New Test for Contagion*

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Abstract

The paper develops an easy-to-apply test for contagion. The new test takes into account all the main challenges contagion tests face; that of endogeneity, distinguishing interdependencies from contagion, and heteroskedasticity. To address these challenges the testing is conducted in the structural vector autoregression (SVAR) framework where we assume the reduced form errors follow a mixed-normal distribution. The framework enables us to apply a recently developed SVAR model identification method with no need to restrict any of the instantaneous linkages between the variables. We apply our test to the eurozone government bond data. The sample period is the years 2009–2010 and, so, coincides with the beginning of the sovereign debt crisis in the eurozone. Evidence of contagion is found. Especially, it appears that the contagion effects were quite complex without any single source country of contagion.

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1 Introduction

Contagion is a surprisingly hard topic. The main idea is simple; markets might transmit one country's economic woes to other countries. Thus, local crises potentially become regional or even global. However, the empirical research on the matter is full of challenges.[1] The first one is that of endogeneity; the second concerns the often apparent heteroskedasticity in financial time series; and, finally, any empirical contagion model should be careful how to define contagion.

The endogeneity issue arises because the financial markets often react to news almost instantaneously. So, one usually needs to work with a system of simultaneous equations. Disentangling a cause and consequences is then hard unless the researcher is prepared to do–sometimes even harsh–identifying assumptions. Also, financial time series often exhibit strains of heteroskedasticity which might even be the very consequence of contagion. Hence, the selected model should address such possible changes in the market volatilities.

When it comes to defining contagion, one needs to be careful. As pointed out by Forbes and Rigobon (2002), simply detecting high correlations between countries' market returns during a crisis does not necessarily constitute contagion. Financial markets are usually highly interdependent also during normal times; then, high correlation during a crisis might just be a continuation of the pre-crisis linkages. Only, if there is evidence of crisis-contingent structural changes in the shock transmission mechanisms across the countries, one should talk of contagion.[2] Otherwise, crisis-time correlations are only evidence of high interdependencies between the countries. Separating contagion from interdependence and, so, testing for crisis-contingent structural changes in the international shock propagation mechanisms is now a norm in the empirical contagion literature (see, for example, Dungey, Fry, Gonzalez-Hermosillo, and Martin (2005); Corsetti, Pericoli, and Sbracia (2005); Caporale, Cipollini, and Spagnolo (2005); Pesaran and Pick (2007); Billio and Caporin (2010); Metiu (2012)).[3]

[1]There are several good surveys on the contagion literature, both those emphasizing theoretical models and those focusing on the empirical work. For some quite thorough literature reviews, see for example Pericoli and Sbracia (2003); Forbes and Rigobon (2001); Dungey, Fry, Gonzalez-Hermosillo, and Martin (2005); or Dornbusch, Park, and Claessens (2000).

[2]Although some researchers do not recommend using the term contagion given its negative connotations, we will stick to this established terminology. Notice, however, that our definition of contagion, that of there being crisis-contingent structural changes in shock propagation mechanisms, includes not only all possible negative effects that a negative shock in one country might have on others, but also the so-called "flight-to-quality" effects where a crisis somewhere makes investors to sell assets in countries regarded as weak and to buy them in countries seen to be safer. For a discussion and criticism on the validity of the term contagion, see the introduction in Favero and Giavazzi (2002).

[3]For motivation and some examples on the distinction between contagion and interdepen-

In his modeling then, the researcher should take into account all the three challenges while still keep his model identifiable and easy to follow. In this spirit, this paper develops a general but still simple-to-apply test for contagion which addresses all the listed challenges. Our test is build on the structural vector autoregressive (SVAR) model of Favero and Giavazzi (2002). The Favero and Giavazzi model is not identifiable as such but we augment it with an additional assumption concerning the distribution of the reduced from errors. We assume these errors follow a mixed-normal distribution. This additional assumption allows us employ the recently proposed SVAR identification method by Lanne and Lütkepohl (2010). We then implement the contagion test Favero and Giavazzi propose by testing the stability of the parameters that control the international transmission of the country specific shocks. The model can be estimated with the method of maximum likelihood and the contagion testing is performed by using the standard likelihood ratio test.

In our empirical application we use our test to see whether there is evidence of contagion in the government bond markets of a selected group of the eurozone member countries in the years 2009–2010. The data we use is the ten years government bond spreads over Germany of Greece, Portugal, Ireland, Spain, and Italy. Evidence of contagion is found and, furthermore, the contagion effects seem to be quite complex. According to the analysis there was not one source country of contagion but several. This point, that during a crisis there might be more complex reciprocal contagion effects than only from one crisis country to others, is often ignored in the contagion literature. By using the estimated mixture probability of the mixed-normal distribution as a weight, we also calculate weighted correlation coefficients of the country spreads both during the normal times and the crisis. Because these coefficients automatically take into account the possible heteroskedasticities in the spreads, they are better suited for correlation analysis than the ones used in many of the earlier contagion studies.[4]

Our test, perhaps, most closely resembles two previously presented contagion tests: the determinant of the change in the covariance matrix test by Rigobon

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dence, see Forbes and Rigobon (2001).

