Contagion, Spillover, and Interdependence

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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 produces time varying biases. Several empirical methodologies are evaluated from this perspective: non-parametric techniques such as correlations and principal components, as well as parametric methods such as OLS, VAR, event studies, ARCH, Non-linear regressions, etc. The paper concludes that there is no single technique that can solve the full fledge problem and discusses three methodologies that can partially address some of the questions in the literature.

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1 Introduction

Almost every paper on contagion starts with a definition on 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 across time. For example, at some time I had defined contagion strictly as the "unexpected" or "surprising" component of the transmission of shocks across countries; at others, as a change in the behavior during crises; and lately as purely any form of propagation across countries irrespectively of the circumstances. Interestingly, whatever the definition of contagion I had at any particular point in time, it was the case that all of them were (and still are) a common feature of the data. This was good because I could always claim that I found evidence of "contagion" in the data. Here, however, instead of claiming victory I would like to concentrate on what have we learned, and what are the challenges ahead. Therefore, let me use the word "contagion" and "spillovers" as describing very loosely 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.

On a more serious note, I believe the distinction between contagion, and spillover (or interdependence or linkages) is tenuous. The reason is that 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 un-modeled. In my view, this is rather unsettling. The definition of what constitutes spillover and what is contagion change by just rewriting the model.

Furthermore, even when all the interdependence is modeled still there is a question about magnitude. For instance, for some researchers and market participants the relationship between the US and Canada is so strong that if the stock markets were correlated in 75 percent they might not find it surprising, others will find such degree of correlation too high – hence surprising and therefore "contagious". In other words, if the strength in the co-movement is of the order of magnitude of the researcher's believes, then it is called spillover, but if the co-movement 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, there are two aspects that I believe the profession agrees. First, spillovers are always present – in good and bad times; on the other hand, 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". It occurs when the propagation of shocks intensifies during those times — and it is inherently a parameter instability feature in the data. This has motivated me to look at the problem by splitting the empirical problem as follows: the estimation of the transmission mechanism during normal times (which could or not be contagious), and the estimation of the change in the transmission mechanisms after certain macroeconomic event (shift-contagion).

The literature has devoted an enormous amount of attention studying what drives shocks across countries, and what are their most important propagation mechanisms. Although evidence of contagion can be traced back to more than a century ago, where some of the most dramatic

events were the Great Depression and the Debt Crises in the early 80's, 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 − academics and practitioners. In the early 80's Costa Rica and Mexico defaulted on their debt, and a cascade of countries in Latin America started defaulting and renegotiating their sovereign debts. How was it possible that countries so seemingly unrelated economically — with small trade connections — could all be acting together? By the early 90's were all joined financial markets after the Brady Plan they encounter themselves dwelling in a crises after Mexico devalues in 1994. A country that had followed the advise of economic orthodoxy suffer a currency crisis, and Argentina followed. An even that was so surprising that Guillermo Calvo coined the "Tequila Crisis". 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 that a panel said "whenever anyone in the world sneezes, Argentina gets pneumonia".

There were several features of these events that granted such interest. First, previous crises implied propagations from large countries to smaller ones − the case of the Great Depression is a good example. On many grounds the fact that a large country has impact on a smaller economy isn't surprising at all. The late 90's 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 80's and the subsequent collapse of Finland was considered an example in which the collapse in one country affected the exports of the other main trading partner. So, again, there was nothing terribly surprising about the transmission of shocks across two highly interrelated economies. In the late 90's crises, however, countries with very small trading 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 that occurred in 1994. Even less relationship existed between Russia and the pair that suffer the most in Latin America: Argentina and Brazil in 1998. Finally, except for the fact that the countries belong to the same region, there was little in common among the MIT countries in 1997 - Malaysia, Indonesia and Thailand.[1]

Recently the subprime crisis in the US 2008 and the fiscal crises in Europe in 2010 have renewed the interest on contagion, and more importantly, on its prevention. These crises share many of the emerging market crises features. According to the BIS the size of the subprime securitized assets in 2007 was 860 Billion US dollars. Meanwhile, the size of the whole financial sector (formal and shadow financial sectors) was north of 25 trillion dollars. So, the subprime market was less than 4 percent of the financial system. Still it had a massive impact in the US and around the world. As most contagious events, a shock to a seemingly small and isolated market had a global impact.

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[1]It is important to recognize that not all crises are *equally* contagious. In contrast with the crises I have highlighted, I find it interesting that some crises at the beginning of the 21st century had a completely different behavior. Indeed, the lack of contagion after the Brazilian 1999, Turkish 2000, and Argentinean 2002 crises is apparent even to a casual observer. This evidence is reported in Miller, Thampanishvong, and Zhang (2003) and in 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.

Europe's fiscal crises share the same characteristics. Greece is after all a tiny proportion of GDP, Trade, and Financial Flows in the Euro area. Even though they have strong trading and financial ties it is hard to explain the amount of anxiety in the markets that caused the Greek Tragedy.

