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Contagion, Spillover, and Interdependence

ABSTRACT This paper reviews the empirical literature on international spillovers and contagion. Theoretical models of spillover and contagion imply that the reduced-form observable variables suffer from two possible sources of bias: endogeneity and omitted variables. These econometric problems, in combination with the heteroskedasticity that plagues the data, produce time-varying biases. Several empirical methodologies are evaluated from this perspective: nonparametric techniques, such as correlations and principal components; and parametric methods, such as OLS, VAR, event studies, ARCH, and nonlinear regressions. The paper concludes that there is no single technique that can solve the full-fledged problem and discusses three methodologies that can partially address some of the questions in the literature.

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Almost every paper on contagion starts with a definition of what exactly the author means by contagion and spillover. I would like this paper to be an exception—first and foremost because my own definition of contagion has shifted over time. For example, I have sometimes defined contagion strictly as the unexpected or surprising component of the transmission of shocks across countries; at other times as a change in the behavior during crises; and lately as purely any form of propagation across countries, irrespective of the circumstances. Interestingly, whatever the definition of contagion I used at any particular point in time, all of them were (and still are) a common feature of the data. This was good, because I could always claim that I had found evidence of "contagion" in the data. Here, however, instead of claiming victory, I would like to concentrate on what we have learned and what challenges lie ahead. Therefore, let me use the words *contagion* and *spillovers* to very loosely describe the phenomenon in which a shock from one country is transmitted to another. Well, as Britney Spears might say, *Oops, I just did it again*. I started the paper, *again*, with a definition of contagion and spillover.

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On a more serious note, I believe the distinction between contagion and spillover (or interdependence or linkages) is tenuous. Both are transmission mechanisms whose distinctions are model- and belief-dependent. For example, models of trade tend to underestimate the spillover across countries because the models fail to capture the linkages that exist through the financial system. In those models, spillover occurs through the modeled channel, while contagion is what is left unmodeled. In my view, this is rather unsettling. The definition of what constitutes spillover versus contagion changes simply by rewriting the model.

Furthermore, even when all the interdependence is modeled, there is still a question of magnitude. For instance, for some researchers and market participants, the relationship between the United States and Canada is so strong that they would be surprised by a 75 percent correlation in the stock markets, whereas others would find such a degree of correlation too high—and hence surprising and therefore contagious. In other words, if the strength of the comovement is of an order of magnitude in line with the researcher's beliefs, then it is called a spillover, but if the comovement is higher, then it is interpreted as contagion. Again, in my view, the distinction seems rather semantic. In fact, I cannot imagine our profession will ever resolve this discussion.

In the end, the profession seems to agree on two aspects. First, spillovers are always present—in good and bad times. In contrast, while contagion could be present at all times, it tends to be more relevant during crises or periods of stress. This leads to the second point: I believe most agree on the definition of shift contagion, which occurs when the propagation of shocks intensifies during crises or stress episodes—and it is inherently a parameter instability feature in the data. This has motivated me to split the empirical problem as follows: the estimation of the transmission mechanism during normal times (which could or could not be contagious) and the estimation of the change in the transmission mechanisms after a certain macroeconomic event (shift contagion).

The literature has devoted an enormous amount of attention to studying what drives shocks across countries and what their most important propagation mechanisms are. Although evidence of contagion can be traced back more than a century, during which time some of the most dramatic events were the Great Depression and the debt crises in the early 1980s, most of the academic interest started to appear after the Mexican 1994, Asian 1997, and Russian 1998 currency collapses. Without a doubt, the extent and intensity of the transmission of these shocks around the globe surprised many—both academics and practitioners. In the early 1980s, Costa Rica and Mexico defaulted on their debt, and a cascade of countries in Latin America started defaulting

and renegotiating their sovereign debt. How was it possible that countries so seemingly unrelated economically—with small trade connections—could all be acting together? By the early,1990s, several of these countries had restructured their debt under the Brady Plan. Nevertheless, Mexico—a country that had followed the advice of economic orthodoxy—suffered a currency crisis and devalued in 1994. Argentina followed. These development were so surprising that Guillermo Calvo coined the phrase "the Tequila Crisis."[1] Guillermo had to coin more terms, though. In 1997 he called it the Asian Flu, and by the end of 1998 he was talking about the Russian Cold. Shocks to Mexico, Thailand, and Russia devastated Argentina! This was well summarized by Sebastian Edwards, who stated on a panel that "whenever anyone in the world sneezes, Argentina gets pneumonia."

Several features of these events prompted such interest. First, previous crises implied propagation from large countries to smaller ones—the Great Depression is a good example. On many grounds, the fact that a large country has an impact on a smaller economy is not at all surprising. The late 1990s crises, on the other hand, occurred in relatively small markets and still had large global effects.

Second, in the past, most of the countries affected by the shocks had strong trade relationships with the country where the crisis originated. For example, the collapse in Russia at the end of the 1980s and the subsequent collapse of Finland were considered to be a natural sequence, in which the events in one country affected the exports of the other main trading partner. There was nothing terribly surprising about the transmission of shocks across two highly interrelated economies. In the late 1990s, however, countries with very small trade links were heavily influenced by crises and shocks in other countries. For instance, there was no clear trade relationship between Mexico and Argentina that could explain the contagion in 1994. There was even less of a relationship in 1998 between Russia and the pair that suffer the most in Latin America, Argentina and Brazil. Finally, in 1997, the MIT countries—Malaysia, Indonesia and Thailand—had little in common other than belonging to the same region.[2]

1. Calvo (1996).

2. Not all crises are equally contagious. In contrast to the crises I have highlighted, some crises at the beginning of the twenty-first century had a completely different behavior. Indeed, the lack of contagion after the Brazilian 1999, Turkish 2000, and Argentine 2002 crises is apparent even to a casual observer (see Miller, Thampanishvong, and Zhang, 2003; Kaminsky, Reinhart, and Végh, 2003). Only small countries with very strong ties (Argentina to Uruguay, for instance) were affected by the exchange rate regime collapses. Theories of contagion that explain the excessive transmission in some of the crises should also be able to account for the lack of contagion that took place in others.

Recently, the subprime crisis in the United States in 2008 and the fiscal crises in Europe in 2010 have renewed interest in contagion and, more important, in its prevention. These crises share many features of the emerging market crises. According to the Bank for International Settlements (BIS), the size of the subprime securitized assets in 2007 was U.S. $860 billion. Meanwhile, the size of the whole financial sector (taking into account both the formal and shadow financial sectors) was north of U.S. $25 trillion. So the subprime market was less than 4 percent of the financial system, yet it had a massive impact in the United States and around the world. As in most contagious events, a shock to a seemingly small and isolated market had a global impact.

Europe's fiscal crises share the same characteristics. Greece is, after all, a tiny proportion of gross domestic product (GDP), trade, and financial flows in the euro area. Even though European countries have strong trading and financial ties, it is hard to explain the amount of anxiety in the markets in response to the Greek Tragedy.

Finally, in 2018, a small increase in interest rates in the United States caused havoc in emerging markets. I would not be surprised if the profession encountered another contagious event worth studying by mid-2020. The story would be the same: A tiny country (or market) has a crisis and the shock is propagated everywhere. All countries suffer, and Argentina crashes the most.