[4]Perhaps the main insight of the Forbes and Rigobon (2002) paper was to underline that, because during crises volatility in financial markets usually rises, (conditional) correlation coefficients calculated during the crisis are upwards biased. So, if the analysis is based on comparing pre-crisis correlations in returns against crisis times correlations, and if higher correlation during the crisis is considered as evidence of contagion, as it used to be in the earlier research (see, for example, Calvo and Reinhart (1996); King and Wadhwani (1990); Lee and Kim (1994)), the results might be biased. The higher-than-before correlation during a crisis could be only results of higher volatility, not any new structural shock transmission channels. So, the conditional correlation coefficients need to be adjusted for heteroskedasticity. But the adjustment Forbes and Rigobon suggest assumes no endogeneity in the model. This is of course a very strong assumption.

(2003b), and the multivariate contagion test of Dungey, Fry, Gonzalez-Hermosillo, and Martin (2005, 11–12). The former calculates the covariance matrices (of the market returns) in normal and crisis times, calculates the changes in the covariances, and takes the determinant of this changes-in-covariances matrix. If the determinant is zero, the paper argues, the shock propagation mechanisms have stayed stable during the crisis. Hence, there is not contagion. Rigobon's test, however, basically requires that we know which are the crisis countries and that part of the sample countries are non-crisis countries. Our test do not require this. We simply need to be able to identify the crisis periods from the normal times. This can usually be done more or less accurately.[5] The latter test, that of Dungey and the others, is, in contrast, a latent factor model mostly applicable for asset return time series with zero mean. Such series are usually obtained by taking the first differences of the variables. Once one is interested in investigating the financial variables in their level values, a (S)VAR framework is probably more suitable.

Because the main objective of the contagion tests is to measure changes in the instantaneous dependencies between financial variables, the more traditional SVAR identification methods that rely of restricting some these dependencies are, of course, not that desirable. Instead of restricting any of the potential contagion effects *a priori*, we would like to let the data determine, as freely as possible, if they exists or not.[6] Similar to the the Lanne-Lütkepohl identification method some other authors have used particularities in the data as a source of the needed extra information for the SVAR model identification. For example, Rigobon (2003a) introduces a heteroskedasticity based identification method that has been successfully applied in the contagion–and the volatility spillover–literature (see, for example, Caporale, Cipollini, and Spagnolo (2005); Caporale, Cipollini, and Demetriades (2005); Rigobon (2002); Rigobon and Sack (2003)). However, unlike Lanne and Lütkepohl who assume the non-normalities are exhibited in the reduced form errors' joint distribution, Rigobon assumes heteroskedasticity in the structural shocks. The original Favero and Giavazzi model, in its turn, assumes the structural shocks are homoskedastic and, because of contagion, the reduced form errors might be heteroskedastic. This favors using the Lanne-Lütkepohl method in our context.

The rest of the paper is organized as follows. Section 2 provides a short review of the theoretical contagion channels. Section 3, presents our empirical model and

[5]For examples of clear-cut crisis periods see, in addition to the Rigobon paper, Forbes and Rigobon (2002); Dungey, Fry, Gonzalez-Hermosillo, and Martin (2005); Corsetti, Pericoli, and Sbracia (2005).

[6]Of course, sometimes there are well justified institutional reasons for restrictions on (almost) instantaneous effects. For example, Billio and Caporin (2010) consider markets in different timezones.

the test for contagion. The Favero and Giavazzi model is also reviewed. Section 4 provides the empirical application of our test. Finally, section 5 gives some concluding remarks.

2 Channels of Contagion

Why would crises be transmitted across countries in the first place? In theory, there are several possible reasons for contagion. It could be a result of information asymmetries among investors (King and Wadhwani, 1990; Kodres and Pritsker, 2002); of more global financial markets in a world where investors face information costs together with (legal or institutional) restrictions on short selling (Calvo and Mendoza, 2000); or of self-fulfilling investor expectations as a crisis hitting one country forces investors to update their beliefs also on its peer countries, making economies to jump between multiple equilibria (Masson, 1999).

However, perhaps the most compelling contagion theories build on the idea that it is the international banks who via their balance sheets propagate shocks across the countries. Allen and Gale (2000) consider a case where an unexpected regional liquidity demand shock causes a chain of bankruns across regions. Plainly put, they argue that the less complete is the international network of banks and their reciprocal claims, the less there is ”cushion” to protect a single bank against a bankrun. Also, Mendoza and Quadrini (2010) show, when the banks follow the mark-to-market accounting rule and face minimum capital requirements, a negative shock to to the value of equity of a bank (or a group of banks in one country) might create a chain reaction leading into a global credit crunch as the banks need to limit their lending.

Kiyotaki and Moore (2002) discuss two other ways companies' balance sheets might propagate shocks. First, based on their well known model on credit cycles (Kiyotaki and Moore, 1997), they argue that when firms (partly) finance their operations with bank loans that are backed by a collateral, and the value of collateral is proportional to the market price of the firms' assets, a negative production shock to even a small group of firms might affect a larger group of firms all of whom rely on bank lending. The reason is that as the negative production shock decreases the current value of production of the initially affected firms, the market value of their assets, also, decreases. However, then, the decrease in the market value of the collaterals is not limited only to the first-hit companies but affects also all the other firms who have provided similar types of collaterals for their bank loans. Hence, this second group of firms need also to cut down some of their investments and, so, their future production. The initial shock multiplies across the economy. The second mechanism the authors consider corresponds to a situation where a group of firms are linked to each other via a chain of mutual claims and liabilities.

Then, whenever one of the firms defaults its debt and, if this debt is an asset of another firm, the default might lead into a chain of defaults.

All of these theories explain why a shock might get transmitted across firms, industries, or countries. However, it is hard to estimate them. So, the empirical research has mainly taken the alternative approach that was explained in the introduction, that of contagion vs. interdependence. There is evidence of contagion only if there is evidence of structural changes in the international shock transmission mechanisms during a crisis. However, the theories reviewed in this section, in their turn, explain such structural changes.

