income cause the social structure to change. In particular, a richer, more modern society is more likely to be market-based and individualist. The change in social social structure could affect the difference in disease prevalence by facilitating the transmission of diseases spread from human-to-human. Notice that this logic does not imply that differences in disease x are correlated with the estimation error ϵ in (3). This story suggests that social structure S depends on A, something already represented in our specification (equation 4), and it suggests that there should be an additional equation representing the idea that the instrument x depends on social structure: $x = \psi_1 + \psi_2 S + \nu$. In this structure, as long as $e[\epsilon\nu] = 0$, x is still a valid instrument for S. In other words, as long as technology diffusion affects the difference in disease through social structure, rather than directly, this form of reverse causality *does not invalidate the use of disease differences as instruments.* It only implies that γ_3 is perhaps not an

Figure 7: Technology and individualism. Comin and Mestieri (2012)'s technology diffusion measure (vertical axis) plotted against Hofstede's individualism index (horizontal axis).



unbiased estimator of the effect of disease on social institutions. Our estimates suggest that more disease is associated with less individualism. If individualism spreads disease, then this estimate is downwards-biased. In other words, the true effect of disease on social institutions would be larger than the one we estimate.

4.3 Main Results: Social Institutions and Technology Diffusion

Our main result is to quantify the effect of social structure on technology diffusion. Figure 7 illustrates the relationship between social structure and the speed of technology diffusion in a scatter plot. It reveals that more individualist societies tend to also be societies where technologies diffuse quickly. In interpreting this correlation, reverse causality is obviously a concern. Faster technology diffusion raises incomes, which might well change the social structure. Likewise, the economic development that results from technology diffusion could produce a wave of urbanization, which influences social structure. Therefore, we use the differences in pathogen prevalence as an instrument for social structure.

The first two columns of table 3 show that the degree of individualism in a country's social structure has a large effect on a country's rate of technology diffusion. A 1-standard deviation in the Hofstede index is 28.5. When we use *diff_germ_std* as an instrument, a 1-standard deviation increase in individualism results in $28.5 \cdot 1.31 = 37.3\%$ increase in the speed of technology diffusion. The mean of the diffusion variable is near zero so this is not easily interpretable relative to its mean. But its standard deviation is 63.4%. Thus, a degree of individualism that is 1 standard deviation above the average is associated with technology diffusion that is 59% of a standard deviation higher than average. Across specifications, the estimates of the effect of social structure are remarkably stable. Individualism consistently explains 27-28% of the variation in technology diffusion rates.

Table 3: **Social Structure and Technology Diffusion (main result)**

Dependent variable:	Technology Diffusion Rate				
Disease Instrument:	diff_germ	diff_germ_std	diff_germ	diff_germ_std	none
	Historical		Contemporary		(OLS)
Individualism	1.63	1.31	1.62	1.31	1.40
	(0.33)	(0.34)	(0.33)	(0.34)	(0.28)
Over-ID p-val	0.12	0.77	0.11	0.74	
	Accept	Accept	Accept	Accept	
R^2	0.27	0.28	0.27	0.28	0.27
N	62	62	62	62	72

The first row reports $100 * \beta_2$ coefficient from an IV estimation of $A = \beta_1 + \beta_2 S + \epsilon$. Technology diffusion rate (A) comes from the Comin and Mestieri (2012) measure of the intensive technology adoption in a country. Individualism S is the Hofstede index. The variables *diff_germ*, *diff_germ_std* are defined in equations (1) and (2). Each estimation, except OLS, also uses *pronoun* and *english* as instruments, as defined in table 1. The over-ID test is a Sargan test statistic. The null hypothesis is that the instruments are uncorrelated with ϵ. Accept means that null hypothesis cannot be rejected at the 5% or even the 10% confidence level. All coefficients are significant at the 5% level.

The Sargan test statistics (in the row labeled over-ID) are chi-square statistics for the test of the null hypothesis that the instruments are uncorrelated with the regression residual ϵ. For every IV specification, we cannot reject this null hypothesis at the 5% or even the 10% level. However, we could reject the null hypothesis at a 15% confidence level in the estimation in columns (1) or (3). This suggests that the *diff_germ* variable is unlikely to be a valid instrument. Note that when we use the standardized *diff_germ_std* variable as an instrument, the p-value rises to 77%, suggesting that the instrument is likely to be uncorrelated with the regression residual. We also computed Basmann statistics. They were quite close in value to the Sargan statistics in every instance.

Controlling for other possible explanatory variables. A natural question is whether social structure is simply a proxy for some other economic variable. To assess this, we choose a variety of other variables thought to explain technology adoption or income and control for their effects too. In doing so, we recognize that these control variables may themselves be endogenous. Inferring causality from these results would therefore be problematic. However, we continue to use *diff_germ_std*, *pronoun* and *english* as instruments and add the following variables, one-by-one, to the first- and second-stage estimations:[8] Controlling for life expectancy at birth, which could capture a direct effect of pathogens on technology diffusion, or social infrastructure, which could promote technology diffusion and discourage disease, reduces the size of the coefficient on individualism by a factor of roughly 1/2. The other control variables we try are: (1) ethnic-linguistic

[8]Our procedure and our choice of variables here largely follow Hall and Jones (1999). The variable "social infrastructure" is constructed by Hall and Jones to measure the quality of institutions.

fractionalization (the probability that two randomly matched people belong to different ethnic or linguistic groups), which could affect both social structure and the diffusion rate of technology, (2) latitude, which is likely to be correlated with the epidemiological environment, (3) disease-adjusted life expectancy, to capture the direct effect of health on technology, (4) a country's degree of capitalism or socialism, which could be highly correlated with social structure and which probably affects incentives for technology adoption, and (5) population density, which affects disease, social structure and technology diffusion. These all leave the estimate of the effect of individualism largely unchanged. Appendix B reports the complete set of results for each of these estimations. In sum, there is a statistical relationship between social structure and technology diffusion that is above and beyond that which comes from other commonly-used determinants of income.