This is certainly not the first review of the methodologies for measuring spillovers or contagion. Many very good reviews have been out there for quite some time.[3] The objective of this paper is to discuss the methodologies that have been used to measure contagion and spillovers, identifying their advantages and disadvantages and introducing the next generation of empirical methods in this literature. The proper estimation of contagion is a crucial issue for central banking. This is important for financial regulation, where banks are interconnected with each other; for local financial markets, where bonds, stocks, and different assets are related to each other; and for international markets, where the unit of analysis is countries. Monetary and regulatory authorities are constantly estimating the underlying relationships to understand when to act and by how much. My fear is that because these estimates have been obtained by using methodologies that are not robust,

3. For a concise review of the theories, see Claessens, Dornbusch, and Park (2001). For a summary of the theories and some of the early methods used in contagion, see Goldstein, Kaminsky, and Reinhart (2000) and Forbes and Rigobon (2001a, 2001b). For a critical view of the empirical methods used by earlier papers, see Rigobon (2002). For a recent survey on the empirical strategies in contagion, see Dungey and Fry (2004).

the decisions are clearly wrong. A recent document published by the BIS comes to mind.[4]

Characteristics of the Data

Taking the theories of contagion or spillover to the data is not an easy task. The first problem is that the specifications implied by these models generally cannot be estimated with a simple ordinary least squares regression. I come back to this point later in the paper. The second problem is that the data share some particular traits that are not necessarily implied by the theories. These are not rejections of the theories but rather derive from the context in which the transmission of shocks occurs. Three features are quite important, and I will repeatedly come back to them throughout my discussion. These three characteristics are relatively uncontroversial. First, the data have heteroskedasticity, and in particular contagion events are associated with massive increases in volatility. Most of the literature studies spillovers in financial variables, which suffer from conditional heteroskedasticity. In the case of contagion, it is common for variances to increase tenfold, for both financial and real variables. In other words, stock markets, interest rates, and exchange rates become massively more volatile; and credit, consumption, investment, and GDP also experience increases in variance. Heteroskedasticity is a fundamental characteristic of the data where spillovers and contagion are evaluated. From the empirical point of view, this characteristic represents one of the biggest impediments in the measurement of the international transmission of shocks. The reason, which I will repeat over and over again, is that in a misspecified regression, the degree of misspecification changes when the volatilities of the shocks move around. Consequently, researchers will have a problem determining whether they are estimating the bias or the spillover. The heteroskedasticity is a feature of the data and not of the theories. This implies that correlations move around and, more important, that correlations increase in a contagious crisis. In the following section, I criticize correlations as a measure of contagion or spillover, but any theory or empirical strategy has to account for the fact that in the reduced form, correlations increase during crises.

4. See Claessens and Kose (2018). The book has over 120 pages, and I believe most of the models discussed there are based on dubious facts.

Second, contagion events tend to be short-lived. In fact, the frequencies in which the event is measured require high-frequency data.[5] Contagion tends to propagate a crisis in a matter of weeks, and it takes months to resolve. This is important in the sense that contagion does not have long-run growth effects. It is a short-run hiccup, but because of its size, it does require policy action. The 2008 U.S. crisis has taken forever for the United States to resolve, but its contagion in emerging markets took place between September 2008 and mid-2009, and emerging markets were already growing again by mid-2010.

Finally, spillovers are inherently evaluated as a financial phenomenon. Stock prices, interest rates, and exchange rates are the escape valve in the system. Of course, GDP, consumption, investment, trade, and financial credit are also affected. Nevertheless, the detection of spillovers requires relatively high-frequency data, so most of the empirical research ends up focusing on the financial variables. Very few theories explicitly make this connection, as I explain later. It is always implicitly assumed that if a decline in GDP is experienced, then the stock market moves in tandem, but this is an assumption rather than a result. Hence there is a lack of connection between the theories and the empirical work.

Short Review of the Theories of Spillovers and Contagion

This section reviews the theories behind the international propagation of shocks. The theoretical literature can be divided in three broad views: fundamental, financial, and coordination.[6]

Fundamental View

The fundamental view of contagion and spillover explains the propagation of shocks across countries by appealing to real channels. The papers in this literature include explanations based on bilateral trade, trade of similar goods with a common market, and monetary policy coordination and macroeconomic similarities.[7]

5. Where high frequency for macroeconomists means days or weeks.

6. For surveys of the theories behind contagion, see Claessens, Dornbusch, and Park (2001) and Claessens and Kose (2018).

7. The most prominent papers include Gerlach and Smetts (1995), Corsetti and others (1999), Corsetti, Pericoli, and Stracia (2005), and Basu (1998).

For example, on the bilateral trade explanation (which happens to be the first paper on contagion!), if a country has a crisis and its consumption declines, then the country's imports are likely to decline as well.[8] Therefore the trading partners experience a decline in the demand for their exports: either their prices drop—a deterioration in the terms of trade—or they reduce production. In both cases, their GDP declines, and there is a recession and quite likely a depreciation. All international real business cycle models exhibit this transmission channel.

This can easily be extended to two unrelated countries (peripheral countries) trading with a third one (the center country). If the country at the center suffers from a crisis, the demand for the exports of the peripheral countries declines. So the two seemingly unrelated economies experience common shocks that are transmitted through the trade channel.

Monetary policy and other macroeconomic policies are also linked by trade. Therefore the transmission is not exclusively through relative prices but can also occur through monetary policy coordination and other similar macroeconomic policies. For example, if the United States increases its interest rates, other countries have to evaluate their monetary policy paths. The increase in the United States is a common shock to the world, and several emerging markets would suffer the negative consequences. In Latin America, Argentina, Brazil, and Venezuela have undergone massive recessions in 2016–2019—though Venezuela's is self-inflicted. Countries such as Chile, Colombia, and Mexico are slowing down, but to a much lesser extent. Interestingly, the best predictor of "suffering" is if the country exclusively touches the Atlantic Ocean.[9]

The theories based on fundamental transmission mechanisms were used to explain the transmission from the Great Depression and European crises in the 1970s and 1980s. In those instances, trade played a very important role in the transmission of the shocks. Most of these papers study the interaction between real shocks, real variables, and nominal exchange rates, even though most of the contagion was evaluated in countries depreciating their currencies.[10]

The finance literature combines trade and asset prices in a single framework. Pavlova and Rigobon use a general equilibrium model to analyze the

8. Gerlach and Smets (1995).

9. I know, not much of a theory.

10. See Forbes (2001) and Forbes and Rigobon (1999) for two good examples evaluating the strength of the fundamental channels of contagion.

interactions between international asset prices, the exchange rate, and trade in goods.[11] In that paper, we confirm that the same intuitions derived in exchange rate markets can be applied to equity and bond prices. Additionally, Martin studies asset prices in a multicountry model and shows how shocks from one country change conditional correlations exactly in the spirit of contagion.[12] These two papers put together the simple intuitions of trade within asset price models.

Financial View

The financial view concentrates on constraints and inefficiencies in banking sectors and international equity markets. The idea of this channel is that imperfections in the financial system are exacerbated during a crisis, and such imperfections limit the extent to which financial services can be provided to different countries—which ex ante might have been seen as independent. This theory, in general, implies that a shock increases the propagation of shocks across countries. In most of these models, trade channels—and other fundamental channels—are shut down. In other words, the theories based on financial linkages assume that real linkages are not present and that the only reason behind the propagation of shocks is that financial markets are imperfect and subject to a variety of constraints. This is obviously an extreme assumption, but it allows for a clearer analysis of the reasons behind the transmission mechanisms.