3 The Model

Denote country i's government bond yield in period t as y_{it} and that of the German government bond as y_t^*. Country i's bond spread over Germany in period t then becomes $s_{it} = y_{it} - y_t^*$. Consider the case of $n \geq 2$ countries and assume the following SVAR model for the spreads:

$$\mathbf{s}_t = \mathbf{A}\mathbf{s}_{t-1} + \mathbf{B}\left(\mathbf{I}_n + \mathbf{C}\mathbf{D}_t\right)\boldsymbol{\varepsilon}_t, \tag{1}$$

where $\mathbf{s}_t$ is the $(n \times 1)$ vector of the country spreads; $\mathbf{A}$, $\mathbf{B}$ and $\mathbf{C}$ are $(n \times n)$ coefficient matrices; and $\mathbf{I}_n$ is the $(n \times n)$ identity matrix. The $(n \times 1)$ vector $\boldsymbol{\varepsilon}_t = [\varepsilon_{1t}, \ldots, \varepsilon_{nt}]'$ corresponds to the country specific structural shocks which are assumed to be uncorrelated of each other. The matrix $\mathbf{D}_t = \mathrm{diag}(d_{1t}, \ldots, d_{nt})$ is diagonal and includes country specific crisis dummies. All these dummies equal to zero during the normal times. But if there is a crisis in period t that originates in country i, we will have $d_{it} = 1$ and $d_{jt} = 0$ for all $j \neq i$.

The model in equation (1) was proposed by Favero and Giavazzi (2002) to model contagion in government bonds. During the normal times, because $\mathbf{D}_t = \mathbf{0}$, the international transmission of structural shocks is determined solely by the matrix $\mathbf{B}$. So, this matrix captures the interdependencies between the countries. However, when there is a crisis in country i and, so, we have $d_{it} = 1$, the transmission channel of the country i's structural shock ε_{it} to country j is augmented by a new factor, the coefficient c_{ji}. If c_{ji} was equal to zero, there would not be crisis-contingent structural changes in the transmission mechanism of ε_{it} to country j. So, in such a case, there would not be contagion from country i to country j.

Favero and Giavazzi conclude that, as a test for contagion, one would be willing to test jointly whether all the $n(n-1)$ off-diagonal elements c_{ji} are equal to zero (Favero and Giavazzi (2002, 234)). If they are, there will not be contagion during a crisis; if not, there will be *some* contagion during a crisis. Unfortunately, the model in equation (1) is unidentified and the Favero and Giavazzi contagion test is not implementable. The next subsection proposes a concise way to perform the test. Briefly, the idea is to test if all the transmission channels stay stable during a period of a crisis when compared to the normal times.

3.1 Test for contagion

For our test, we need to make an additional assumption concerning the matrix $\mathbf{C}$. Assume that all its main diagonal elements c_{ii} equal to zero. These coefficients depict the possible, crisis-contingent structural changes in the within country effects of the structural shocks. We will discuss little later how plausible this assumption is. At this point it suffices to say that the assumption guarantees that we can interpret any structural change in the transmission mechanisms during a crisis being only from changes in the cross-country effects of the structural shocks. This said, once we redefine $\tilde{\mathbf{B}} = \mathbf{B}(\mathbf{I}_n + \mathbf{C}\mathbf{D}_t)$, testing contagion boils down to testing whether the matrix $\tilde{\mathbf{B}}$ remains stable during a crisis or not.

During the normal times, when $\mathbf{D}_t = \mathbf{0}$, we simply have that the B-matrix equals to the interdependencies between the countries: $\tilde{\mathbf{B}} = \mathbf{B}$. Let us denote this matrix as $\tilde{\mathbf{B}}^{\mathrm{N}}$. During a crisis instead, we have $\tilde{\mathbf{B}} = \mathbf{B}(\mathbf{I}_n + \mathbf{C}\mathbf{D}_t)$. Let us denote this as $\tilde{\mathbf{B}}^{\mathrm{C}}$. We then have the following two hypothesis:

$$\mathrm{H}_0 : \tilde{\mathbf{B}}^{\mathrm{N}} = \tilde{\mathbf{B}}^{\mathrm{C}} \text{ and } \mathrm{H}_1 : \tilde{\mathbf{B}}^{\mathrm{N}} \neq \tilde{\mathbf{B}}^{\mathrm{C}}, \tag{2}$$

where the null-hypothesis refers to the *no contagion, only-interdependence* result; and the alternative hypothesis to the *contagion* result. Like the following subsection shows, testing of the null-hypothesis can be implemented with the standard likelihood ratio (LR) test.