Effect of social structure on productivity and income. To interpret these results economically, it is helpful to re-estimate the effect of social structure with dependent variables that are more familiar to macroeconomists: the Solow residual and output per worker, again instrumenting individualism with the language variables and differences in diseases. The coefficients in Table 4 tell us that a 1-standard-deviation increase in the Hofstede index corresponds to a 23 unit increase in productivity. Since the Solow residual is measured as a fraction of its value for the US, this increase is 23% of the US value of productivity. For output per worker, the effects are even larger. A 1-standard deviation increase in individualism increases output per worker by 48 or 50, which represents an increase of 48-50% of US output per worker, depending on the set of instruments.

Table 4: **Social Structure, Productivity and Income**

Dependent variable:	Solow Residual		Output per capita	
Disease Difference Instrument:	Historical	Contemperanous	Historical	Contemperanous
Other Instruments:	pronoun, english	pronoun, english	pronoun, english	pronoun, english
Individualism	0.99	0.78	2.10	1.84
	(0.40)	(0.40)	(0.45)	(0.45)
R^2	0.20	0.18	0.42	0.41
N	58	58	59	59

The first row reports $100 * \beta_2$ coefficient from an IV estimation of $a = \beta_1 + \beta_2 S + \epsilon$, where a is the Solow residual or output (GDP) per capita. Solow residual and output per capita come from the Penn World Tables mark 5.6. The disease instruments are standardized differences, as in (2). Other variables are described in table 3. All estimates are significant at 5% level.

To get a more concrete idea of what these numbers imply, consider the case of individualistic Finland ($I = 63$) and collectivist Ghana ($I = 20$). The difference in their Hofstede individualism index is $63 - 20 = 43$, while the difference in their technology diffusion rates A is 1.2.. From Table 3,

the difference in the Hofstede index between the two countries explains $43 * .0131 = .56$ - just under half - of the total difference in A. Similarly, the difference in output per capita between Finland and Ghana is 2.6. From Table 4 the difference in I explains $43 * 0.021 = .90$ of this difference, which is over one-third of the per capita output gap.

4.4 Could Social Structure Really Change in Response to Disease?

The idea that people might choose their social circles based on disease avoidance might sound far-fetched. But researchers in animal behavior have long known that other species choose their mates with health considerations in mind (Hamilton and Zuk, 1982). Furthermore, primate research has shown that the animals most similar to human beings behave similarly to the agents in our model. Their mating strategies, group sizes, social avoidance and barriers between groups are all influenced by the presence of socially transmissible pathogens (Loehle, 1995).

One might also question whether historical societies knew enough about contagion to make informed choices about social networks. Yet, historical documents reveal a reasonable understanding of epidemiology. For example, in the sixteenth century, when smallpox reached the Americas and became a global phenomenon, people understood that the skin lesions and scabs that accompany smallpox could transmit the disease. They knew that survivors of smallpox and other infections were immune to re-infection. The practice of inoculation, whereby people were intentionally exposed to disease was practiced hundreds of years ago in China, Africa and India. Similarly, the plague was recognized to be contagious. Therefore, control measures focused primarily on quarantine and disposal of dead bodies. Even two thousand years ago, in biblical times, leprosy was understood to be contagious. Lepers, or suspected lepers, were forced to carry a bell to warn others that they were coming. Thus, the idea that one should avoid contact with others who carry particular contagious diseases is not just a modern idea.

5 Conclusions

Measuring the effect of social network structure on the economic development of countries is a challenging task. Social structure is difficult to measure and susceptible to problems with reverse causality. We use a theory of social network evolution to identify properties of social networks that can be matched with data and to select promising instrumental variables that can predict network structure. The theory predicts that societies with higher disease prevalence are more likely to become collectivist: their social networks will have dense connections within a group, but few connections to non-group members. Such networks inhibit disease transmission, but they also inhibit idea transmission. This model guides us to choose sociological measures of individualism

and collectivism to measure the prevalence of collectives in social networks. It also suggests that disease prevalence might be a useful instrument for a social network because it is one important concern that societies incorporate when they choose their network.

Quantifying the model reveals that small initial differences in the epidemiological environment can give rise to large differences in network structure that persist. Over time, these persistent network differences can generate substantial divergence in technology diffusion and output. We find evidence of this social network effect in the data. Exploiting the differential mode of transmission of germs, we are able to identify the significant effect of social structure on technology diffusion and income. More broadly, the paper's contribution is to offer a theory of the origins of social institutions, propose one way these institutions might interact with the macroeconomy, and show how to quantify and test this relationship.

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A Proofs of Propositions

Proof of result 1 *In a collectivist network, where $\gamma = 4$, there are n unique collectives.*

Claim 1: Any three adjacent nodes are a collective.

Proof: Consider nodes j, $j+1$ and $j+2$. Since every node is connected to its adjacent nodes, $j+1$ is connected to j and $j+2$. And since every node is also connected to nodes 2 places away, j is connected to $j+2$. Since all 3 nodes are connected to each other, this is a collective.

Claim 2: Any sets of 3 nodes that are not 3 adjacent nodes are not a collective.

Proof: Consider a set of 3 nodes. If the nodes are not adjacent, then two of the nodes must be more than 2 places away from each other. Since in a collectivist network with $\gamma = 4$, nodes are only connected with other nodes that are 2 or fewer places away, these nodes must not be connected. Therefore, this is not a collective.

Thus, there are n unique sets of 3 adjacent nodes (for each j there is one set of 3 nodes centered around j: $\{j-1, j, j+1\}$). Since every set of 3 adjacent nodes is a collective and there are no other collectives, there are n collectives in the network. □

Proof of result 2 *In an individualistic network, where each person i is connected to $i-\psi$, $i-1$, $i+1$, and $i+\psi$, where $\psi > 2$, there are zero collectives.*

Proof: Consider each node connected to an arbitrary i, and whether it is connected to another node, which is itself connected to i. In addition to being connected to i, node $i-\psi$ is connected to $i-2\psi$, $i-\psi-1$, and $i-\psi+1$. None of these is connected to i. Node $i-1$ is also connected to $i-2$, $i-\psi-1$ and $i+\psi-1$. But none of these is connected to i. Node $i+1$ is also connected to $i+2$, $i-\psi+1$ and $i+\psi+1$. But none of these is connected to i. Finally, node $i-\psi$ is also connected to $i+\psi-1$, $i+\psi+1$ and $i+2\psi$. But none of these is connected to i. Therefore, there are no collectives among any connections of any arbitrary node i. □