Finally, by 2018 a small increase in interest rates in the US is causing havoc in emerging markets. By mid 2020 I would not be surprised that the profession will encounter another contagious event worth studying. 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 not the first review of the methodologies to measure spillovers or contagion, and many very good reviews have been out there for quite some time. My preferred ones are the following: A concise review of the theories is presented in Claessens, Dornbusch, and Park (2001). The theories, and some of the early methods used in contagion have been summarized in Goldstein, Kaminsky, and Reinhart (2000), Forbes and Rigobon (2001a) and Forbes and Rigobon (2001b). For a critical view of the empirical methods used by earlier papers see Rigobon (2002), and for a recent survey on the empirical strategies in contagion see Dungey and Fry (2004).

The objective of this paper is to discuss the methodologies that have been used to measure contagion and spillovers; (i) what are their advantages and disadvantages, and (ii) to introduce 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 are 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 from methodologies that are not robust, the decisions are clearly wrong. A recent document published by the BIS comes to mind.[2]

1.1   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 in general cannot be estimated with a simple OLS. I come back to this point in section 2. 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, it is just the context in which the transmission of shocks occur. Three features are quite important, and I will repeatedly come back to them throughout my discussion. These are also three characteristics that are relatively uncontroversial.

First, the data has heteroskedasticity and in particular contagion events is associated with massive increases in volatility. Most of the literature studies spillovers in financial variables – that suffer from conditional heteroskedasticity. In the case of contagion, it is common for variances to increase tenfold. This is the case for financial variables as well as real variables. In other words, stock markets, interest rates, exchange rates become massively more volatile; and credit, consumption, investment, and GDP also experience increases in variance. Heteroskedasticity is a fundamental

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[2]See **?**. The book has 120+ pages, and I believe most of the models discussed there are based on dubious facts.

characteristic of the data where spillovers and contagion is evaluated. From the empirical point of view this characteristic represents on 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 a misspecified regression changes the degree of misspecification when the volatilities of the shocks move around. This implies that the researcher has a problem determining if what he/she is estimating is 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 importantly that correlations increase in a contagious crisis. In the following section I will criticize correlations as a measure of contagion or spillover, but any theory or empirical strategy has to account that in the reduced form correlations increase during crises.

Second, contagion events tend to be short lived. In fact, the frequencies in which the event is measured require high frequency data. [3] Contagion tends to propagate a crisis in a matter of weeks, and takes months to be resolved. 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. Recently, the 2008 US crisis has taken forever for the US to resolve, but its contagion to emerging markets only took place between September 2008 to mid 2009, and by mid 2010 emerging markets were already growing.

Finally, spillovers are inherently evaluated as a financial phenomena. Stock prices, interest rates, and exchange rates are the scape valve in the system. Of course, GDP, consumption, investment, trade, and financial credit are also affected. Nevertheless, the detection of spillovers required relatively high frequency data and it is in the financial variables where most of the empirical research ends up occurring. Very few theories, as I will explain later, make this connection explicitly. 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.

2   Short Review of the Theories of Spillovers and Contagion

The objective of this section is to review the theories behind the international propagation of shocks. The theoretical literature can be divided in three broad views: fundamental, financial, and coordination.[4]

2.1   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 macro similarities. To me, the most prominent papers are Gerlach and Smets (1995), Corsetti, Pesenti, Roubini, and Tille (1998), Corsetti, Pericoli, and Stracia (2003), and Basu (1998).

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[3]Where high frequency for macroeconomists mean days or weeks.

[4]There are many surveys of the theories behind contagion. See Claessens, Dornbusch, and Park (2000) and **?** for surveys on the theories.

For example, on the bilateral trade explanation (which happened to be the first paper on contagion! Gerlach and Smets (1995)), if a country has a crisis and its consumption declines, then their imports are likely to decline as well. Therefore, the trading partners experience a decline in the demand of their exports: either their prices drop – a deterioration of the terms of trade – or they reduce production. In both cases, their GDP declines, there is a recession, and quite likely a depreciation. In fact, all international real business cycle models exhibit this transmission channel.

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

Furthermore, I have emphasized trade but monetary policy and other macroeconomic policies are also linked by trade. Therefore, the transmission is not exclusively through relative prices but through monetary policy coordination, and other similar macroeconomic policies. For example, on January 2016 the US is considering increasing the interest rates which will force other countries to evaluate their monetary policy paths. The increase in the US is a common shock to the world and therefore several emerging markets suffer the negative consequences. In Latin America, between 2016-2018 Argentina, Brazil, and Venezuela are going to massive recessions — although Venezuela 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.[5]

The theories based on fundamental transmission mechanisms were used to explain the transmission from the Great Depression, and for the 70's and 80's crises in Europe. 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.[6]

Recently, the finance literature put together trade and asset price within a single framework. Pavlova and Rigobon (2007) analyze in a general equilibrium model, the interactions between international asset prices, the exchange rate, and trade in goods. In that paper , we confirm that the same intuitions derived in exchange rate markets can be carried out to equity and bond prices. Additionally, Martin (2013) studies asset prices in a multi country model and shows how shocks from one country change conditional correlations exactly in the spirit of contagion. These two papers have been able to put together the simple intuitions of trade within asset price models.