3.2 Implementation of the test

Assume the reduced form model corresponding to our SVAR model in equation (1) is the following:

$$\mathbf{s}_t = \mathbf{A}\mathbf{s}_{t-1} + \mathbf{u}_t, \tag{3}$$

where the $(n \times 1)$ reduced form error vector $\mathbf{u}_t = \mathbf{B}(\mathbf{I}_n + \mathbf{C}\mathbf{D}_t)\boldsymbol{\varepsilon}_t = \tilde{\mathbf{B}}\boldsymbol{\varepsilon}_t$. This corresponds to the B-model framework of the SVAR models (for more details on the B-model, see, for example, Lütkepohl (2007, 362–64)). Then, the question is how to identify the structural shocks and, so, estimate the matrix $\mathbf{B}$. We will follow the idea of Lanne and Lütkepohl (2010) and exploit non-normalities in the data to identify the model. We assume especially that $\mathbf{u}_t$ follows a mixed-normal distribution, so that

$$\mathbf{u}_t = \begin{cases} \mathbf{e}_{1t} \sim N(\mathbf{0}, \boldsymbol{\Sigma}_\mathbf{1}) & \text{with probability} \quad \gamma, \\ \mathbf{e}_{2t} \sim N(\mathbf{0}, \boldsymbol{\Sigma}_\mathbf{2}) & \text{with probability} \quad 1-\gamma, \end{cases} \tag{4}$$

where $N(\mathbf{0}, \mathbf{\Sigma_i})$ denotes a multivariate normal distribution with zero mean and $(n \times n)$ covariance matrix $\mathbf{\Sigma_i}$. Hence, $\mathbf{e}_{1t}$ and $\mathbf{e}_{2t}$ are two serially independent Gaussian error vectors. For the mixture probability $\gamma \in (0, 1)$ to be identifiable, one needs to assume that the covariance matrices $\mathbf{\Sigma}_1$ and $\mathbf{\Sigma}_2$ are (at least partly) distinct.

3.2.1 Identification of the structural model

Lanne and Lütkepohl show that there exist a nonsingular $(n \times n)$ matrix $\mathbf{W}$ and a $(n \times n)$ diagonal matrix $\mathbf{\Psi} = \text{diag}(\psi_1, \ldots, \psi_n)$ with all diagonal elements being strictly positive, such that the covariance matrices in the mixed-normal distribution in equation (4) can be decomposed in the following way: $\mathbf{\Sigma}_1 = \mathbf{W}\mathbf{W}'$ and $\mathbf{\Sigma}_2 = \mathbf{W}\mathbf{\Psi}\mathbf{W}'$. This result follows from the covariance matrices being symmetric and positive definite (for details, see the appendix in the Lanne and Lütkepohl paper). Provided that all the elements ψ_i are distinct from each other, the matrix $\mathbf{W}$ is unique (apart from changing the signs of the elements in every column).

The covariance of the reduced form errors then becomes

$$\mathbf{\Sigma_u} = \gamma \mathbf{\Sigma_1} + (1-\gamma)\mathbf{\Sigma_2} = \mathbf{W}\left(\gamma \mathbf{I}_n + (1-\gamma)\mathbf{\Psi}\right)\mathbf{W}'. \tag{5}$$

On the other hand, from $\mathbf{u}_t = \tilde{\mathbf{B}}\boldsymbol{\varepsilon}_t$ it follows that $\mathbf{\Sigma_u} = \tilde{\mathbf{B}}\mathbf{\Sigma}_{\boldsymbol{\varepsilon}}\tilde{\mathbf{B}}'$. When we normalize the SVAR model by assume all the structural errors have a unit variance[7], so we assume $\boldsymbol{\varepsilon}_t \sim (\mathbf{0}, \mathbf{I}_n)$, it follows that $\mathbf{\Sigma_u} = \tilde{\mathbf{B}}\tilde{\mathbf{B}}'$. Comparing this with the covariance matrix in equation (5) allows us to choose

$$\tilde{\mathbf{B}} = \mathbf{W}\left(\gamma \mathbf{I}_n + (1-\gamma)\mathbf{\Psi}\right)^{1/2}.$$

Once we also assume[8] that the elements $\{\psi_1, \ldots, \psi_n\}$ are in some pre-defined order, for example in the descending order, on the main diagonal of the matrix $\mathbf{\Psi}$, the matrix $\tilde{\mathbf{B}}$ is unique (Lanne, Lütkepohl, and Maciejowska, 2010).

[7] The unit variance assumption is a common way to normalize a SVAR model. Alternatively, one could allow the structural shock variances σ_{ε_i} being any positive real number and, instead, assume that all the main diagonal elements of the matrix $\tilde{\mathbf{B}}$ equal to one. Kilian (2011) discusses several possible ways to normalize a SVAR model.