Diameter of network 1. Proof: Without loss of generality, consider the agent in the last position, the agent with location n on the circle.*Case 1: n even.* If n is even, then the farthest node from n is $n/2$. If each person is connected to the γ closest people, where γ is even, then they are connected to $\gamma/2$ people on either side. Therefore, the shortest path will be the one that advances $\gamma/2$ places around the circle, at each step in the path, until it is within $\gamma/2$ nodes of its end point. For example, agent n reach $\gamma/2$ in one step, γ in two steps and $n/2$ in $(n/2)/(\gamma/2) = n/\gamma$ steps, if n/γ is an integer. If dividing n by γ leaves a remainder m, then one step in the path to reach $n/2$ must

be only $m < n/2$ nodes away. Thus, when n is even, the shortest path to the furthest node $n/2$ is $ceil(n/\gamma)$, where $ceil(x) = x$ if x is an integer, and is otherwise, the next largest integer.

Case 2: n odd. If n is odd, then $(n-1)/2$ and $(n+1)/2$ are equally far from node n. Each is $(n-1)/2$ nodes away. Following the same logic as before, the shortest path will be the one that advances $\gamma/2$ places around the circle, and reaches the furthest node in $ceil((n-1)/2)/(\gamma/2) = ceil((n-1)/\gamma)$ steps.

Lastly, note that when n is even, $ceil(n/\gamma) = ceil((n-1)/\gamma)$. Note that, since $\gamma > 1$ and both γ and n are integers, $ceil(n/\gamma)$ and $ceil((n-1)/\gamma)$ will only differ if $(n-1)/\gamma$ is an integer, so that adding $1/\gamma$ to it will make $ceil(n/\gamma)$ the next largest integer. But if γ is even and $(n-1)/\gamma$ is an integer, then $n-1$ must be even, which makes n odd. Thus, $ceil(n/\gamma) = ceil((n-1)/\gamma)$.□

Average path length in network 1. Proof: Without loss of generality, consider the distance from the last node, n. n can be connected to nodes 1 though $\gamma/2$ and $n-1$ through $n-\gamma/2$ in 1 step. More generally, it can be connected to nodes $(s-1)\gamma/2+1$ through $s\gamma/2$ and $n-(s-1)\gamma/2-1$ through $n-s\gamma/2$, in s steps. For each s, there are γ nodes for which the shortest path length to n is s steps. We know from result 1 that when γ is even and n/γ is an integer, the longest path length (the diameter) is n/γ. Thus, the average length of the path from n to any other node is $1/n\sum_{s=1}^{n/\gamma}\gamma s$. Using the summation formula, this is $(\gamma/n)(n/\gamma)(n/\gamma+1)/2 = 1/2 + n/(2\gamma)$. □

Diameter of network 2. *The diameter of an individualistic network, with $n > 4$ nodes where each node i is connected to $i-4$, $i-1$, $i+1$, and $i+4$, is round$(n/8)+1$.*

Proof: Without loss of generality, consider distances from the agent located at node n. n can reach nodes 1, 4, $n-1$ and $n-4$ in one step. It can reach nodes 2, 3, 5, 8 and $n-2$, $n-3$, $n-5$ and $n-8$ in two steps. In any number of steps $s > 1$, agent n can reach nodes $4(s-2)+2, 4(s-1)-1, 4(s-1)+1, 4s$ (moving clockwise around the circle) as well as $n-4(s-2)-2, n-4(s-1)+1, n-4(s-1)-1, n-4s$ (moving counter-clockwise).

Let the operator $floor(x)$ be the largest integer y such that $y \leq x$. Define $\tilde{n} \equiv 4 * floor(n/8)$. Then $\tilde{r} \equiv n - 2 * \tilde{n}$ is the remainder when n is divided by 8. There are eight cases to consider, one for each possible value of $\tilde{r}$.

Case 1: $\tilde{r} = 0$**.** If the total number of nodes in the network n is a multiple of 8, then it takes $(1/4) * n/2$ steps to connect node n with node $n/2$, the geographically farthest node in the network. But it takes one more step to reach $n/2-1$, $n/2+1$. The nodes $n/2-2$ and $n/2+2$ can be reached in 2 steps from $n/2-4$ and $n/2+4$, each of which is one step closer to n than $n/2$ is. Thus, every node can be reached in $n/8+1$ steps, making the diameter of the network $n/8+1$.

Case 2: $\tilde{r} = 1$**.** In this case, $\tilde{n}$ and $\tilde{n}+1$ are equally far away from n in the network. Each requires $\tilde{n}/4$ steps. But it takes one more step to reach $\tilde{n}-1$, $\tilde{n}-2$, $\tilde{n}+2$ or $\tilde{n}+3$. Since $\tilde{n} = 4floor(n/8)$, $\tilde{n}/4 = floor(n/8)$, and thus the diameter is one step more than that, which is $floor(n/8)+1$.

Case 3: $\tilde{r} = 2$**.** In this case, $\tilde{n}$ and $\tilde{n}+2$ are equally far away from n in the network. Each requires $\tilde{n}/4$ steps. But it takes one more step to reach $\tilde{n}-1$, $\tilde{n}-2$, $\tilde{n}+1$, $\tilde{n}+3$ or $\tilde{n}+4$. Thus, the diameter is again $floor(n/8)+1$.

Case 4: $\tilde{r} = 3$**.** In this case, $\tilde{n}$ and $\tilde{n}+3$ are equally far away from n in the network. Each requires $\tilde{n}/4$ steps to reach. It is still the case that it takes one more step to reach $\tilde{n}-1$, $\tilde{n}-2$ and $\tilde{n}+1$. $\tilde{n}+2$ can be reached in one additional step from $\tilde{n}+3$, as can $\tilde{n}+4$. And $\tilde{n}+5$ can be reached in 2 additional steps from $\tilde{n}+4$, which is one step closer to n than $\tilde{n}+3$. Thus, every node can still be reached in $floor(n/8)+1$ steps.