2.2 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 in which financial services can be provided to different countries – that ex-ante might have been seen as independent. This theory

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[5]I know, not much of a theory.

[6]See Forbes (1999) and Forbes (2000) for two good examples evaluating the strength of the fundamental channels of contagion. See also Gregorio and Valdes (2001) for an alternative empirical methodology

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 allows for a clearer analysis of the reasons behind the transmission mechanisms.

In general, the argument of the "contagion" goes as follows. Assume that two countries receive financial services from a third party. The financial services can be direct lending, insurance, provision of liquidity, etc. 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. The reduction in the service to the second country has real effects because the presence of financial imperfections. In the end this affects asset prices, 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 theory of common lender advanced by Goldstein, Kaminsky, and Reinhart (2000) and Kaminsky and Reinhart (2002) assumes that a single bank is lending to two countries whose outputs are, in principle, unrelated. A crisis in one country affects the balance sheet of the bank, forcing it to stop lending to the second country. So, even if two countries are independent from the real linkages point of view, still their international flows comove, as well as other macro 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 effect 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 (2002). [7] 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 either behave as if they have a higher degree of risk aversion or are subject to margin calls. Both reasons forces 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 Russia 1998 and the aftermath of LTCM.

Finally, new theories of financial spillover have highlighted the network across financial institutions as the vehicle of propagation (See Allen and Gale (2000) form the first attempt, and see Elliott, Golub, and Jackson (2014) for a theoretical foundation of contagion through a network). This is a promising area of research although the measurement of the interconnections in the network are 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.

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[7]See also Yuan (2005), and Mendoza and Smith (2002) for the theories studying margin calls and their real effects. As well as Kyle and Xiong (2001) for a theory where wealth shocks create contagion, and Gromb and Vayanos (2002) for a model where market participants face portfolio constraints.

2.3 Coordination View

The third class of theories is based on coordination failure. The coordination view studies investors and policy makers behavior and coordination problems as the explanation behind contagion. In this theories most of the contagion comes from investors' actions. It is usually a learning or herding problem.

The theories based on the coordination of markets participants include those explanations 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 can drive market participants to withdraw resources jointly across countries. Not only investors are affected by informational problems, it is also conceivable that also policy makers coordinate and decide to abandon a particular macroeconomic policy – usually the exchange rate regime – when another country follows 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.

For the first multiple equilibrium framework of contagion see Masson (1997). In this case contagion is defined as the shifting from a good to a bad equilibria. Two nice applications of the herding informational cascades to capital flows are Calvo and Mendoza (2000) and Chari and Kehoe (1999). In these papers the spillover occurs because information in one country leads investors to take actions in the other. Theories of learning have also been used to explain contagion (in particular).[8]

Finally, one of my preferred theories of spillover is political contagion. In his paper Drazen (1998), he analyses the abandonment of the Exchange Rate Mechanisms (ERM) in 1991 in Europe. The intuition is that belonging to the ERM was equivalent to belonging to a "gentleman's club". Belonging to the club provides benefits in terms of reputation and "class". However, belonging to the club requires significant sacrifice. In Drazen's model, once a country decides to abandon the club two things occur: First, the cost of abandoning for the next gentleman is smaller. Second, the value of remaining in a smaller club is also smaller. Therefore, the abandonment of once country increases the likelihood that a second one drops 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. Chavez came to power in Venezuela in 1999 (elections in 1998) at a time in which Latin America was mostly following policies that were close to the center — in the political spectrum. The political success of Chavez propagated to other countries in different degrees. Who suffer the most? Bolivia, Brazil, Ecuador, El Salvador, and of course Argentina. However, by 2018, it could be argued that all Latin American countries had to deal with the threat of extreme populism.

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[8]Two papers come to mind Kodres and Pritsker (2002) and Rigobon (1998).

3   Measuring Contagion and Spillover

The theoretical papers on international spillover have two important empirical implications. First, most of the models exhibit non-linearities.[9] Second,all the models imply either endogeneity or omitted variables. For example, asset pricing models – such as Pavlova and Rigobon (2007) and Pavlova and Rigobon (2008) – imply reduced form factor models that are similar to the reduced forms obtained from endogenous systems of equations. 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.

It is quite usual to compare the measurement of international spillover to the notions of contagion that have been developed in the medical literature. However, because of the theoretical implications just highlighted, the problems are very different. In medicine there are two approaches: a direct measurement of contagion, and an indirect one. In other words, how do we usually estimate the degree of contagion of a particular virus? One procedure relies on blood tests where the presence of the virus is detected, while the second one concentrates on the symptoms.