[8] This assumption does not affect the generality of our test. This can be seen in the following way: first, as Kohonen (2012) shows, whenever we choose $\tilde{\mathbf{B}} = \mathbf{W}(\gamma\mathbf{I}_n + (1-\gamma)\mathbf{\Psi})^{1/2}$, where the elements on the diagonal of the matrix $\mathbf{\Psi}$ are in some pre-specified order, we could as well choose $\hat{\mathbf{B}} = (\mathbf{W}\mathbf{P}')(\gamma\mathbf{I}_n + (1-\gamma)\mathbf{P}\mathbf{\Psi}\mathbf{P}')^{1/2}$ as our B-matrix. Here, $\mathbf{P}$ is an arbitrary $(n \times n)$ permutation matrix. Second, using any $\hat{\mathbf{B}}$ instead of $\tilde{\mathbf{B}}$ simply reshuffles the order of the structural shocks in the vector $\boldsymbol{\varepsilon}_t$. To see this, notice that the part $(\gamma\mathbf{I}_n + (1-\gamma)\mathbf{P}\mathbf{\Psi}\mathbf{P}')^{1/2} = (\mathbf{P}(\gamma\mathbf{I}_n + (1-\gamma)\mathbf{\Psi})\mathbf{P}')^{1/2}$ in $\hat{\mathbf{B}}$ is diagonal, so it equals to $\mathbf{P}(\gamma\mathbf{I}_n + (1-\gamma)\mathbf{\Psi})^{1/2}\mathbf{P}'$. It follows that $\hat{\mathbf{B}} = \tilde{\mathbf{B}}\mathbf{P}'$ (remember that $\mathbf{P}$ is orthogonal, and so $\mathbf{P}\mathbf{P}' = \mathbf{I}_n$). Hence, the matrix $\hat{\mathbf{B}}$ is simply a column-wise permutation of $\tilde{\mathbf{B}}$. On the other hand, denote as $\hat{\boldsymbol{\varepsilon}}_t$ the structural shocks that correspond to the matrix $\hat{\mathbf{B}}$, hence we have $\mathbf{u}_t = \tilde{\mathbf{B}}\boldsymbol{\varepsilon}_t = \hat{\mathbf{B}}\hat{\boldsymbol{\varepsilon}}_t$, and then $\hat{\boldsymbol{\varepsilon}}_t = (\hat{\mathbf{B}})^{-1}\tilde{\mathbf{B}}\boldsymbol{\varepsilon}_t = (\tilde{\mathbf{B}}\mathbf{P}')^{-1}\tilde{\mathbf{B}}\boldsymbol{\varepsilon}_t = \mathbf{P}'^{-1}\tilde{\mathbf{B}}^{-1}\tilde{\mathbf{B}}\boldsymbol{\varepsilon}_t = \mathbf{P}\boldsymbol{\varepsilon}_t$, where $\mathbf{P}'^{-1} = \mathbf{P}$ is a result from $\mathbf{P}$ being orthogonal. So, $\hat{\boldsymbol{\varepsilon}}_t$ is simply a row-wise permutation on

3.2.2 Estimation of the unrestricted model

We will test the null-hypothesis of no-contagion in equation (2) against the alternative hypothesis of contagion by estimating two separate models; an unrestricted model that corresponds to the alternative hypothesis, and a restricted model that corresponds to the null-hypothesis. The models are estimated with the method of maximum likelihood (ML), so we can us the standard LR test to test the null-hypothesis against the alternative hypothesis. Assuming the (S)VAR models are stationary, we can refer to the standard ML testing theory.

The unrestricted SVAR model corresponding to the alternative hypothesis of $\tilde{\mathbf{B}}^{\mathrm{N}} \neq \tilde{\mathbf{B}}^{\mathrm{C}}$ is the following:

$$\mathbf{s}_t = \mathbf{A}\mathbf{s}_{t-1} + d_N \tilde{\mathbf{B}}^N \boldsymbol{\varepsilon}_t + d_C \tilde{\mathbf{B}}^C \boldsymbol{\varepsilon}_t,$$

where d_N and d_C are dummies indicating normal times and crisis times, respectively. The corresponding reduced form model is

$$\mathbf{s}_t = \mathbf{A}\mathbf{s}_{t-1} + d_N \mathbf{u}_t^N + d_C \mathbf{u}_t^C,$$

where the normal and crisis times error vectors $\mathbf{u}_t^N$ and $\mathbf{u}_t^C$ follow mixed-normal distributions

$$\mathbf{u}_t^N = \begin{cases} \mathbf{e}_{1t}^N \sim N\left(\mathbf{0}, \mathbf{W}_1 \mathbf{W'}_1\right) & \text{with probability} \quad \gamma_1, \\ \mathbf{e}_{2t}^N \sim N\left(\mathbf{0}, \mathbf{W}_1 \boldsymbol{\Psi}_1 \mathbf{W}'_1\right) & \text{with probability} \quad 1-\gamma_1, \end{cases}$$

and

$$\mathbf{u}_t^C = \begin{cases} \mathbf{e}_{1t}^C \sim N\left(\mathbf{0}, \mathbf{W}_2 \mathbf{W'}_2\right) & \text{with probability} \quad \gamma_2, \\ \mathbf{e}_{2t}^C \sim N\left(\mathbf{0}, \mathbf{W}_2 \boldsymbol{\Psi}_2 \mathbf{W}'_2\right) & \text{with probability} \quad 1-\gamma_2. \end{cases}$$

The normal times error term $\mathbf{u}_t^N$ has density

$$\begin{aligned} f(\mathbf{u}_t^N) =& \gamma_1 (2\pi)^{-n/2} \det(\mathbf{W}_1 \mathbf{W'}_1)^{-1/2} \exp\left\{-\frac{1}{2} \mathbf{u}^{N'}_t (\mathbf{W}_1 \mathbf{W'}_1)^{-1} \mathbf{u}_t^N\right\} + \\ &+ (1-\gamma_1)(2\pi)^{-n/2} \det(\mathbf{W}_1 \boldsymbol{\Psi}_1 \mathbf{W'}_1)^{-1/2} \exp\left\{-\frac{1}{2} \mathbf{u}^{N'}_t (\mathbf{W}_1 \boldsymbol{\Psi}_1 \mathbf{W'}_1)^{-1} \mathbf{u}_t^N\right\}. \end{aligned} \tag{6}$$

the vector $\boldsymbol{\varepsilon}_t$. Only one of these permutations will coincide with the situation where the country one specific shock ε_{1t} is placed first, the country two specific shock ε_{2t} second, etc. But as long as we are simply interested in testing the stability of the effects of the structural shocks (as we are in this paper), we do not need to identify this "correct" permutation. It is enough to assume that our structural model in equation (1), augmented with the distributional assumption (4), is correct–this is something that we naturally assume in the first place–and simply work with some predefined order of the elements $\{\psi_1, \ldots, \psi_n\}$.