Case 5: $\tilde{r} = 4$**.** In this case, $\tilde{n}$ and $\tilde{n}+4$ are equally far away from n in the network. Each requires $\tilde{n}/4$ steps to reach. But now, getting to $\tilde{n}+2$ requires 2 additional steps. Thus, the diameter of this network is $floor(n/8)+2$.

Case 6: $\tilde{r} = 5$**.** In this case, $\tilde{n}$ and $\tilde{n}+5$ are equally far away from n in the network. Each requires $\tilde{n}/4$ steps to reach. Getting to either $\tilde{n}+2$ or $\tilde{n}+3$ requires 2 additional steps. Thus, the diameter of this network is $floor(n/8)+2$.

Case 7: $\tilde{r} = 6$**.** In this case, $\tilde{n}$ and $\tilde{n}+6$ are equally far away from n in the network. Each requires $\tilde{n}/4$ steps to reach. In one additional step, one can connect from $\tilde{n}$ to $\tilde{n}+1$ or $\tilde{n}+4$ or from $\tilde{n}+6$ to $\tilde{n}+2$ or $\tilde{n}+5$. It takes two additional steps from $\tilde{n}$ to connect to $\tilde{n}+3$. Thus, the diameter of this network is $floor(n/8)+2$.

Case 8: $\tilde{r} = 7$**.** In this case, $\tilde{n}$ and $\tilde{n}+7$ are equally far away from n in the network. Each requires $\tilde{n}/4$ steps to reach. In one additional step, one can connect from $\tilde{n}$ to $\tilde{n}+1$ or $\tilde{n}+4$ or from $\tilde{n}+7$ to $\tilde{n}+3$ or $\tilde{n}+6$. It takes two additional steps from either $\tilde{n}$ or $\tilde{n}+7$ to connect to $\tilde{n}+2$ or $\tilde{n}+5$. Thus, the diameter of this network is $floor(n/8)+2$.

The one condition that encapsulates all 8 of these cases is diameter=round$(n/8)+1$. To see this, recall that $\tilde{r}$ is the remainder when n is divided by 8. When this remainder is zero, then $(n/8)+1$ =round$(n/8)+1$. When this remainder is less than 4, then floor$(n/8)+1$ =round$(n/8)+1$. When this remainder is 4 or more (4-7), then round$(n/8)$ =floor$(n/8)+1$, and therefore floor$(n/8)+2$ =round$(n/8)+1$. Thus, in each case of the 8 cases, the diameter of the network is equal to round$(n/8)+1$.□

Average path length of network 2. *In the example individualistic network, when $n/8$ is an integer, the average path length is $7/8+n/16$. This is less than the average path length in a collectivist network with $\gamma = 4$, when the network is large ($n > 6$).*

Proof: Without loss of generality, consider distances of each node from node n. n can reach 4 different nodes: 1, 4, $n-1$ and $n-4$ in one step. It can reach 8 different nodes 2, 3, 5, 8 and $n-2$, $n-3$, $n-5$ and $n-8$ in two steps. More generally, for a number of steps $s \geq 2$, agent n can reach 8 new nodes with each step. These nodes are: $4(s-2)+2, 4(s-1)-1, 4(s-1)+1, 4s$ (moving clockwise around the circle) as well as $n-4(s-2)-2, n-4(s-1)+1, n-4(s-1)-1, n-4s$ (moving counter-clockwise). This rule holds until the number of steps s reaches $n/8$, the number of steps to travel approximately half way around the circle. At that point, the number of additional nodes that can be reached in an additional step depends on the size of the network. There are 8 cases to consider.

Recall that $\tilde{n} \equiv 4 * floor(n/8)$ and that $\tilde{r} \equiv n - 2 * \tilde{n}$ is the remainder when n is divided by 8. There are eight cases to consider, one for each possible value of $\tilde{r}$.

If the total number of nodes in the network n is a multiple of 8, then it takes $n/8$ steps to connect node n with node $n/2$. Using the algorithm above, it also takes $n/8$ steps to connect with nodes $n/2-6, n/2-5, n/2-3, n/2+6, n/2+5$ and $n/2+3$. But this is 7 total nodes instead of 8 total nodes because when the total number of steps being considered is $n/8$ ($s = n/8$) nodes $4s$ and $n-4s$ are both equal to node $n/2$.

It takes one more step to reach $n/2-1$, $n/2+1$. The nodes $n/2-2$ and $n/2+2$ can be reached in 2 steps from $n/2-4$ and $n/2+4$, each of which is one step closer to n than $n/2$ is. Thus, 4 additional nodes can be reached in $n/8+1$ steps.

Counting up, there is 1 node (n) reachable in zero steps, 4 nodes reachable in 1 step, 8 nodes reachable in s steps for $s \epsilon \{2, 3, \ldots, n/8-1\}$, 7 nodes reachable in $n/8$ steps and 4 nodes reachable in $n/8+1$ steps. That makes the average path length $1/n$ times the sum of all the path lengths to the n nodes: $1/n[4 + 8\sum_{s=2}^{n/8-1} s \ + 7 * n/8 + 4 * (n/8+1)]$. Applying the summation formula, $8\sum_{s=2}^{n/8-1} s = 8(n/8)(n/8-1)/2 - 8$, where the -8 corrects for the fact that the sum begins at $s = 2$, rather than at $s = 1$. Substituting in this formula and collecting terms, this is $1/n[4 + 8(n/8)(n/8-1)/2 - 8 + 11n/8 + 4] = 1/8n[n(n-8)/2 + 11n] = 7/8 + n/16$. □

Proof of result 3 *For a large network ($n > 8$) where $n/8$ is an integer, the individualistic network has a smaller diameter and a shorter average path length than a collectivist network with equal size n and equal degree $\gamma = 4$.*

Suppose $\psi_k(0) = 1$ for some k and $\psi_j(0) = 0 \, \forall \, j \neq k$. For a person living in location j, the sick person lives s_{jk} steps away. Since the probability of contagion is equal to 1, person j will be sick in s_{jk} periods and then die, i.e. $\Psi_j(0) = s_{jk}$. Averaging over all locations j, we have that the average lifetime is equal to the average path length from k to all other nodes: $E_j[\Psi_j(0)] = E_j[s_{jk}]$. For the maximum lifetime we have that $\max_j[\Psi_j(0)] = \max_j[s_{jk}] = diam(N)$; this is, the person whose location is furthest from k (diameter) will live the longest. Since $n > 8$ and $n/8$ is an integer, both the average path length and the diameter are longer for $N1$.