In the direct measurement, the speed and intensity at which the virus transmits from one individual to another is directly evaluated by the concentration of the virus in the blood stream. This procedure, however, requires measuring the presence of the virus. In international economics this is equivalent to observing the fundamental force that drives the spillover. In other words, this requires economists to measure directly risk appetite, contingent contracts, incentives, the information each agent possess, etc. In practice, this methodology 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, etc. Second, and even worse, the literature rarely agrees on what needs to be measured. Therefore, even if we were able to measure a particular fundamental determining interest rates across countries, it is not clear that such channel is the one most of the literature care about. In sum, it is hard to see the "virus".

The second procedure is to observe and evaluate the symptoms. Assume that one of the symptoms of the virus is high fever (for the sake of the discussion lets assume the threshold is 104). 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 104, given that another person in the population has 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, etc. Shocks that indeed affect the whole population. These correlations and frequencies define what is considered "normal". If a virus is introduced into the city it is expected that the frequency of 104 temperatures increases, and that the conditional probabilities 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 co-movement in "normal" times that are intensified during "contagious" time. The idea is, therefore, to evaluate how different the propagation is during a contagious event, from the propagation that exists in normal times. The

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[9]However, some papers linearize such relationships and estimate simple linear functions, while other techniques are more agnostic.

problems of the indirect procedure are several: what defines "normal"? how can we evaluate the propagation in "contagious" times?

This section is divided in three relatively technical subsections. It is impossible to discuss the weaknesses of the empirical methods without a formal framework. Subsection 3.1 discusses the models used to highlight the biases caused by endogeneity and omitted variables. Subsection 3.3 discusses the bias in some of the standard methodologies used to estimate contagion. I pay especial attention to the parameter instability that is spuriously arises in the presence of heteroskedasticity. Finally, subsection 3.4

3.1 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 the structural shocks, and the parameters are the structural parameters. These are the coefficients and shocks that describe the underlying linkages across countries and financial variables. These are supposed to capture the theories of international spillover and contagion.

3.1.1 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,

$$x_t = z_t + v_t + \epsilon_t \tag{1}$$
$$y_t = \alpha z_t + \beta v_t + \eta_t \tag{2}$$

where z_t is the factor in "normal" times; while 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 where ϵ_t and η_t are some country specific shock.[10] 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-times" shock: $\sigma_v^2 > \sigma_z^2$. This is implicitly capturing the fact that contagious events exhibit higher volatility. Also, we assume that conditional on the same variance, contagious events are propagated 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 co-movement (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.

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[10]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.

3.1.2 Endogenous Model

There is an endogeneity representation that shares the same reduced form,

$$x_t = \beta y_t + \epsilon_t \tag{3}$$
$$y_t = \alpha x_t + \eta_t \tag{4}$$

with reduced form,

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

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

In fact, these two models have the exact same implications on the difficulty of estimating spillovers and contagion in the data. Before proceeding to the discussion of each of the methodologies it is worth to review two concepts: First, the graphical representation of the joint residuals in these models always takes the form of a rotated ellipse. Second, the rotation is summarized by the variance–covariance matrices in each of these models.

3.2 Graphical Representation of the Distribution of Errors and Covariance Matrices

3.2.1 Ellipses

In equations 1 and 2 and equations 3 and 4, the only meaningful moment we can compute to estimate the degree of contagion is the covariance matrix. An important question is then, what does the covariance matrix represent? The errors in these models are distributed as a multinomial and their contours are ellipses. To fix concepts, let us start with a simple endogenous system of equations 3 and 4

$$x_t = \beta y_t + \epsilon_t$$
$$y_t = \alpha x_t + \eta_t$$

where α and β are the coefficients summarizing the endogeneity; and where the two errors (ϵ and η) are called the structural shocks, they are independent (have no correlation) and the variance of ϵ is σ_ϵ^2 and η is σ_η^2. The covariance matrix between x and y represents a rotated elipse. In other words, the 95th percentile of the errors is distributed as a rotated elipse. We can solve for two independent normal distributions from the structural equations as follows (with some abuse of notation)

$$\phi_1 = \frac{x_t - \beta y_t}{\sigma_\epsilon} \sim N(0,1)$$
$$\phi_2 = \frac{y_t - \alpha x_t}{\sigma_\eta} \sim N(0,1)$$