After neglect the constant terms, the conditional density of $\mathbf{s}_t$ during the normal times becomes

$$\begin{aligned} f^N(\mathbf{s}_t|\mathbf{s}_{t-1}) =& \gamma_1 \det(\mathbf{W}_1)^{-1} \exp\left\{-\frac{1}{2}(\mathbf{s}_t - \mathbf{A}\mathbf{s}_{t-1})'(\mathbf{W}_1\mathbf{W}'_1)^{-1}(\mathbf{s}_t - \mathbf{A}\mathbf{s}_{t-1})\right\} + \\ &+ (1-\gamma_1)\det(\mathbf{W}_1)^{-1}\det(\mathbf{\Psi}_1)^{-1/2} \times \\ &\exp\left\{-\frac{1}{2}(\mathbf{s}_t - \mathbf{A}\mathbf{s}_{t-1})'(\mathbf{W}_1\mathbf{\Psi}_1\mathbf{W}'_1)^{-1}(\mathbf{s}_t - \mathbf{A}\mathbf{s}_{t-1})\right\}. \end{aligned} \tag{7}$$

Similarly, after the obvious changes in the indexation, we get the density function $f(\mathbf{u}_t^C)$ and the conditional density $f^C(\mathbf{s}_t|\mathbf{s}_{t-1})$ for the crisis times. So, the joint conditional density function of the unrestricted model is

$$f(\mathbf{s}_t|\mathbf{s}_{t-1}) = d_N f^N(\mathbf{s}_t|\mathbf{s}_{t-1}) + d_C f^C(\mathbf{s}_t|\mathbf{s}_{t-1}),$$

where, as before, $d_N = 1$ and $d_C = 0$ during the normal times, and vice versa during the crisis times.

Collect all the parameters into the vector $\boldsymbol{\theta}$ and assume for convenience, when we have a sample of T periods, that the first T_1 periods consists of the normal times and the last $T_2 = T - T_1$ periods consists of a crisis period. (This assumption, of course, need not to hold, and there can be more than one crisis period. Then only, the conditional density and the log-likelihood function need to appropriately modified. This should be very straightforward.) Then, the corresponding log-likelihood function is

$$\begin{aligned} l(\boldsymbol{\theta}) &= \sum_{t=1}^{T} \log f(\mathbf{s}_t|\mathbf{s}_{t-1}) \\ &= \sum_{t=1}^{T_1} \log f^N(\mathbf{s}_t|\mathbf{s}_{t-1}) + \sum_{t=1}^{T_2} \log f^N(\mathbf{s}_t|\mathbf{s}_{t-1}). \end{aligned}$$

This can be maximized with the standard nonlinear optimization algorithms. Notice that in practice both of the time intervals T_1 and T_2 need to be long enough so that all of the parameters can be estimated. So, this rules out very short crisis periods.

3.2.3 Estimation of the restricted model

The SVAR model corresponding to the null-hypothesis of no-contagion, where we assume $\tilde{\mathbf{B}}^{\mathrm{N}} = \tilde{\mathbf{B}}^{\mathrm{C}} = \tilde{\mathbf{B}}$, is

$$\mathbf{s}_t = \mathbf{A}\mathbf{s}_{t-1} + \tilde{\mathbf{B}}\boldsymbol{\varepsilon}_t.$$

The corresponding reduced form model equals to the model in equation (3) with the error vector $\mathbf{u}_t$ following the mixed-normal distribution in equation (4), where the covariance matrices are decomposed as $\mathbf{\Sigma}_1 = \mathbf{W}\mathbf{W}'$ and $\mathbf{\Sigma}_2 = \mathbf{W}\mathbf{\Psi}\mathbf{W}'$. The density of the error term $\mathbf{u}_t$ is similar to the density function in equation (6) with only the obvious changes in the indexation. Likewise, the conditional density of $\mathbf{s}_t$ is similar to the conditional density function in equation (7). Then it follows that the log-likelihood function of the restricted model becomes

$$l(\boldsymbol{\theta}) = \sum_{t=1}^{T} \log f(\mathbf{s}_t | \mathbf{s}_{t-1}),$$

where the vector $\boldsymbol{\theta}$ again collects all the parameters of the restricted model. Also this function can be maximized with the standard optimization algorithms.

We can now test contagion by first estimating both of the models, the restricted and the unrestricted one, and then use the LR test to see if the data supports the null-hypothesis of no-contagion. For a stationary VAR model, the LR test statistic asymptotically follows the χ^2-distribution with $n(n+1)+1$ degrees of freedom. The condition

$$\tilde{\mathbf{B}}^{\mathrm{N}} = \tilde{\mathbf{B}}^{\mathrm{C}} \iff$$
$$\mathbf{W}_1 \left(\gamma_1 \mathbf{I}_n + (1-\gamma_1)\mathbf{\Psi}_1\right)^{1/2} = \mathbf{W}_2 \left(\gamma_2 \mathbf{I}_n + (1-\gamma_2)\mathbf{\Psi}_2\right)^{1/2}$$

imposes n^2 restrictions on the W-matrices, n restrictions on the Ψ-matrices, and one restriction on the mixture probability in our model.

3.2.4 Discussion on our assumptions

As it was stated in the beginning of subsection 3.1, we need to assume that all the main-diagonal elements of the matrix $\mathbf{C}$ of the Favero and Giavazzi model equal to zero. With the help of this assumption we can identify any structural change in the matrix $\tilde{\mathbf{B}} = \mathbf{B}\left(\mathbf{I}_n + \mathbf{C}\mathbf{D}_t\right)$ as contagion. This subsection mainly concentrates on discussing the implications of his assumption. But also, we point out that our framework provides, perhaps, a little more natural interpretation for crisis periods that that of Favero and Giavazzi.