Analogously, suppose that a new idea is introduced by person k in period 0. Since the idea is transmitted with probability 1, the number of periods it takes to reach person j is given by $\alpha_j(0) = s_{jk}$. Thus the average discovery time is equal to the average path length from k to other nodes, $E_j[\alpha_j(0)] = E_j[s_{jk}]$, and the maximum discovery time $\max_j[\alpha_j(0)] = \max_j[s_{jk}] = diam(N)$. Thus the discovery process is slower in $(N1)$.

Proof of Result 4 A new technology shock advances the technological frontier if it arrives to an agent that has a technology level that is as high as any other agent in the network. Suppose that at t, the technology of each agent is the same in both types of networks and agent j (and only him[9]) is at the technological frontier. In the next period, with probability $1-(1-p)^4$, agent j transmits his technology to at least one of his connections and the expected number of people that have the latest technology in $t+1$ is $1+4p$. That probability is the same in both networks. Each agent has an identical probability λ of inventing a new technology. Thus, the probability that a technology shock hits an agent who has the highest technology level at $t+1$ and advances the frontier is $(1+4p)\lambda$, in either network.

Now consider time $t+2$. In expectation, $1+12p$ people have the latest technology in N2 but only $1+8p$ in N1. Thus the probability of moving the frontier is $\lambda(1+12p)$ in N2. That probability is larger than the same probability in N1, which is given by $\lambda(1+8p)$. Continue in this fashion until every agent in the network has acquired such level of technology. At that point, all agents have the same level of technology and the probability of advancing the frontier is again equal in both networks. In every period, we find that the probability of advancing the technological frontier is weakly higher in N2 than in N1, with strict inequality in at least one period. Therefore, we conclude that the probability of a technology shock moving the frontier in N2 is than the probability of moving the frontier in N1.

[9]The reasoning is analogous if more than one agent receives the original shock at the same time.

Proof of Result 5 Observe that the state where all agents have the same type is absorbing. We will show that such state can be reached from any state with positive probability and therefore the process will be absorbed with probability 1 (by Lemma 1).

Lemma 1 *In an finite Markov chain that is absorbing (it has at least one absorbing state and from every state it is possible to go to an absorbing state), the probability that the process will be absorbed is 1. For proof see Grinstead and Snell (1997).*

Suppose agent j is the only one whose type is different to the rest of the network. The number of j-types increases in the next period if: (i) agent j survives, (ii) all the nodes directly connected to agent j die (first tier nodes) and (iii) all the nodes connected to the nodes connected directly to agent j also die (second tier nodes). To see this, index the first tier connections with i and let $k^*(i) = argmax_{\{k:\eta_{ik}(t)=1\}} A_k(t)$. By assumption, if i dies at t, we have $\tau_i(t+1) = \tau_{k^*(i)}(t)$. Then if the three situations described happen, we have that $k^*(i) = argmax_{\{k:\eta_{ik}(t)=1\}} A_k(t) = argmax\{A_j(t), 0\} = j \ \forall i$. Therefore $\forall i$ we have $\tau_i(t+1) = \tau(k^*(i)) = \tau_j(t)$.

Now we compute a lower bound for the probability of (i)-(iii) happening at any time. First, assume $\tau_j(t) = co$. Recall that j's own type governs the links to the right and others' types govern links to the left, so in this case the first tier connections for which $\eta_{jk} = 1$ are $k = \{j-4, j-1, j+1, j+2\}$. The second tier connections (nodes connected to j's connections that are not directly connected to j) are the following: $\{j-8, j-5, j-3, j-2, j+3, j+5, j+6\}$. Therefore, with probability of at least $(1-\xi)\xi^{11}$ node j survives and all his first and second tier connections have an accident and die, reaching the absorbing state.[10]. Second, if we assume that $\tau_j(t) = in$, then his direct connections are $\eta_{jk} = 1$ for $k = \{j-2, j-1, j+1, j+4\}$ and the second tier connections are $\{j-3, j+2, j+3, j+5, j+6\}$. Therefore, with probability of at least $(1-\xi)\xi^9$ node j survives and all his first and second tier connections have an accident and die reaching the absorbing state.

In summary, we have shown that if there is one agent left with different type to the rest, with positive probability we can reach the absorbing state. If there are two or more agents whose type is different than the rest of the network, we can apply an analogous reasoning to reach the absorbing state in some finite number of steps. Since we can reach an absorbing state from any state with positive probability, the result follows from Lemma 1.

Proof of Result 6 Observe that the state with zero infected people is an absorbing state. At any given time t, for any number of sick people $m \in \{1, ..., n\}$, with probability $(1-\pi)^m > 0$ the disease is not spread and it dies out, reaching the absorbing state. Since we can reach the absorbing state from any other state with positive probability, and the number of states is finite, by Lemma 1 the probability that the process will be absorbed is 1.

B Data Appendix

Summary statistics for each of the variables we use are described in table 5.

B.1 Disease Data

Historical disease data. The historical pathogen prevalence data is from Murray and Schaller (2010), who built on existing data sets and employed old epidemiological atlases to rate the prevalence of nine infectious diseases in each of 230 geopolitical regions world. The nine diseases coded were leishmanias, schistosomes, trypanosomes, leprosy, malaria, typhus, filariae, dengue, and tuberculosis. For all except tuberculosis, the prevalence estimate was based primarily on epidemiological maps provided in Rodenwaldt and Jusatz (1961) and Simmons, Whayne, Anderson, and Horack (1945). Much of their data was, in turn, collected by the Medical Intelligence Division of the United States Army. In the rare cases in which two epidemiological sources provided contradictory information, priority was placed on data provided by the older source. In cases in which the relevant maps were unavailable (this was especially true for leprosy) or insufficiently detailed (this was especially true for many of the Pacific island nations), prevalence ratings were informed also by verbal summaries found in Simmons, Whayne, Anderson, and Horack (1945). The prevalence of tuberculosis was based on a map contained in the National Geographic Society's (2005) *Atlas of the World*, which provides incidence information in each region for every 100,000 people. Prevalence of tuberculosis was coded according to a 3-point scheme: $1 = 3-49$, $2 = 50-99$, $3 = 100$ or more. For 160 political regions, they were able to estimate the prevalence of all nine diseases. The majority of these regions are nations (e.g.,Albania, Zimbabwe);

[10] Clearly the probability of this event is higher because of the infection process.