In general, the contagion argument goes as follows. Assume that two countries receive financial services from a third party. The financial services can be direct lending, insurance, the provision of liquidity, and so forth. The assumption is that a shock in one country affects the balance sheet of the financial intermediary, limiting its ability to continue offering the same services to the other country. The reduction in the service to the second country has real effects owing to the presence of financial imperfections. In the end, this affects asset prices and exchange rates, as well. Therefore, the countries are interrelated because both are receiving financial services from a common financial institution or market.

For example, the common-lender theory advanced by Goldstein, Kaminsky, and Reinhart and by Kaminsky and Reinhart assumes that a single bank is lending to two countries whose outputs are, in principle, unrelated.[13]

11. Pavlova and Rigobon (2007).
12. Martin (2013).
13. Goldstein, Kaminsky, and Reinhart (2000); Kaminsky and Reinhart (2003).

A crisis in one country affects the bank's balance sheet, forcing the bank to stop lending to the second country. So, even if two countries are independent in terms of real linkages, their international flows still comove, as do other macroeconomic variables. These theories were developed to study the Asian crises in 1997. In this case, the Japanese banks were the culprit of the contagion.

The theories based on margin calls, liquidity aspects, or wealth effects are similar in spirit to the common lender. In these cases, the financial intermediary is the capital market instead of the banking sector. The most prominent example of these theories is Calvo and Mendoza.[14] In these models, a shock in one country lowers the value of the portfolio holdings of the intermediary. The fall in wealth implies that financial intermediaries behave either as if they have a higher degree of risk aversion or are subject to margin calls. Both reasons force them to sell off assets in the same asset class. In the end, this implies downward pressure on all the assets held by the intermediary, causing contagion. Most of these theories were developed to understand the transmission during the 1998 Russian crisis and the aftermath of the Long-Term Capital Management (LTCM) crisis.

Finally, new theories of financial spillover highlight the network across financial institutions as the vehicle of propagation.[15] This is a promising area of research, although measurement of the interconnections in the network is still an open question. In particular, what makes two markets connected? Is it their high correlation or their conditional distribution? What if the high correlation is the outcome of an omitted variable? These are all still open questions in the literature.

Coordination View

The third class of theories is based on coordination failure. The coordination view studies investors' and policymakers' behavior and coordination problems as the explanation behind contagion. In these theories, most of the contagion comes from investors' actions and is usually a learning or herding problem. Theories based on the coordination of market participants include explanations

14. Calvo and Mendoza (2000). See also Yuan (2005) and Mendoza and Smith (2002) for theories on margin calls and their real effects; Kyle and Xiong (2001) for a theory in which wealth shocks create contagion; and Gromb and Vayanos (2002) for a model where market participants face portfolio constraints.

15. See Allen and Gale (2000) for the first attempt; see Elliott, Golub, and Jackson (2014) for a theoretical foundation of contagion through a network.

where the spillover is due to multiple equilibrium, herding, learning, and political contagion.

In these papers, the transmission of shocks occurs because there is an informational problem that drives market participants (investors) to withdraw resources jointly across countries. In addition, policymakers can coordinate and decide to abandon a particular macroeconomic policy—usually the exchange rate regime—when another country implements the same policy. In the end, the transmission exists because the actors in the market coordinate and move from one equilibrium to the other and not because the countries have something in common—except for the policy shift.

In the first multiple equilibrium framework of contagion, contagion is defined as a shift from a good to a bad equilibrium.[16] When the herding informational cascades are applied to capital flows, the spillover occurs because information in one country leads investors to take actions in another.[17] Theories of learning have also been used to explain contagion (in particular).[18]

Finally, one of my preferred theories of spillover is political contagion. Drazen analyzes the abandonment of the Exchange Rate Mechanism (ERM) in Europe in 1991.[19] The intuition is that belonging to the ERM was equivalent to belonging to a gentlemen's club. Belonging to the club provided benefits in terms of reputation and class, but it also required significant sacrifice. In Drazen's model, once a country decides to abandon the club, two things occur: the cost of abandoning for the next gentleman is smaller, and the value of remaining in a smaller club is also smaller. Therefore, the abandonment of one country increases the likelihood that a second one will drop out as well. In his framework, the fluctuation in reputational cost leads all countries to jointly adopt or abandon a particular policy.

It could be argued that the political spread of populism in Latin America followed Drazen's mechanisms. Chávez came to power in Venezuela in 1999 (elections in 1998), at a time when Latin America was mostly following policies close to the center of the political spectrum. The political success of Chávez propagated to other countries in different degrees. Who suffered the most? Bolivia, Brazil, Ecuador, El Salvador, and, of course, Argentina.

16. Masson (1998).
17. Calvo and Mendoza (2000); Chari and Kehoe (1999).
18. For example, Kodres and Pritsker (2002); Rigobon (1998).
19. Drazen (2000).

However, by 2018, all Latin American countries had to deal with the threat of extreme populism.

Measuring Contagion and Spillover

The theoretical papers on international spillover have two important empirical implications. First, most of the models exhibit nonlinearities.[20] Second, all the models imply either endogeneity or omitted variables. For example, asset pricing models imply reduced-form factor models that are similar to the reduced forms obtained from endogenous systems of equations.[21] The theoretical models based on coordination or networks, on the other hand, implicitly explain contagion as a latent factor—which is not present in tranquil times.

In the measurement of international spillovers, comparisons are often made to the notions of contagion that have been developed in the medical literature. the problems are very different, however, from the perspective of the theoretical implications just highlighted. In medicine, there are two approaches: a direct measurement of contagion and an indirect approach. For example, a direct measure of the degree of contagion of a particular virus might rely on blood tests to detect the presence of the virus, while an indirect measure would concentrate on the symptoms. In the direct measurement, the speed and intensity at which the virus is transmitted from one individual to another are directly evaluated by the concentration of the virus in the bloodstream. In international economics, this is equivalent to observing the fundamental forces that drive the spillover. This requires economists to directly measure risk appetite, contingent contracts, incentives, the information each agent possess, and so on. In practice, this method is hard to implement, for two reasons. First, it is almost impossible to measure the fundamentals at the required level of granularity. For example, we observe interest rates or average default rates, but not perceptions, heterogeneity, beliefs, risk preferences, and so forth. Second, and even worse, the literature rarely agrees on what needs to be measured. Even if we were able to measure a particular fundamental determining interest rates across countries, it might not be a channel that most of the literature cares about. In sum, it is hard to see the "virus."

20. However, some papers linearize these relationships and estimate simple linear functions, while other techniques are more agnostic.

21. For example, Pavlova and Rigobon (2007, 2008).

The second approach in medicine is to observe and evaluate the symptoms. Assume that one of the symptoms of the virus is a high fever (for the sake of the discussion, let's assume the threshold is 104° Fahrenheit). In a population within a city that is not suffering from the virus, the frequency of the event "high temperature" is relatively low. In fact, the likelihood that one person has a temperature of 104°, given that another person in the population has a temperature of 104°, is relatively low as well. So, in normal times, high temperatures are rare, and the events are almost independent. They are not totally independent because high fever in a particular city could be caused by pollution, climate, food, and so on, which affect the whole population. These correlations and frequencies define what is considered normal. If a virus is introduced into the city, the frequency of 104° temperatures is expected to increase, and the conditional probabilities will also increase as well. In other words, the propagation of the event "high temperature" increases with the presence of the virus. This is the typical problem we have in finance and international economics. There are factors that create comovement in normal times that are intensified during contagious time. The idea, therefore, is to evaluate how different the propagation during a contagious event is from the propagation that exists in normal times. The problems of the indirect procedure are several, including how to define normal and how to evaluate the propagation in contagious times.