For convenience, assume only two countries. Let us first consider normal times which means that both of the crisis time dummies d_{1t} and d_{2t} equal to zero. The Favero ang Giavazzi model in equation (1) then implies the following normal-times covariance matrix between the countries' spreads:

$$\mathrm{Cov}(s_{1t}, s_{2t}) = \begin{bmatrix} b_{11} & b_{12} \\ b_{21} & b_{22} \end{bmatrix} \begin{bmatrix} b_{11} & b_{21} \\ b_{12} & b_{22} \end{bmatrix},$$

where the normalization $\mathrm{Var}(\varepsilon_1) = \mathrm{Var}(\varepsilon_2) = 1$ is already incorporated. Consider next, for example, a crisis period where the turmoil originates in country 1, so we have $d_{1t} = 1$ and $d_{2t} = 0$. The crisis-times covariance matrix between the country spreads becomes

$$\text{Cov}(s_{1t}, s_{2t}) = \begin{bmatrix} b_{11} & b_{12} \\ b_{21} & b_{22} \end{bmatrix} \begin{bmatrix} (1+c_{11})^2 & (1+c_{11})c_{21} \\ (1+c_{11})c_{21} & c_{21}^2+1 \end{bmatrix} \begin{bmatrix} b_{11} & b_{21} \\ b_{12} & b_{22} \end{bmatrix}.$$

So we see that compared to the normal times, both c_{11} and c_{21} imply structural changes in the contemporaneous linkages between the countries during a crisis in country 1. However, the main role that the coefficient c_{11} plays is to create a crisis-contingent, exogenous increase in the variance of country 1's own spread. This is because a non-zero c_{11} means bigger than before within country effect of the structural shock ε_1.

On the contrary, the coefficient c_{21} has true contagion effects in the model. First, compared to the normal times, it raises the variance of country 2's spread. And remember, this rise is because of the crisis in country 1. Second, even when we have $c_{11} = 0$, a non-zero c_{21} will increase (or possibly decreases) the covariance between the countries' spreads during the crisis. This is because a non-zero c_{21} means a crisis-contingent, structural change in the transmission of ε_1 to country 2. Also, if we had $c_{21} = 0$ and $c_{11} \neq 0$, the only effect of the crisis in country 1 would be to increase the variance of s_{1t}.

This said, compared to the Favero and Giavazzi model, when we assume that $c_{ii} = 0$ for all $i = 1, \ldots, n$, we prevent the crisis-contingent, exogenously higher variance of the spread in the source country of the crisis. But to counterbalance this restriction, when it comes to the distribution of the structural shocks, we are not so restrictive as Favero and Giavazzo are. They assume the structural shocks vector $\boldsymbol{\varepsilon}_t$ follows a multi-normal distribution. In our framework this random vector does not need to be Gaussian but could follow some more fat-tailed distribution. The only thing we assume is that all the individual elements of the vector are uncorrelated with each other, and of course the variances are normalized to one.

Our framework, then, allows for a relatively intuitive interpretation of a beginning of a crisis period. Imagine there is an extreme (negative) realization of the structural shock in a country. As a reaction to this extreme event, the markets could trigger on some of the theoretical contagion mechanisms that were discussed in section 2. This would create structural changes into the structural shocks' transmission mechanisms and, so, increase the volatility in the system. Once switched on, it could take some time before the contagions mechanisms settle down. As the more volatile period persists, there is a crisis. This way, in our framework, although contagion still would be an exogenous event (triggering of the contagion mechanisms), its reason would be endogenous (an extreme realization of one country's idiosyncratic shock). In contrast, in the Favero and Giavazzi model a crisis is a fully exogenous event.

Our framework has one additional advantage compared to the Favero and Giavazzi model and, also, to the majority of other contagion tests presented in the literature. Our test do not require us to identify the source country of contagion (or a crisis). Also, we can easily handle a case where there might be several source countries in the same, for example because several countries almost simultaneously meet extremely negative idiosyncratic shocks. For our test, it suffices to identify the spell of a crisis. Of course, sometimes it might be hard to do so, but, as it was argued in the introduction, often the beginning (an the end) of a crisis period can be identified more or less accurately. Similarly, the beginning of the euro sovereign debt crisis can be traced to the turn of the years 2009 and 2010.

4 Application of our test to the euro government bonds

In November 2009 the then-newly elected Greece government informed that the country's public debt level was almost double the size the official statistics had claimed before. But it was not until the beginning of 2010 before the situation escalated into a crisis with Greece being bailed out, and especially the Irish and Portuguese spreads substantially increasing (see figure 1).

INSERT FIGURE 1 AROUND HERE.

In this section we apply our contagion test to analyze contagion between five eurozone member countries: Greece, Ireland, Portugal, Spain, and Italy. As emphasized by shadings in the figure 1, we consider the year 2009 as the normal times and the year 2010 as the crisis time. Of course, in 2009 the world was already in the middle of the financial crisis that originated in the U.S. subprime mortgage markets. However, it was not until the beginning of the euro sovereign debt crisis that the markets started to question the solvency of some of the euro member countries. So, the year 2009 was still more or less a period of normal mutual linkages between the eurozone's sovereign bond spreads.