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

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

Notice the similarity with the general equation of a rotated ellipse

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

The two axes of the ellipse cannot be computed in closed form solution, but they depend on the slope of the curves (structural parameters) as well as the relative variances of the shocks. In Figure 1 a graphical representation is shown. The blue curve represents the supply and the red is the 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 90th 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 shocks to the supply, hence, 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.
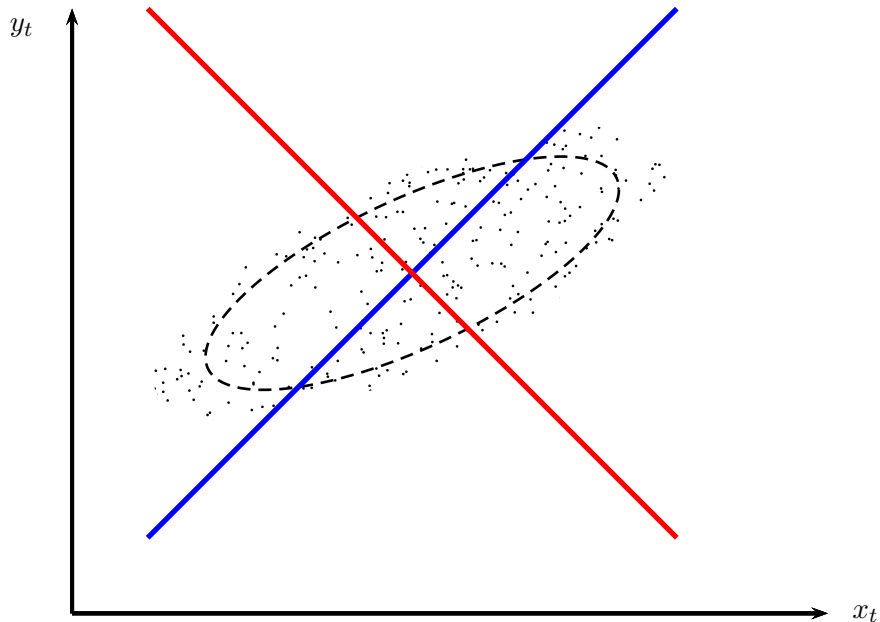

Figure 1: Distribution of Errors

3.2.2 Covariance Matrices

The form of the ellipse is also summarized by the covariance matrix computed in the reduced form. Additionally, most of the methodologies we study are based on the covariance matrix. Therefore, all the sources of bias can be tracked to it. Finally, as mentioned previously, in these two models the only statistic that can be computed from the data – that allows to recover 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

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\begin{aligned}
var(x_t) &= \sigma_z^2 + \sigma_v^2 + \sigma_\epsilon^2 \\
var(y_t) &= \alpha^2 \sigma_z^2 + \beta^2 \sigma_v^2 + \sigma_\eta^2 \\
covar(x_t, y_t) &= \alpha \sigma_z^2 + \beta \sigma_v^2
\end{aligned}
$$

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

$$
\begin{aligned}
var(x_t) &= \frac{1}{(1-\alpha\beta)^2} \left(\sigma_\epsilon^2 + \beta^2 \sigma_\eta^2 \right) \\
var(y_t) &= \frac{1}{(1-\alpha\beta)^2} \left(\alpha^2 \sigma_\epsilon^2 + \sigma_\eta^2 \right) \\
covar(x_t, y_t) &= \frac{1}{(1-\alpha\beta)^2} \left(\alpha \sigma_\epsilon^2 + \beta \sigma_\eta^2 \right)
\end{aligned}
$$

3.3 Empirical Strategies

This section discusses the biases and possible solutions for the different empirical methodologies. It is organized as follows: First, I discuss non-parametric techniques such as correlation and principal components. Second, I analyze the biases that exist in linear models - such as OLS, VAR's, ARCH, GARCH, and event studies. Third, I discuss the bias in limited dependent models.

I will concentrate all the discussion in the endogeneity model. The results are easily replicated in the omitted variable case. In almost every case we analyze the statistic we are computing and its dependence on the relative volatility of the structural shocks. Let us define

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

I describe the biases that arises in correlations and principal components methodologies. One advantage of these two methods (as well as copulas) is the fact 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 it depends on the relative variances of the shocks. As I mentioned before, heteroskedasticity is a characteristic of the data, and therefore the bias will be shifting in the sample — and hence estimates will be unstable. Finally, I end the discussion with probability models – another non-parametric approach that benefits from its simplicity.

3.3.1 Correlation

Correlation is one of the preferred methodologies to capture or measure co-movement. It is commonly argued that when the correlations shift it is due to changes in structural parameters. This is not always correct. The correlation is not an unbiased estimator when volatilities changes. Second, and more importantly, 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, and therefore, it does not have a structural interpretation. Most economists understand this in seconds, but this 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. One, 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 a second one, the uninteresting one, is due to the heteroskedasticity in the data. In fact, if we assume that $\beta = \alpha$ still it is the case that the correlation increases in "contagious" times even though the propagation of the shock by construction is identical.[11]

3.3.2 Principal Components

Principal components is a non-parametric methodology 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 our example there are two principal components.

Assume that our endogenous system represent a supply and demand equation (assume that one of the coefficients is positive and the other is negative). In figure 2 I present 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 axises. 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 there are two conclusions that are easily derived from it. The variance explained by the first principal component (the first eigenvector) is