This section is divided into three relatively technical subsections. It is impossible to discuss the weaknesses of the empirical methods without a formal framework. The first subsection discusses the models used in order to highlight the biases caused by endogeneity and omitted variables; the second, the bias in some of the standard methods used to estimate contagion, with a focus on the parameter instability that spuriously arises in the presence of heteroskedasticity; and the third, three newer techniques that partially address some of the problems.

Simple Models of Spillovers

Let us formalize the econometric problems of measurement in a simple framework. The two models described here are known as the structural model. The idea is that these are the equations and shocks that govern the system—when studied to its primitives. So the shocks are called structural shocks, and the parameters are structural parameters. These are the coefficients and shocks that describe the underlying linkages across countries and financial variables. They are supposed to capture the theories of international spillover and contagion.

OMITTED VARIABLE MODEL. Assume the returns of two asset prices are explained by two common factors and some idiosyncratic shocks. Assume the factors are unobservable. Then

$$x_t = z_t + v_t + \varepsilon_t \tag{1}$$

and

$$y_t = \alpha z_t + \beta v_t + \eta_t, \tag{2}$$

where z_t is the factor in normal times; v_t is the factor that appears during a contagious event, meaning it is zero during normal times and different from zero in crisis times; and ε_t and η_t are some country-specific shock.[22] In other words, z_t is the factor that explains "high temperature" appearing in two individuals during normal times while v_t is the virus. We assume that the variance of the virus is larger than the variance of the nomal-time shock:

$$\sigma_v^2 > \sigma_z^2.$$

This is implicitly capturing the fact that contagious events exhibit higher volatility. Also, conditional on the same variance, contagious events are assumed to propagate with higher intensity, which means that $\beta > \alpha$. These assumptions imply that the spillover is shifting through time and that contagion, in particular, is an event where comovement (and therefore correlation) is higher.

Equations 1 and 2 are the omitted variable representation of the estimation problem. This is perhaps the most flexible specification.

ENDOGENOUS MODEL. There is an endogeneity representation that shares the same reduced form:

$$x_t = \beta y_t + \varepsilon_t \tag{3}$$

and

$$y_t = \alpha x_t + \eta_t, \tag{4}$$

22. In this formulation, the nuisance variables, z_t and v_t, are the unobservable factors. They can be normalized to have a coefficient or loading of one on the first asset. Conversely, they could be normalized to have a variance of one, and the loadings on the shocks are different from one for both assets.

with reduced form

$$x_t = \frac{1}{1-\alpha\beta} + (\beta\eta_t + \varepsilon_t)$$

and

$$y_t = \frac{1}{1-\alpha\beta} + (\eta_t + \alpha\varepsilon_t),$$

where η_t and ε_t could be renormalized to become z_t and v_t in equations 1 and 2.

These two models have the exact same implications for the difficulty of estimating spillovers and contagion in the data. Before proceeding to the discussion of each of the methodologies, I review two concepts: first, the graphical representation of the joint residuals in these models always takes the form of a rotated ellipse, and second, the rotation is summarized by the variance-covariance matrices in each of these models.

ELLIPSES. In equations 1 and 2 and equations 3 and 4, the only meaningful moment that can be computed to estimate the degree of contagion is the covariance matrix. An important question, then, is what the covariance matrix represents. The errors in these models are distributed as a multinomial, and their contours are ellipses. To fix concepts, I start with a simple endogenous system of equations 3 and 4:

(3) $$x_t = \beta y_t + \varepsilon_t$$

(4) $$y_t = \alpha x_t + \eta_t,$$

where α and β are the coefficients summarizing the endogeneity and where the two errors (ε and η), called the structural shocks, are independent (have no correlation). The variance of ε is σ_ε^2 and of η is σ_η^2. The covariance matrix between x and y represents a rotated ellipse. In other words, the ninety-fifth percentile of the errors is distributed as a rotated ellipse. It is possible to solve for two independent normal distributions from the structural equations as follows (with some abuse of notation):

$$\phi_1 = \frac{x_t - \beta y_t}{\sigma_\varepsilon} N(0,1)$$

$$\phi_2 = \frac{y_t - \alpha x_t}{\sigma_\eta} N(0,1).$$

Because ϕ_1 and ϕ_2 are independent with mean zero and variance one, it is possible to describe the ζ confidence interval as

$$\phi_1^2 + \phi_2^2 = \zeta.$$

This is exactly an ellipse. Substituting,

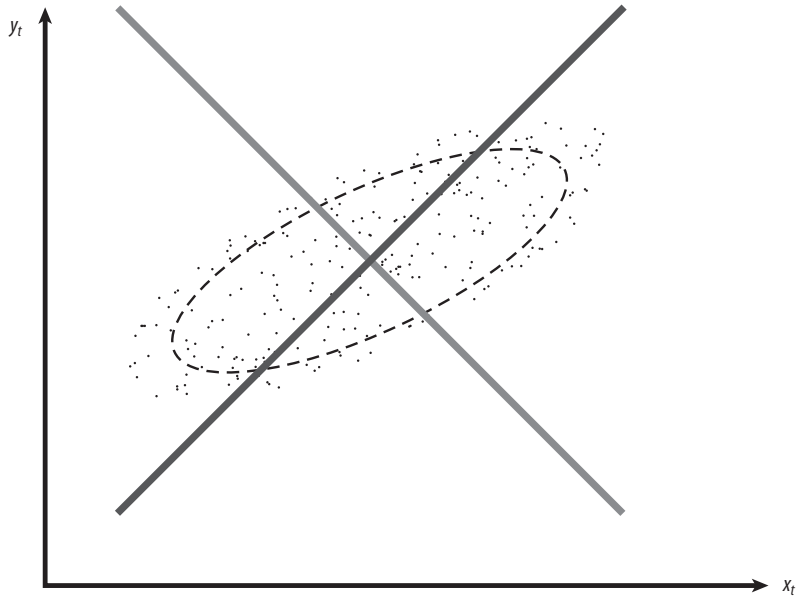
$$\left(\frac{x_t - \beta y_t}{\sigma_\varepsilon}\right)^2 + \left(\frac{y_t - a x_t}{\sigma_\eta}\right)^2 = \zeta. \tag{5}$$

Notice the similarity with the general equation of a rotated ellipse:

$$\left(\frac{x_t \cos(\theta) + y_t \sin(\theta)}{a}\right)^2 + \left(\frac{x_t \cos(\theta) + y_t \cos(\theta)}{b}\right)^2 = \zeta. \tag{6}$$

The two axes of the ellipse cannot be computed in closed form solution, but they depend on the slope of the curves (structural parameters) and the relative variances of the shocks. Figure 1 shows a graphical representation. The thick

FIGURE 1. Distribution of Errors



black curve represents supply and the thick gray curve demand (when there are no shocks). The points reflect some random realization of structural shocks that leads to a point far from the depicted schedules. The ellipse represents the ninetieth percentile. In this particular case, β is assumed to be negative (representing the demand), while α is positive. In figure 1, the variance of the demand shocks is larger than the variance of the supply shocks, so the ellipse is closely aligned with the supply curve. In the limit, if the variance of the demand is infinitively large, the ellipse would coincide exactly with the supply curve.