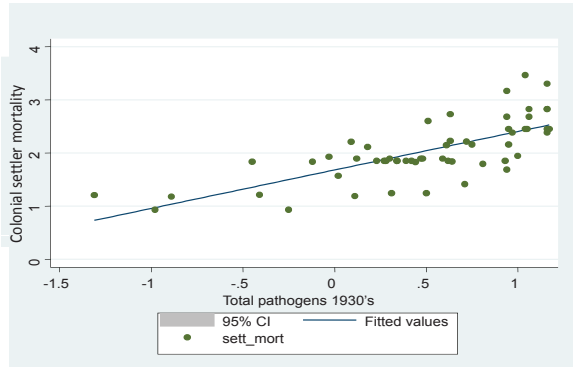
Table 5: **Summary statistics**

Variable	Obsv	Mean	Std Dev	Min	Max
Technology Diffusion	72	-0.014	0.63	-2.39	1.00
Solow Residual	64	8.19	0.647	0.628	9.02
GDP per capita	65	9.29	0.90	7.02	10.48
Individualism	75	42.27	22.98	6	91
Pronoun	65	0.68	0.47	0	1
English	70	0 .077	0.24	0	0 .974
human (1930)	75	10.25	5.33	1	19
zoonotic (1930)	75	2.87	1.49	0	6
diff_germ (1930)	75	7.38	4.76	-1	16
diff_germ_std (1930)	75	0.0011	0.995	-2.04	2.63
human (2011)	78	43.35	5.62	36	55
zoonotic (2011)	78	20.85	3.17	16	28
diff_germ (2011)	78	22.50	3.74	15	31
diff_germ_std (2011)	78	1.14	0.670	-0.357	2.90
Life Exp	73	62.44	9.69	35.95	74.65
Soc Infra	67	0.549	0.262	0.113	1
EFL	60	36.92	29.76	0	93
daly2004	74	19,162	12,513	8,013	66,278
pathcontemp	73	32.33	6.50	23	47

others are territories or protectorates (e.g., Falkland Islands, New Caledonia) or culturally distinct regions within a nation (e.g., Hawaii, Hong Kong). Figure 9 uses a color-coded map to summarize the historical data.

One testament to the accuracy of this data is its high correlation with the historical disease data reported by Acemoglu, Johnson, and Robinson (2001). Figure 8 plots our total pathogen prevalence in the 1930's against the AJR data from the colonial period.

Figure 8: Relationship between colonial settler mortality and 1930's pathogen prevalence.



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Contemporaneous disease data. To assess the accuracy of our historical disease prevalence series, we compare them to contemporaneous data that is presumably better-measured. Data were obtained from the Global Infectious Diseases and Epidemiology Online Network (GIDEON, http://www.gideononline.com) in 2011-12 and report primarily 2011 prevalence rates. The sources for data included in GIDEON currently include health ministry publications (electronic and print) and peer review journal publications. A partial listing is available at

http://www.gideononline.com/resources.htm. The quality and frequency of data input vary by source. A total of 34 specific pathogenic diseases are coded, each on a 1-3 prevalence scale. There are some diseases that GIDEON classifies on a 6-point scale, according to the per-capita reported infection rate. The cutoff rates for each level vary by disease; for example, a "4" for rabies means an infection rate between .01 and .02 per 100,000 people, while the same range delimits a "3" for tetanus. We convert from the 1-6 scale to a 1-3 scale as follows: a 1 remains a 1, a 2 or a 3 is coded as a 2, and any number above 3 is coded as a 3. The total pathogen prevalence variable is the sum of the values for each disease within each country.

Our two pathogen prevalence indices appear to be accurate because they are highly correlated (0.77). They are also highly correlated with a similar index created by Gangestad & Buss (1993) to assess pathogen prevalence within a smaller sample of 29 regions. Correlations are 0.89 with our index from 1930's data and 0.83 with our index of 2011 data. This high correlation explains why the results with contemporaneous data are nearly identical. For example, the coefficient on the historical nine-pathogen index in table 2 is -2.73, while the analogous coefficient on the contemporaneous index is -2.72.

B.2 Measuring Individualism

Hofstede (2001) defines individualism in the following way:

> Individualism (IDV) on the one side versus its opposite, collectivism, that is the degree to which individuals are integrated into groups. On the individualist side we find societies in which the ties between individuals are loose: everyone is expected to look after him/herself and his/her immediate family. On the collectivist side, we find societies in which people from birth onwards are integrated into strong, cohesive in-groups, often extended families (with uncles, aunts and grandparents) which continue protecting them in exchange for unquestioning loyalty.

The Hofstede individualism index values are based on the results of a factor analysis of work goals across countries. The index was constructed from data collected during an employee attitude survey program conducted by a large multinational organization (IBM) within its subsidiaries in 72 countries. The survey took place in two waves, in 1969 and 1972 and included questions about demographics, satisfaction and work goals. The answers to the 14 questions about "work goals" form the basis for the construction of the individualism index. The individual answers were aggregated at the country level after matching respondents by occupation, age and gender. The countries mean scores for the 14 "work goals" were then analyzed using factor analysis that resulted in the identification of two factors of equal strength that together explained 46% of the variance. The individualism factor is mapped onto a scale from 1 to 100 to create the individualism index (hereafter IDV) for each country. The highest IDV values are for the United States (91), Australia (90), and Great Britain (89); the lowest are for Guatemala (6), Ecuador (8) and Panama (11). Subsequent studies involving commercial airline pilots and students (23 countries), civil service managers (14 counties) and consumers (15 countries) have validated Hofstede's results.