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

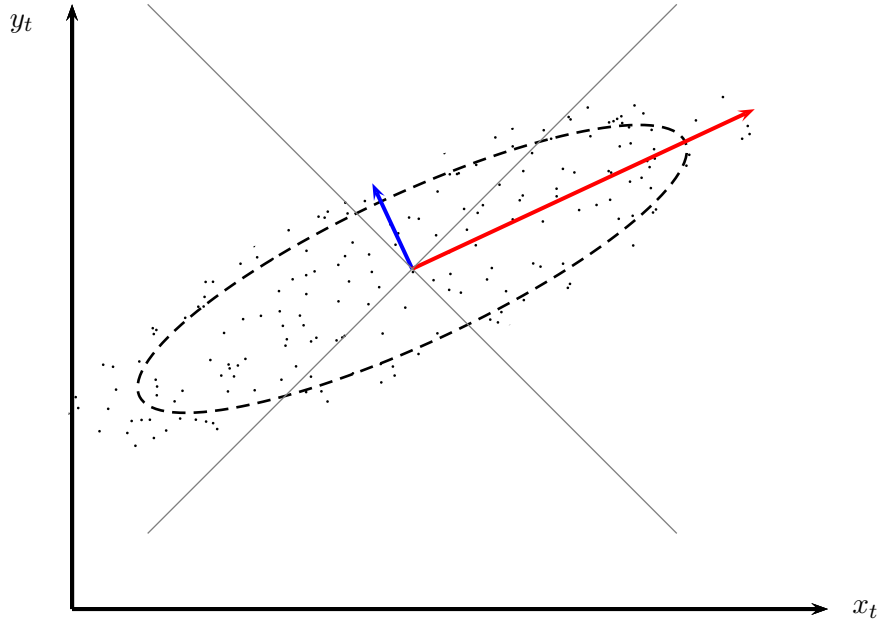[11]This is the main point of Forbes and Rigobon

Figure 2: Principal Components

As in the case of the correlation, the variance of the first principal component (and the eigen-vector that it represents) are 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 we 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 co-movement using correlations and principal components shifts in the sample for two reasons – because the structural parameters shift or because the data suffers from heteroskedasticity. Without additional information it is impossible to distinguish.

3.3.3 Linear Regression Models

Linear models assume that the relationship between the variables in the two countries can be described by a simple linear model

$$y_t = a x_t + \nu_t \tag{10}$$

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.[12]

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[12]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

The first question is if OLS captures the actual estimate (is there a bias or not). As before, I will 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:

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

Several remarks are worth highlighting. First, the estimate is biased. It is a biased estimate of α 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 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 importantly the estimate does not depend on the relative variance

A Vector Auto Regression (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 than 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 OLS – are consistent estimates. However, this is equivalent to say 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, even though they take into account the conditional heteroskedasticity in the data, they are not designed to deal with the problem of endogeneity or the problem of more factors than observed variables (the omitted variable problem). In general, the estimation is performed on the reduced form. Hence, it inherits the identification problems of endogeneity and omitted variable biases.

A final remark on the linear regressions and the parameter instability. Lately the literature has spent huge efforts in the estimation of parameter varying models. My first reaction to those papers is to ask if there is a possibility that misspecification and heteroskedasticity are 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.

3.3.4   Extreme Observation Models: Event Studies

Even 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 $\hat{a} = \frac{1}{\beta}$.

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spillover effect in the data.

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

Therefore, if the event is known, meaning if the researcher knowns in which country and market the shock is being originated, then the estimation can be performed as an event study.

The rationale and intuition of this identification strategy was introduced by Wright (1928) and it is called *Near Identification*. It is called near identification because the assumption that θ takes the extreme values of zero or infinity is a strong assumption. I will 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 (2008) for a thorough description of the methodology to improve event studies.

3.3.5 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 example using conditional probabilities to measure "contagion" can be found in Eichengreen, Rose, and Wyplosz (1996). Copulas are also very common in the literature. Some of the early attempts try to characterize the joint distribution at the tales.[13]

The general intuition is twofold: First, in order 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, in order to determine shift-contagion (parameter instability) the conditional probabilities are compared between small and large shocks, or between positive versus negative shocks. The problem is that, as before, is that these measurements do not capture structural parameters.

For example, in the endogenous model and the omitted variables models we have been studying, the conditional probability at the tails can be driven either by ϵ_t or η_t or 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.

3.4 Newer Methods

I personally believe there is not a single technique that can solve the empirical challenges the literature on contagion is trying to tackle. What is worse is that the theories are far too restrictive and therefore a structural estimation approach is bound to be insufficient. Therefore, I would like to summarize three techniques that are partially addressing some of the problems. In particular, I will separate the estimation problem from the parameter stability question. I think that under

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[13]The literature on copula's is very large. One of my preferred papers is Rodriguez (2007).

the assumption of parameter stability, then the estimation of the spillovers can be conducted. The methodology here is called identification through heteroskedasticity that 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 methodology is based on heteroskedasticity while the other is based on quintile regressions. However, if the question of interest is one in which both the estimates are important, and also their stability is to be tested, then I do not know of any methodology that can provide a satisfactory answer.