COVARIANCE MATRICES. The form of the ellipse is also summarized by the covariance matrix computed in the reduced form. Additionally, most of the methodologies used in this area are based on the covariance matrix, so all the sources of bias can be traced to it. Finally, as mentioned previously, the only statistic in these two models that can be computed from the data—which allows the recovery of the structural parameters—is the covariance matrix. In the case of the omitted variable model (equations 1 and 2), the covariance matrix is given by

$$\operatorname{var}(x_t) = \sigma_z^2 + \sigma_v^2 + \sigma_\varepsilon^2,$$

$$\operatorname{var}(y_t) = \alpha^2\sigma_z^2 + \beta^2\sigma_v^2 + \sigma_\eta^2,$$

and

$$\operatorname{cov}(x_t, y_t) = \alpha\sigma_z^2 + \beta\sigma_v^2;$$

while in the endogeneity model (equations 3 and 4), it is

$$\operatorname{var}(x_t) = \frac{1}{(1-\alpha\beta)^2}\left(\sigma_\varepsilon^2 + \beta^2\sigma_\eta^2\right),$$

$$\operatorname{var}(y_t) = \frac{1}{(1-\alpha\beta)^2}\left(\sigma^2\sigma_\varepsilon^2 + \sigma_\eta^2\right),$$

and

$$\operatorname{cov}(x_t, y_t) = \frac{1}{(1-\alpha\beta)^2}\left(\alpha\sigma_\varepsilon^2 + \beta\sigma_\eta^2\right).$$

Empirical Strategies

This section discusses the biases and possible solutions for the different empirical methodologies. I start with a discussion of nonparametric techniques

such as correlation and principal components. I then analyze the biases that exist in linear models, such as ordinary least squares (OLS), vector autoregression (VAR), autoregressive conditional heteroskedasticity (ARCH), generalized autoregressive conditional heteroskedasticity (GARCH), and event studies. Finally, I discuss the bias in limited dependent models.

I concentrate all the discussion on the endogeneity model. The results are easily replicated in the omitted variable case. In almost every case, I analyze the statistic being computed and its dependence on the relative volatility of the structural shocks. Define

$$\theta = \frac{\sigma_\eta^2}{\sigma_\varepsilon^2}. \tag{7}$$

I describe the biases that arise in correlations and principal components methods. One advantage of these two methods (as well as copulas) is that they are agnostic about the underlying model. This is a major advantage because the transmission mechanism does not need to be specified by the econometrician. The problem is that they do not measure the structural parameters and are inherently unstable. After discussing these two methods, I concentrate on linear regressions and extreme outcome regressions. The biases that arise in the estimation of OLS are particularly important because they depend on the relative variances of the shocks. As mentioned, heteroskedasticity is a characteristic of the data. The bias will therefore be shifting in the sample, and hence estimates will be unstable. Finally, I end the discussion with probability models—another nonparametric approach that benefits from its simplicity.

CORRELATION. Correlation is one of the preferred methods to capture or measure comovement. It is commonly argued that when the correlations shift, it is because of changes in structural parameters. This is not always correct. The correlation is not an unbiased estimator when volatilities change. Second, and more important, the correlation is a poor estimate of the spillover.

What is the correlation between x and y in equations 3 and 4? From the covariance matrix, it is easy to show that the correlation is given by

$$\rho = \frac{\alpha + \beta\theta}{\sqrt{(1+\beta^2\theta)(\alpha^2+\theta)}} \tag{8}$$

In this environment, correlations are a bad measurement of comovement. First, the correlation is not a measure of α or β. It is a combination of these

two coefficients, so it does not have a structural interpretation. Most economists understand this in seconds, but it is not obvious to many.

Second, the correlation changes when the relative variances shift—when θ changes. In fact, in this simple model, there are two sources that create higher correlation. The first source, the interesting one, is due to the larger coefficient in the endogenous variables, which mostly answers the question of how different β and α are; and the second, the uninteresting one, is due to the heteroskedasticity in the data. In fact, if we assume that $\beta = \alpha$, it is still the case that the correlation increases in contagious times even though the propagation of the shock, by construction, is identical.[23]

PRINCIPAL COMPONENTS. Principal components analysis is a nonparametric method that finds a linear combination of the variables of interest that maximizes the explanatory power. In any data, there are as many principal components as variables. Hence, in this example, there are two principal components.

Assume that the endogenous system represents a supply and demand equation (assume that one of the coefficients is positive and the other is negative). Figure 2 presents the two equations with some random realizations of the shocks. These realizations are distributed along the rotated ellipse (as explained before). The ellipse has two axes. The long one is the vector that represents the first principal component (the linear combination between x and y that maximizes the explanatory power of the two variables). The orthogonal smaller vector is the second principal component.

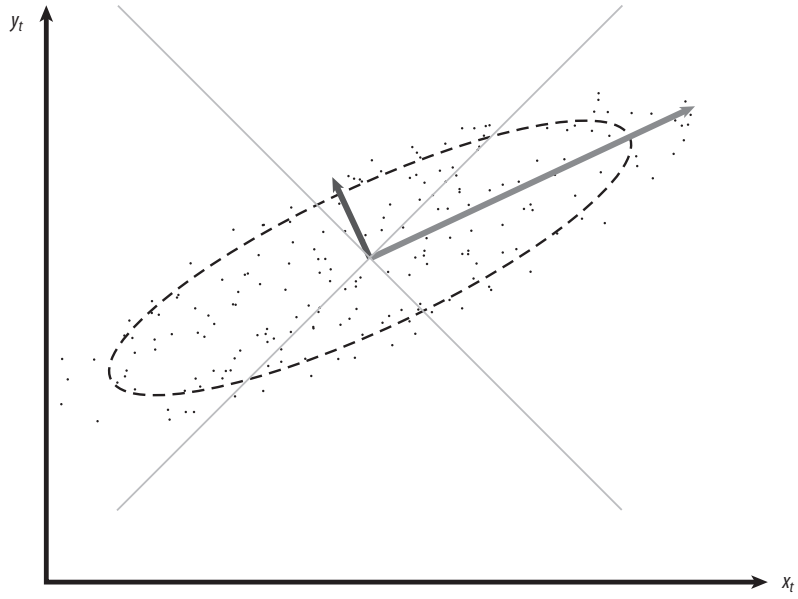
The closed form solutions for the principal component are relatively complicated, but two conclusions are easily derived from it. The variance explained by the first principal component (the first eigenvector) is

$$\lambda = \frac{1}{2} + \frac{1}{2}\sqrt{1 - 2\frac{(1 - 2\alpha^2\beta^2)\theta^2}{\left[(1+\alpha^2) + (1+\beta^2)\theta\right]^2}}. \tag{9}$$

As in the case of the correlation, the variance of the first principal component (and the eigenvector that it represents) is not a direct measure of the spillovers. Both the correlation and the principal component depend on α and β, but they are not directly measuring either. Second, and similar to what

23. This is the main point of Forbes and Rigobon (2001a, 2001b).

FIGURE 2. Principal Components



I argued before, a change in the conditional variance (θ) implies a change in the variance explained by the principal component.