IBM survey text (a subset). The original Hofstede survey is too lengthy to include in its entirety. Below, we list a subset of the questions asked. We categorize questions according to which aspect of collectivism they measure, as described in section 3.2. That grouping is not in the original survey. The survey instructions read as follows:

We are asking you to indicate how important each of these is to you. Possible answers: of utmost importance to me (1), very important (2), of moderate importance (3), of little importance (4), of very little or no importance. How important is to you to:

Category 1: Questions about the importance of personal freedom and individual benefits from the organization

1. Have considerable freedom to adopt your own approach to the job (I)
2. Have a job which leaves you sufficient time for your personal or family life (I)
3. Have challenging work to do (I)

In contrast, the last example question emphasizes the opposite, how the organization benefits from the individual's skills:

4. Fully use your skills and abilities on the job (C)

Category 2: Value of cooperation

1. Work with people who cooperate well with each other (C)

2. Have training opportunities (C)

Category 3: Willingness to change job or location

1. Live in an area desirable to you and your family (I)

We have followed the question with (I) when high importance (a low numerical score) indicates more individualism. When the higher importance indicates less individualism (more collectivist) we denote that with (C). We report these particular questions because all have factor loadings of 0.35 or more in absolute value.

Cross-Country Network Analysis There is a small literature that analyzes and compares social network structures across countries. It is summarized and extended by Fischer and Shavit (1995). Surveys typically ask respondents to name people with whom they confided, were friends, asked for help, ect. The survey takers would then interview the named friends to find out their networks and interview the friends they named as well. By repeating this process many times, the researchers could map out fairly complete social networks in specific geographic locations. For our purposes, the key finding from these studies is that the frequency of network collectives varies greatly across countries. These studies do not typically report the number of collectives. They report a related measure, network density. Density is the fraction of possible links between individuals that are present. Importantly, a network that is fully dense also has the maximum possible number of collectives. Because this research design involves lengthy interviews of many respondents, it has been done only on a handful of countries. But it is useful to see how the prevalence of network collectives correlates with Hofstede's individualism index.

Table 6: **Measures of network interdependence and individualism**

Region	Country	Network interdependence	Individualism (for country)
Haifa	Israel	0.57	54
N. California	U.S.	0.44	91
all	U.S.	0.40	91
E.York, Toronto	Canada	0.33	80
London	U.K.	0.34	89
Taijin	China	0.58	20
West Africa		0.45-0.77	20

The theory predicts a negative relationship between network interdependence (closely related to collectivism) and the individualism index. Interdependence is measured as the fraction of all possible links in a social network that are present. It is also referred to as "network density." West Africa here includes Ghana, Nigeria and Sierra Leone.

Correlation of individualism with other measures of culture. To better understand what Hofstede's individualism index (IDV) measures, we examine related cultural measures that are highly correlated with the index.

Family structure. In a collectivistic society, people grow up with members of an extended family and sometimes also neighbors, housemates, other villagers, lords and servants. Collectivists have strong ties and frequent contact with family members. In individualistic societies, people grow up in nuclear families. Their family ties are weaker. Extended family live elsewhere and visit infrequently.

Group identity. In collectivist societies, people learn to think about themselves as part of collective, with a group identity. That identity is determined by birth. Similarly, friendships come from existing group ties. Members of the collective are distinct from non-members. In the individualistic society, people learn to think about themselves as an individual, not a member of a group. There is no distinction between group members and and non-members. Gudykunst, Gao, Schmidt, Nishida, Bond, Leung, and and (1992) surveyed 200 students in each of 4 countries: Australia and US (high IDV) and Hong Kong and Japan (lower IDV). Half of the respondents were asked to imagine a group member; the others were asked to imagine a non-member. They were then asked to report if they would: talk about themselves with the person, ask about the other, expect *shared attitudes and networks*, and have confidence

in the other. The differences between how respondents viewed group members and non-members correlated exactly (negatively) with their country's IDV scores.

Other ways of modeling individualism and collectivism in networks. **Weak vs. strong ties** Granovetter (1973) introduced the idea of strong ties and weak ties in networks. Strong ties are close friends, while weak ties are acquaintances. Granovetter argues that more novel information comes from weak ties than from strong ties. The reasoning is very similar to that in our model. Because people who are very closely socially related have similar information sets, they are more likely to convey redundant information and are less likely to have novel information. Weak ties are more likely to be connected to people that we do not know and therefore are possible conduits for new information. Granovetter argues that people with few weak ties are at an informational disadvantage because they have difficulty accessing information in other parts of the social network. Thus, a society comprised of agents with mostly strong ties and few weak ties will not transmit information (or disease) as easily. Thus another way to formulate our model that would lead to the same conclusions would be to characterize collectivist societies as ones with strong ties and individualist societies as one with weak ties.

Random vs. fixed networks Another characteristic of individualist economies is that more commerce is mediated by a market, rather than being based on personal relationships. One could think of a market as being like a random search model. Buyers encounter suppliers with various prices and decide to do business or not. A random search model looks almost identical to a random network, where agents are connected to others in the network with some probability. In contrast, the collectivist economy is one where transactions take place only between people who are connected and those connections do not change over time. This captures the essence of market vs. relational transactions. For most network structures, the random network will achieve faster diffusion of technology and diseases than the fixed network (see Jackson (2008)). Thus, modeling individualist and collectivist societies as fixed or random networks would also not change the basic message of the paper.

B.3 Other Control Variables

An inevitable question arises: "What if you also control for X?" We would like to know if individualism is highly correlated with and thus proxying for some other economic phenomenon. The problem with answering this question is that what we would like to control for is likely an endogenous variable. We could treat it as such and instrument for it. But in most cases, our instruments are not strong predictors. Or, we could just, suspend disbelief, assume that these are exogenous variables, abandon any pretense of saying anything about causality, and just see what statistical relationship they have with the other variables in the estimation. We take the second approach. Each row of table 7 reports the coefficients of a second stage regression of technology diffusion on the Hofstede individualism index, one other control variable, and a constant. Since we have assumed that the control variable is exogenous, we use it as an instrument in the first stage, in addition to a constant and our standard instruments: pronoun, english and the standardized difference in pathogens variable, diff_germ_std.