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

3.4.1   Spillover Estimation: Identification Through Heteroskedasticity

The identification problem has been at the root of some of the most important innovations in econometrics 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 being 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.[14]. In the appendix of that book he discusses three possible techniques to solve the problem of the demand-supply estimation: The first one is what we know as instrumental variables nowadays in economics. The second one is what is known as near identification – which is the precursor of event studies, regression discontinuity, and randomized controlled trials. And the third methodology provides the intuition of identification through heteroskedasticity. So, technically, all the procedures we use in econometrics were invented in 1928 in a book about animal oils.[15] Interestingly, however, the first one took over the profession with a vengeance, the second one has just started to take over development economics, corporate finance, and other areas of economics, while the third one has just barely being used – and the few applications are in macroeconomics and international economics.[16]

First, let me explain the problem of identification. Equations 3 and 4 describe the behavior of the data entirely with 4 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 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). RCT's assume that all the variation is due

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[14]See Wright (1928)

[15]I do not know you, but this has always made me feel as if the techniques we use are way less sexy and cool than I previously though

[16]For the theoretical derivations see Rigobon (2003) for the genera case and Sentana and Fiorentini (2001) for an excellent derivation in the context of ARCH models. For applications on Monetary Policy and Macroeconomics see Rigobon and Sack (2003), Rigobon and Sack (2004), Rigobon and Sack (2008). For a recent application see Nakamura and Steinsson (2015), among many others. Applications on the measurement of spillovers see Ehrmann, Fratzscher, and Rigobon (2005) and the many papers Marcel Fratzscher has written.

to the treatment – again, this is implicitly assuming 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 equation. Assume that the parameters are stable and that the data has heteroskedasticity. For simplicity assume that there are two heteroskedastic regimes. In this case, it is possible to estimate one covariance matrix in each regime.

$$
\Omega_1 = \begin{bmatrix} var(x_{t,1}) & covar(x_{t,1}, y_{t,1}) \\ & var(y_{t,1}) \end{bmatrix} = \frac{1}{(1-\alpha\beta)^2} \begin{bmatrix} \sigma_{\epsilon,1}^2 + \beta^2 \sigma_{\eta,1}^2 & \alpha\sigma_{\epsilon,1}^2 + \beta\sigma_{\eta,1}^2 \\ & \alpha^2\sigma_{\epsilon,1}^2 + \sigma_{\eta,1}^2 \end{bmatrix}
$$

$$
\Omega_2 = \begin{bmatrix} var(x_{t,2}) & covar(x_{t,2}, y_{t,2}) \\ & var(y_{t,2}) \end{bmatrix} = \frac{1}{(1-\alpha\beta)^2} \begin{bmatrix} \sigma_{\epsilon,2}^2 + \beta^2 \sigma_{\eta,2}^2 & \alpha\sigma_{\epsilon,2}^2 + \beta\sigma_{\eta,2}^2 \\ & \alpha^2\sigma_{\epsilon,2}^2 + \sigma_{\eta,2}^2 \end{bmatrix}
$$

There are six unknowns in the system. The two parameters (α and β), and four variances ($\sigma_{\epsilon,1}^2, \sigma_{\epsilon,2}^2, \sigma_{\eta,1}^2$, and $\sigma_{\eta,2}^2$). As can be seen, there are six equations in six unknowns. This means that the system of equations is just identified. Notice that even though in each regime the system is under-identified (less equations than unknowns) the system as a whole is identified. The key assumptions are two: that the structural shocks are indeed structural (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.
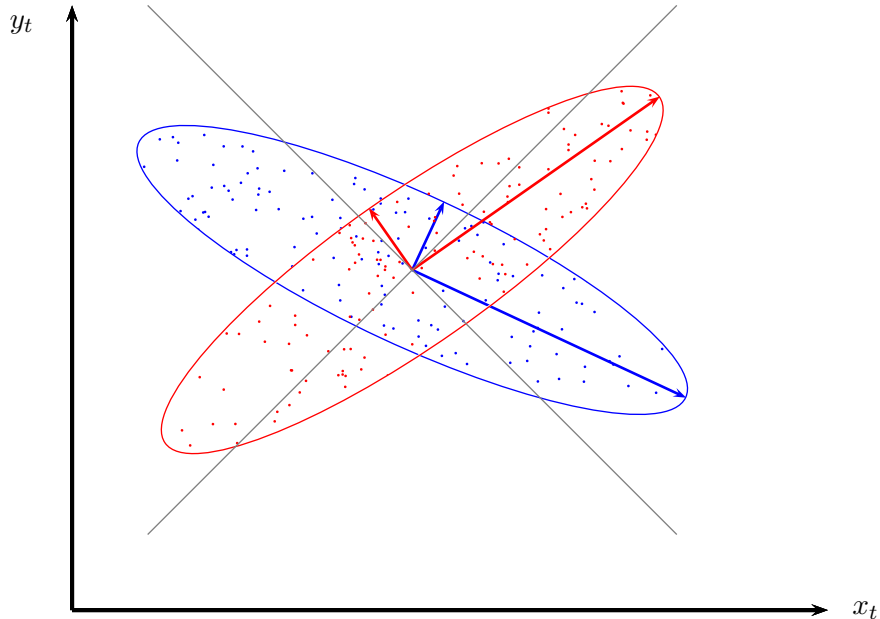

Figure 3: Identification Through Heteroskedasticity

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. In

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Figure 3, I show two cases: One when the shocks to the demand dominate (red), and one when the shocks to the supply dominate (blue). In particular, when the shocks to the demand dominate, then 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, then 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.