In sum, the correlations and the principal components are not a good measure of the direct spillover across markets. They do depend on the structural parameters (the true measures of spillover), but they are not measuring any individual one. Second, the measurement of comovement using correlations and principal components shifts in the sample for two reasons—because the structural parameters shift or because the data suffer from heteroskedasticity. Without additional information, it is impossible to distinguish.

LINEAR REGRESSION MODELS. Linear models assume that the relationship between the variables in the two countries can be described by a simple linear model:

$$y_t = \alpha x_t + v_t. \tag{10}$$

In both models (omitted variables or endogenous variables), the OLS estimate of this equation produces a biased estimate. In other words, equation 10

is intended to represent equation 4 in the endogeneity case; and a is supposed to be an estimate of α.[24]

The first question is whether OLS captures the actual estimate (that is, whether there is a bias). As before, I concentrate on the engodenous model, but all the results are easily extrapolated to the other case. In this case, the OLS estimate of a is

$$\hat{a} = \frac{\operatorname{cov}(y_t, x_t)}{\operatorname{var}(x_t)} = \alpha + (1 - \alpha\beta)\beta\left(\frac{\theta}{1 + \beta^2\theta}\right). \tag{11}$$

Several remarks are worth highlighting. First, the estimate of α is biased, and the bias depends on the endogenous parameter β and the relative variances of the structural shocks (θ). Second, the bias can be positive or negative depending on the sign of $(1 - \alpha\beta)\beta$. Third, a change in the structural parameters (α and β) changes the estimated coefficient, but a shift in the volatility also changes the estimate. Therefore, parameter instability might be the outcome of heteroskedasticity rather than an actual structural shift. Fourth, if $\beta = 0$, then there is no problem and no bias. In this case, the estimate of α can be recovered using OLS, and, more important, the estimate does not depend on the relative variance.

A vector autoregression (VAR) has the exact same problem if the structural VAR is badly specified. In other words, when a VAR is estimated, the researcher actually estimates the reduced form. If the endogenous matrix is unknown, then the estimation problem of the VAR shares the same biases as the simple OLS specification. There is one exception, though. If the structural VAR assumes that there is no problem of endogeneity (for instance, it assumes that $\beta = 0$), then there is no estimation problem, and the VAR—as well as the OLS—provides consistent estimates. However, this is equivalent to saying that in order to solve the estimation problem due to endogeneity, the researcher just needs to assume that there is no endogeneity problem.

The ARCH and GARCH models take into account the conditional heteroskedasticity in the data, but they are not designed to deal with the problem of endogeneity or the problem of more factors than observed variables (that is, the omitted variable problem). In general, the estimation is performed on the

24. In the omitted variable case, a is supposed to capture α as well—which is the difference between the propagation of the common factor z_t to x_t and y_t. So, in both instances, the reduced-form regression is trying to summarize the spillover effect in the data.

reduced form. Hence it inherits the identification problems of endogeneity and omitted variable biases.

A final remark on linear regressions and parameter instability. Lately the literature has devoted huge effort to the estimation of parameter-varying models. My first reaction to those papers is to ask whether misspecification and heteroskedasticity could be present in the data. If that is the case, then it is difficult to interpret the estimated parameter instability as a direct consequence of fundamental parameter instability.

EXTREME OBSERVATION MODELS: EVENT STUDIES. Event studies can help ameliorate the estimation problem. The idea is that on the day of the event, it is possible to assume that θ is close to zero or infinity. The idea is that at the event, all the variation is explained by one single shock. If this is the case, then OLS or VAR produces the correct estimate. Formally, notice that in equation 11, even when α and β are different from zero, if $\theta = 0$, then the estimated coefficient is $\hat{a} = \alpha$. On the other hand, if θ is infinity, then $\hat{a} = \frac{1}{\beta}$.

In other words, when θ is zero, all the variation is explained by equation 3: $\sigma_\eta^2 = 0$. That means that OLS consistently estimates the slope in the other equation 4, which corresponds to α. Similarly, if θ is infinity, then the variation is explained by equation 4: $\sigma_\varepsilon^2 = 0$. This implies that OLS estimates of equation 3 solved for y—which is $\frac{1}{\beta}$.

Therefore, if the event is known, meaning if the researcher knows in which country and market the shock originated, then the estimation can be performed as an event study. The rationale for and intuition of this identification strategy, which was introduced by Wright, are called near identification, because the assumption that θ takes the extreme values of zero or infinity is a strong assumption.[25] I address identification through heteroskedasticity below, but because it is pertinent to the discussion of event studies, there is a simple procedure that can be used to improve the estimation of event studies when the near identification assumption is not perfect. See Rigobon and Sack for a thorough description of the methodology to improve event studies.[26]

PROBABILITY MODELS. In the literature, several attempts have been made to measure changes in the propagation mechanisms as a reflection of a change in the probability of joint events—usually large negative realizations. The first

25. Wright (1928).
26. Rigobon and Sack (2008).

example using conditional probabilities to measure contagion can be found in Eichengreen, Rose, and Wyplosz.[27] Copulas are also very common in the literature. Some of the early attempts try to characterize the joint distribution at the tails.[28]

The general intuition is twofold. First, to measure spillovers, the conditional probability or the copula measures the behavior of the markets after or during extreme observations. This measurement is supposed to capture the strength of the spillover across two markets. Second, to determine shift contagion (parameter instability), the conditional probabilities are compared between small and large shocks or between positive and negative shocks. The problem is that, as before, these measurements do not capture structural parameters.

For example, in the endogenous model and the omitted variables models, the conditional probability at the tails can be driven either by ε_t or by η_t, or by any combination of the two; and the conditional probabilities, as well as the joint distributions, can be described by many combinations of the structural parameters α and β. Therefore the joint distribution is not a description of the true spillover in the data.

Newer Methods

I personally believe there is not a single technique that can solve the empirical challenges that the literature on contagion is trying to tackle. What is worse is that the theories are far too restrictive, and a structural estimation approach is therefore bound to be insufficient. In this section, I summarize three techniques that partially address some of the problems. In particular, I separate the estimation problem from the parameter stability question. Spillovers can be under the assumption of parameter stability. The relevant methodology is called identification through heteroskedasticity, which deals directly with the estimation problem. On the other hand, if the only question of interest is one of parameter stability—regardless of the actual point estimates—then there are two possible methodologies, one based on heteroskedasticity and the other on quintile regressions. However, if the point estimates are important and their stability is also to be tested, then I do not know of any methodology that can provide a satisfactory answer.

I organize this discussion by first addressing the estimation of spillovers (or contagion) conditional on parameter stability. I then address parameter stability.

27. Eichengreen, Rose, and Wyplosz (1996).
28. The literature on copulas is very large. One of my preferred papers is Rodríguez (2007).