The control variables are social infrastructure, a measure of the efficient functioning of political and social institutions, constructed by Hall and Jones (1999); ethno-linguistic fractionalization, a measure of the probability that two randomly-chosen people in the country will belong to different ethnic or linguistic groups, constructed by Taylor and Hudson (1972); latitude, which is the absolute value of the country's latitude, divided by 90; disability-adjusted life expectancy, which is the expected length of time an individual lives free of disability, is measured by the World Health Organization in 2004 (*http://www.who.int/healthinfo/global_burden_disease/estimates_country/en/index.html*); capitalist, which is the "economic organization" variable constructed by Freedom House, scores more capitalist countries higher and more socialist countries lower; and population density is the 1970 population per square mile, as reported by the World Bank.

Table 7: **Controlling for other economic variables**

Dependent variable	Technology Diffusion						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Individualism (S)	0.59	0.69	1.23*	1.35*	1.02*	1.24*	1.46*
	(0.34)	(0.39)	(0.30)	(0.36)	(0.27)	(0.36)	(0.31)
Life expectancy	4.29*						
at birth (LEB)	(0.78)						
Social		112.2*					
Infrastructure (SocI)		(30.17)					
Ethno-linguistic			−1.08*				
fractionalization (EFL)			(0.21)				
Latitude				0.21			
				(0.26)			
Disease-adj					−0.0030*		
life expectancy (DALY)					(0.0006)		
Capitalist						5.89	
(EcOrg)						(4.44)	
Population							0.040*
Density							(0.010)
Constant	-300.7	-98.51	-15.40	-67.68	7.05	-76.77	-72.64
R^2	0.58	0.47	0.52	0.33	0.63	0.34	0.43
Observations	62	60	55	61	61	61	62

2SLS estimates of $100 * \gamma$ coefficients in Diffusion $= \gamma_1 + \gamma_2 \mathrm{S} + \gamma_3 x + \eta$, where the x variables are listed in the first column of the table. The first stage regression is $S = h_1 + h_2 x + h_3 \text{diff\_germ\_std1930} + h_4 pronoun + h_5 english + e$. Standard errors in parentheses. $*$ denotes significance at 5% level.

Table 8: **Classification of Diseases**

Disease type is H if we classify the disease as human, M for multi-type and Z for zoonotic. The classification is based on the disease reservoir. Historical is Y if the disease is present in our 1930's historical data. For disease categories with multiple sub-types (i.e. Filaria - Bancroftian and Filaria - Brugia Timori), there is one combined value in the historical data. That value is the maximum of the value for each strain. It lists 4 if any strain is epidemic, 3 if any strain is endemic, ect. All listed diseases are present in the 2011 data. Different strains are treated like different diseases. All 2011 results are robust to combining strains (results available on request). Source: GIDEON database.

Disease	Agent	Reservoir	Spread By	Type	Historical
Diphtheria	Bacteria	Man	Droplet, Contact, Dairy, Clothing	H	N
Filaria - Bancroftian	Nematoda	Man	Mosquito	H	Y
Filaria - Brugia Timori	Nematoda	Man	Mosquito	H	Y
Measles	Virus - RNA	Man	Droplet	H	N
Meningitis - Bacterial	Bacteria	Man	Air, Secretions	H	N
Meningitis - Viral	Virus - RNA	Man	Fecal-oral, Droplets	H	N
Pertussis	Bacteria	Man	Air, Secretions	H	N
Poliomyelitis	Virus - RNA	Man	Fecal-oral, Food, Water, Flies	H	N
Smallpox	Virus - DNA	Man	Contact, Secretions, Fomite	H	N
Syphilis	Bacteria	Man	Sexual Contact, Secretions	H	N
Typhoid fever	Bacteria	Man	Fecal-oral, Food, Flies, Water	H	N
Dengue	Virus - RNA	Man, Monkey, Mosquito	Mosquito, Blood (rare)	M	Y
Filaria - Brugia Malayi	Nemotoda	Man, Primate, Cat, Civet	Mosquito	M	Y
Leishmania - Cutaneous	Protozoa	Man, Rodent, Other Mammals	Fly	M	Y
Leishmania - Mucocutaneous	Protozoa	Man, Rodent, Sloth, Marsupial	Fly	M	Y
Leishmania - Visceral	Protozoa	Man, Rodent, Dog, Fox	Fly, Blood	M	Y
Leprosy	Bacteria	Man, Armadillo	Patient Secretions	M	Y
Malaria	Protozoa	Man, Mosquito	Mosquito, Blood	M	Y
Trypanosoma - African	Protozoa	Man, Deer, Cattle, Carnivores	Fly	M	Y
Trypanosoma - American	Protozoa	Man, Dog, Cat, Other Mammals	Kissing Bug, Blood, Fruit	M	Y
Tuberculosis	Bacteria	Man, Cattle	Air, Dairy Products	M	Y
Typhus - Epidemic	Bacteria	Man, Flying Squirrel	Louse	M	Y
Anthrax	Bacteria	Soil, Water, Other Mammals	Fly, Hair, Hides, Bone, Air, Meat	Z	N
Leptospirosis	Bacteria	Frog, Cattle, Other Mammals	Water, Soil, Urine, Contact	Z	N
Rabies	Virus - RNA	Dog, Fox, Other Mammals	Saliva, Bite, Transplants, Air	Z	N
Schistosomiasis - Haematobium	Flatworms	Snail, Baboon, Monkey	Water (Skin Contact)	Z	Y
Schistosomiasis - Intercalatum	Flatworms	Snail	Water (Skin Contact)	Z	Y
Schistosomiasis - Japonicum	Flatworms	Snail, Other Mammals	Water (Skin Contact)	Z	Y
Schistosomiasis - Mansoni	Flatworms	Snail, Other Mammals	Water (Skin Contact)	Z	Y
Schistosomiasis - Mattheei	Flatworms	Snail, Other Mammals	Water (Skin Contact)	Z	Y
Schistosomiasis - Mekongi	Flatworms	Snail, Dog	Water	Z	Y
Tetanus	Bacteria	Animal Feces, Soil	Injury	Z	N
Typhus - Endemic	Bacteria	Rat	Flea	Z	Y
Typhus - Scrub	Bacteria	Rodent, Carnivores, Mite	Mite	Z	Y

Figure 9: A world map of historical pathogen prevalence.