It is instructive to re-state the underlying assumptions: structural shocks are uncorrelated (quite uncontroversial) and parameters need to be stable across the regimes (so, this is a good technique to measure spillovers).

3.4.2   Parameter Instability: DCC

There is a simple extension of the identification through heteroskedasticity methodology that allows one to test for parameter instability. When I designed it I called it the DCC which stands for the *Determinant* of the *Change* in the *Covariance* Matrix. This is mainly an overidentification test.

The key assumption is that some of the shocks are heteroskedastic, but others are homoskedastic. In particular, as before assume there are two regimes of heteroskedasticity but assume that the researcher knows that one of the shocks is homoskedastic. In the context of contagion, this is equivalent to assuming that it is known that the crisis originates 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 assume that in 2010 the shocks to Greece, Ireland, Portugal, and Spain are more volatile, but that the shocks to France, Germany, and the Netherlands are equally volatile. This is a strong assumption but one that allows to test for parameter instability in this context.

The methodology 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 we obtain:

$$\Omega_1 - \Omega_2 \;=\; \frac{1}{(1-\alpha\beta)^2}\begin{bmatrix} \sigma_{\epsilon,1}^2 + \beta^2\sigma_\eta^2 & \alpha\sigma_{\epsilon,1}^2 + \beta\sigma_\eta^2 \\ & \alpha^2\sigma_{\epsilon,1}^2 + \sigma_\eta^2 \end{bmatrix} - \frac{1}{(1-\alpha\beta)^2}\begin{bmatrix} \sigma_{\epsilon,2}^2 + \beta^2\sigma_\eta^2 & \alpha\sigma_{\epsilon,2}^2 + \beta\sigma_\eta^2 \\ & \alpha^2\sigma_{\epsilon,2}^2 + \sigma_\eta^2 \end{bmatrix}$$

$$= \frac{1}{(1-\alpha\beta)^2}(\sigma_{\epsilon,1}^2 - \sigma_{\epsilon,2}^2)\begin{bmatrix} 1 & \alpha \\ & \alpha^2 \end{bmatrix}$$

$$\|\Omega_1 - \Omega_2\| \;=\; \frac{1}{(1-\alpha\beta)^2}(\sigma_{\epsilon,1}^2 - \sigma_{\epsilon,2}^2)\left\|\begin{bmatrix} 1 & \alpha \\ & \alpha^2 \end{bmatrix}\right\| = 0$$

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)

$$\Omega_1 - \Omega_2 \;=\; \frac{1}{(1-\alpha_1\beta)^2}\begin{bmatrix} \sigma_{\epsilon,1}^2 + \beta^2\sigma_\eta^2 & \alpha_1\sigma_{\epsilon,1}^2 + \beta\sigma_\eta^2 \\ & \alpha_1^2\sigma_{\epsilon,1}^2 + \sigma_\eta^2 \end{bmatrix} - \frac{1}{(1-\alpha_2\beta)^2}\begin{bmatrix} \sigma_{\epsilon,2}^2 + \beta^2\sigma_\eta^2 & \alpha_2\sigma_{\epsilon,2}^2 + \beta\sigma_\eta^2 \\ & \alpha_2^2\sigma_{\epsilon,2}^2 + \sigma_\eta^2 \end{bmatrix}$$

$$\|\Omega_1 - \Omega_2\| \;\neq\; 0$$

It is easy to show that 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, then 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. See Rigobon (2000) and Dungey and Fry (2004).

3.4.3 Parameter Instability: Quantile Regressions

Finally, a very simple test based on quintile regressions tests for parameter instability. Contagion and parameter instability creates a non-linearity in the OLS estimates. In other words, conditional on larger volatility and different propagation mechanisms the biases on the simple OLS estimates are different across times. It is possible to test for this non-linearity 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 is a test that allows for an unrestricted form of non-linearity (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 between each other, while if there is parameter instability the coefficients shift across the quintiles. See Caporin, Pelizzon, Ravazzolo, and Rigobon (2012) for an application to the European Crises.

4   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 observed variables are endogenous and/or omitted variables are present. Second, the financial data suffers from heteroskedasticity. The first one is a problem of misspecification, and the second one should be relatively easy to deal with. However, the combination of these two problems implies that the degree of misspecification changes throughout the sample. Therefore, the biases are shifting through time. In consequence, correlations, principal components, OLS regressions, event studies, VAR's, Arch and Garch models, Probit and Logit, 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, answering 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?" cannot have a satisfactory answer with standard methodologies.

The problem is even more complicated because there are no natural experiments nor instruments that could solve the identification problem. Therefore, the problem is left to the typical macroeconomic identification strategies that depend on VAR's Choleski decompositions, or 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 20th 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 to 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 the problem in two different perspectives. As said, unfortunately, to my knowledge, there is not a 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 from 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 exist among banks within a country — i.e. financial linkages among the different actors in the economy.

Second, if the question is about parameter stability there are two possible avenues. The first one 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 US 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 or not 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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