SPILLOVER ESTIMATION: IDENTIFICATION THROUGH HETEROSKEDASTICITY. The identification problem has been at the root of some of the most important innovations in econometrics in the last century. All the problems can be boiled down the demand-supply estimation problem. Instrumental variables, regression discontinuity, natural experiments, and randomized controlled trials are all solutions that have been devised in that simple framework. Interestingly, most of the solutions were already suggested almost a century ago in a book on agricultural economics written by Philip Wright.[29] In the appendix of that book, Wright discusses three possible techniques to solve the problem of the demand-supply estimation. The first is what is now known as instrumental variables in economics. The second technique is known as near identification—which is the precursor of event studies, regression discontinuity, and randomized controlled trials. The third technique provides the intuition of identification through heteroskedasticity. So technically, all the procedures used in econometrics were invented in 1928 in a book about animal oils.[30] Of these techniques, the first took over the profession with a vengeance, the second has just started to take over development economics, corporate finance, and other areas of economics, and the third one has just barely been used—and the few applications are in macroeconomics and international economics.[31]

First, let me explain the identification problem. Equations 3 and 4 describe the behavior of the data entirely with four parameters/variables: two shocks, ε and η, and two parameters, α and β. These four constitute the unknowns of the system. The problem of identification arises because the researcher has three equations in four unknowns. The observable variables x and y have mean zero, and in the data only three moments can be estimated—all from the variance-covariance matrix.

Second, what do the solutions tend to do? Every solution needs to "create" an additional equation. For instance, the exclusion restriction in the instrumental variable approach boils down to assuming that one parameter is zero (the exclusion assumption). Randomized controlled trials assume that all the variation is due to the treatment—again, this is implicitly assuming

29. See Wright (1928)

30. This has always made me feel as if the techniques we use are way less sexy and cool than I originally thought.

31. For the theoretical derivations, see Rigobon (2003) for the general case and Sentana and Fiorentini (2001) for an excellent derivation in the context of ARCH models. For applications in monetary policy and macroeconomics, see Rigobon and Sack (2003, 2004, 2008). For applications in the measurement of spillovers, see Ehrmann, Fratzscher, and Rigobon (2011) and the many papers Marcel Fratzscher has written.

that there is no feedback effect. It is a very reasonable assumption when the experiment is properly designed. All these solutions are making a parameter assumption (usually that a parameter is equal to zero). The identification through heteroskedasticity has a slightly different flavor.

The easiest way to explain how identification through heteroskedasticity works is to show the system of equations. Assume that the parameters are stable and that the data have heteroskedasticity. For simplicity, assume that there are two heteroskedastic regimes. In this case, it is possible to estimate one covariance matrix in each regime:

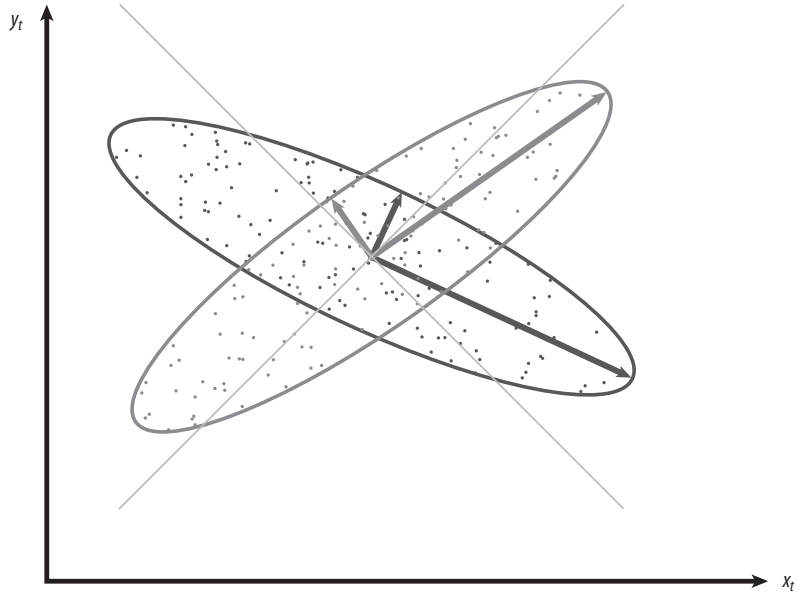
$$\mathbf{\Omega}_1=\begin{bmatrix}\operatorname{var}\left(x_{t,1}\right) & \operatorname{cov}\left(x_{t,1},y_{t,1}\right)\\ & \operatorname{var}\left(y_{t,1}\right)\end{bmatrix}=\frac{1}{\left(1-\alpha\beta\right)^2}\begin{bmatrix}\sigma^2_{\varepsilon,1}+\beta^2\sigma^2_{\eta,1} & \alpha\sigma^2_{\varepsilon,1}+\beta\sigma^2_{\eta,1}\\ & \alpha^2\sigma^2_{\varepsilon,1}+\sigma^2_{\eta,1}\end{bmatrix};$$

$$\Omega_2=\begin{bmatrix}\operatorname{var}\left(x_{t,2}\right) & \operatorname{cov}\left(x_{t,2},y_{t,2}\right)\\ & \operatorname{var}\left(y_{t,2}\right)\end{bmatrix}=\frac{1}{\left(1-\alpha\beta\right)^2}\begin{bmatrix}\sigma^2_{\varepsilon,2}+\beta^2\sigma^2_{\eta,2} & \alpha\sigma^2_{\varepsilon,2}+\beta\sigma^2_{\eta,2}\\ & \alpha^2\sigma^2_{\varepsilon,2}+\sigma^2_{\eta,2}\end{bmatrix}.$$

There are six unknowns in the system: the two parameters (α and β) and the four variances ($\sigma^2_{\varepsilon,1}$, $\sigma^2_{\varepsilon,2}$, $\sigma^2_{\eta,1}$, and $\sigma^2_{\eta,2}$). Thus there are six equations in six unknowns. This means that the system of equations is just identified. Even though the system is underidentified in each regime (that is, there are fewer equations than unknowns), the system as a whole is identified. The key assumptions are two: that the structural shocks are indeed structural (that is, they are uncorrelated) and that the parameters are stable. In the end, the parameter stability allows the heteroskedasticity to add additional equations—which helps solve the identification problem.

The intuition behind the identification through heteroskedasticity comes from the rotation of the residual ellipses. When the variances change, for the same parameters, the ellipses rotate. Figure 3 shows two cases: one when the demand shocks dominate (thick gray curve) and one when the supply shocks dominate (thick black curve). In particular, when the demand shocks dominate, the elipse approximates the supply curve. In fact, it is identical to the supply curve if the variance of the demand is infinite relative to the supply. Conversely, when the supply shocks are larger, the long axis of the elipse tilts toward the demand curve. It is this rotation of the ellipses when the relative variances shift that provides the identification.

FIGURE 3. Identification through Heteroskedasticity



It is instructive to restate the underlying assumptions: structural shocks are uncorrelated (quite uncontroversial), and parameters need to be stable across the regimes. It is thus a good technique for measuring spillovers.

PARAMETER INSTABILITY: DCC. I designed a simple extension of the identification through heteroskedasticity methodology to test for parameter instability, called the determinant of the change in the covariance matrix (DCC). This is mainly an overidentification test. The key assumption is that some of the shocks are heteroskedastic, but others are homoskedastic. As before, assume that there are two heteroskedasticity regimes, but the researcher knows that one of the shocks is homoskedastic. In the context of contagion, this is equivalent to assuming that the crisis is known to have originated in a specific country, and the prior is that the other country's shocks are unaffected by the crisis. In the European case, this is similar to assuming that in 2010 the shocks to Greece, Ireland, Portugal, and Spain were more volatile, but that the shocks to France, Germany, and the Netherlands were equally volatile. This is a strong assumption, but one that allows testing for parameter instability in this context.

The method can be described in the two-country case. Assume that country *y*'s shocks are homoskedastic. This implies that $\sigma_{\eta,1} = \sigma_{\eta,2}$. Subtracting the two covariance matrices yields

$$\begin{aligned}
\boldsymbol{\Omega_1} - \boldsymbol{\Omega_2} &= \frac{1}{(1-\alpha\beta)^2}\begin{bmatrix} \sigma^2_{\varepsilon,1} + \beta^2\sigma^2_{\eta} & \alpha\sigma^2_{\varepsilon,1} + \beta\sigma^2_{\eta} \\ & \alpha^2\sigma^2_{\varepsilon,1} + \sigma^2_{\eta} \end{bmatrix} \\
&\quad - \frac{1}{(1-\alpha\beta)^2}\begin{bmatrix} \sigma^2_{\varepsilon,2} + \beta^2\sigma^2_{\eta} & \alpha\sigma^2_{\varepsilon,2} + \beta\sigma^2_{\eta} \\ & \alpha^2\sigma^2_{\varepsilon,2} + \sigma^2_{\eta} \end{bmatrix} \\
&= \frac{1}{(1-\alpha\beta)^2}\left(\sigma^2_{\varepsilon,1} + \sigma^2_{\varepsilon,2}\right)\begin{bmatrix} 1 & \alpha \\ & \alpha^2 \end{bmatrix} \\
\left\|\boldsymbol{\Omega_1} - \boldsymbol{\Omega_2}\right\| &= \frac{1}{(1-\alpha\beta)^2}\left(\sigma^2_{\varepsilon,1} - \sigma^2_{\varepsilon,2}\right)\left\|\begin{bmatrix} 1 & \alpha \\ & \alpha^2 \end{bmatrix}\right\| = 0.
\end{aligned}$$

Notice that the determinant of the change is not full rank. Now assume in the exact same case (one heteroskedastic shock) that one parameter changes (for the purpose of illustration, assume that α moves around); then the shift in the covariance matrix is (which cannot be simplified)

$$\begin{aligned}
\boldsymbol{\Omega_1} - \boldsymbol{\Omega_2} &= \frac{1}{(1-\alpha_1\beta)^2}\begin{bmatrix} \sigma^2_{\varepsilon,1} + \beta^2\sigma^2_{\eta} & \alpha_1\sigma^2_{\varepsilon,1} + \beta\sigma^2_{\eta} \\ & \alpha_1^2\sigma^2_{\varepsilon,1} + \sigma^2_{\eta} \end{bmatrix} \\
&\quad - \frac{1}{(1-\alpha_2\beta)^2}\begin{bmatrix} \sigma^2_{\varepsilon,2} + \beta^2\sigma^2_{\eta} & \alpha_2\sigma^2_{\varepsilon,2} + \beta\sigma^2_{\eta} \\ & \alpha_2^2\sigma^2_{\varepsilon,2} + \sigma^2_{\eta} \end{bmatrix} \\
\left\|\boldsymbol{\Omega_1} - \boldsymbol{\Omega_2}\right\| &\neq 0.
\end{aligned}$$

If there are *N* endogenous variables with *N* structural shocks, then, if the heteroskedasticity in the data is explained by $S < N$ shocks, and if and only if the parameters are stable, the determinant of the change in the covariance matrix is zero. This test is quite powerful, and it has been tested in several contexts to determine its empirical size and power.[32]

32. See Rigobon (2000) and Dungey and Fry (2004).

PARAMETER INSTABILITY: QUANTILE REGRESSIONS. Finally, a very simple test based on quintile regressions tests for parameter instability. Contagion and parameter instability create a nonlinearity in the OLS estimates. In other words, conditional on larger volatility and different propagation mechanisms, the biases in the simple OLS estimates are different across times. It is possible to test for this nonlinearity in at least three different ways. One very interesting approach relies on quantile regressions. In this case, the purpose is to evaluate the linear coefficient conditional on the different realizations of x_t. This test allows for an unrestricted form of nonlinearity (conditional on the quintile, of course). This procedure can deal with the heteroskedasticity in the data and with parameters being different between positive and negative realizations or between small and large realizations—which presumably will be pulled into different quintiles.

The test is straightforward. If the parameters are stable then the quintile regressions should offer estimates that are not statistically different from each other, while if there is parameter instability, the coefficients shift across the quintiles. See Caporin and others for an application to the European crises.[33]

Final Remarks

The empirical study of spillovers and contagion is one of the most complicated applied questions the literature needs to address. Two features of the data are prominent in this challenge. First, every model of spillover and contagion implies that observed variables are endogenous or that omitted variables are present (or both). Second, the financial data suffer from heteroskedasticity. The former is a problem of misspecification, while the latter should be relatively easy to deal with. However, the combination of these two problems implies that the degree of misspecification changes throughout the sample; the biases thus shift through time. Consequently, correlations, principal components, OLS regressions, event studies, VARs, ARCH and GARCH models, probit, logit, and copulas are all biased and time-dependent. This is not because the structural parameters of the data-generating process are unstable but because the models are all misspecified, and the misspecification is shifting through time. Therefore, the standard methods cannot provide satisfactory answers to simple questions such as "What is the propagation of shocks from country 1 to country 2?" or "Are the spillovers stable through time?" or "Does contagion exist?"

33. Caporin and others (2018).

The problem is even more complicated because there are no natural experiments or instruments that could solve the identification problem. Therefore, the problem is left to the typical macroeconomic identification strategies, which depend on Cholesky decompositions, or to the imposition of some "reasonable" parameter restrictions—which in the end are not that reasonable after all.

From the policy perspective, it is fundamental to have some guidance about the strength of contagion within countries and across the world. Latin American countries have been suffering from these shocks since the beginning of the twentieth century—since nitrates were synthesized in a laboratory and the price of guano dropped to almost nothing. It is hard for Latin America to build the institutions that could provide macroeconomic stability if the structure is unknown. This paper unfortunately does not offer a final answer to the problem. It provides, however, an avenue to think about the problem from two different perspectives. As said, there is no single technique that can deal with all the empirical problems at the same time. Therefore, I have discussed three techniques that address the problem partially and through two different lenses. First, if the researcher is willing to accept the assumption that parameters are stable through time, then the problem of identification can be solved by appealing to the identification through heteroskedasticity. This is, to me, the best method to estimate spillovers across markets and countries. It can be used to estimate the financial network that exists among banks within a country—that is, financial linkages among the different actors in the economy.

Second, if the question is about parameter stability, there are two possible avenues. The first is to assume that the heteroskedasticity of the data is explained by a subset of the structural shocks. This is equivalent to assuming that some of the structural shocks are homoskedastic. In this case, there is a relatively powerful test that can determine whether or not parameters are stable. The test is in the spirit of an overidentification test. The second test is to rely on reduced-form estimation of a quintile regression. This procedure tests whether parameters are stable across positive versus negative shocks and also between large and small realizations. These can be used to understand whether or not a change in monetary policy in the United States is propagated differently to emerging markets. It would be hard to estimate the exact strength of the propagation, but the question of whether it has shifted could be addressed.

These are incomplete answers to the problems of contagion. More research is obviously needed. Continuing to use the standard methods is, however, more dangerous than having partial answers.

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