The data is from the Eurostat database and consists of the daily secondary market yields of the ten years government bonds. As detailed in the beginning of section 3, the yields have then been transformed into spreads over the German bond. The sample period consists of 522 daily observations. Every country has missing observations[9], so before taking the spreads all missing values have been substituted with the previous available observation.

In order to test for the null hypothesis of no contagion against the alternative hypothesis of contagion in 2010 (see equation (2)), we first need to estimate the

[9]Germany has 6, Ireland 13, Greece 10, Spain 8, Italy 5, and Portugal 5.

restricted model that is detailed in section 3.2.3 for the whole sample period. Then, we need to estimate the unrestricted model that is detailed in section 3.2.2. After this we can test for contagion by testing the restricted model against the unrestricted one with using the LR test. As long as our model is stationary, the LR test statistic asymptotically follows the χ^2-distribution with n^2 degrees of freedom, where n is the number of sample countries and n^2 equals to the number of elements in the B-matrix.

In order to select the correct lag length of the (S)VAR model, I have estimated the restricted model with lags from one to three for the full sample period and then used the Bayesian information criterion (BIC) to determine the correct lag length. The BIC supports selecting the lag order of one. Also, we will allow for non-zero constant (deterministic term) in the model.

4.1 Estimation of the restricted model

Table 1 shows the estimation results for the restricted model. The first part of the table shows the estimated constant and the matrix $\mathbf{A}$; for example, Ireland(-1) means the lagged Irish value. It is evident that it is mainly the own lagged values that an any significant effect on the countries' spreads. The next rows report the estimated matrix $\mathbf{W}$, $\mathbf{\Psi}$, and mixture probability γ. Because it is these parameters ($\mathbf{W}$, $\mathbf{\Psi}$ and γ) that determine the contemporaneous linkages between the countries (the B-matrix), we see that are many significant instantaneous effects across the countries.

However, as we have simply assumed the descending order for the main diagonal elements ψ_i, and so we are not after actually identify the instantaneous linkages between the countries (see footnote 8), there is no point in calculating the B-matrix. It is the estimated covariance matrix $\mathbf{\Sigma}_1 = \mathbf{W}\mathbf{W}'$ that corresponds here to the low volatility and $\mathbf{\Sigma}_2 = \mathbf{W}\mathbf{\Psi}\mathbf{W}'$ to the high volatility distribution. The mixture probability (γ) tells the probability of the reduced form error $\mathbf{u}_t$ being from the normal distribution with the covariance matrix $\mathbf{\Sigma}_1$. This probability is around 0.66.

INSERT TABLE 1 AROUND HERE.

4.2 Estimation of the unrestricted model

Table 2 reports the estimated unrestricted model where we now have parameters $\mathbf{W}_1$, $\mathbf{\Psi}_1$ and γ_1 for the normal times (the year 2009), and $\mathbf{W}_2$, $\mathbf{\Psi}_2$ and γ_2 for the normal times (the year 2010). First, the estimated elements of the matrix $\mathbf{A}$ are quite similar to those in the restricted model. And, especially, the own lags are mostly the ones that have statistically significant coefficients. The estimates of the matrices $\mathbf{W}_1$ and $\mathbf{W}_2$, and $\mathbf{\Psi}_1$ and $\mathbf{\Psi}_2$ are little different from the estimates of $\mathbf{W}$ and $\mathbf{\Psi}$ of the restricted model.

Notice that there are again many statistically significant elements in both of the estimated W-matrices, and all the elements in the matrices $\mathbf{\Psi}_1$ and $\mathbf{\Psi}_2$ areS statistically significant. This implies that there statistically significant contemporaneous cross-country effects during both of the years. According to the estimated mixture probabilities γ_1 and γ_2 the probability of the reduced form errors being drawn from the distribution with the covariance matrix $\mathbf{\Sigma}_1$ was almost the same in 2009 and 2012, 0.77 and 0.73 per cent respectively. These probabilities are around ten per cent larger than in the restricted model.

INSERT TABLE 2 AROUND HERE.

4.3 Contagion test & Weighted correlation coefficients

The LR test testing the restricted model against the unrestricted one gets value 382.8 which is greater than the critical value of χ^2-distribution with 31 degrees of freedom at any reasonable significance level; for example, the critical value at the 5 % significance level is 45.0. Hence, we reject the null hypothesis of no-contagion and conclude that there is evidence of contagion between the countries in 2010. A detailed analysis of the bilateral contagion effects between every country pair would require a full identification of the SVAR model which means we should be able to identify the "correct" permutation of the elements on the $\mathbf{\Psi}$ matrices' main diagonals. We leave the question of the full identification for the future research, but for instance, in a little different kind of framework, Kohonen (2012) proposes to use a specific news variable to fully identify the structural model.

However, even without fully identifying the SVAR model, we are able to analyze little further the role each of our sample countries play in the crisis. Based on the estimated unrestricted mode, table 3 reports the mixture probability weighted correlation coefficients for both of the years. These correlation coefficients summarize the countries' bilateral correlations in both of the multi-normal distributions, $N(\mathbf{0}, \mathbf{\Sigma}_1)$ and $N(\mathbf{0}, \mathbf{\Sigma}_2)$, of our mixture-normal distributions. The weighted correlation coefficients, for example for the year 2009, between countries i and j were calculated as the following:

$$r_{ij}^{(w)} = \gamma \cdot r_{ij}^{(1)} + (1 - \gamma) \cdot r_{ij}^{(2)},$$

where γ is the estimated mixture probability of the year, for example $\gamma_1 = 0.77$ for 2009; and the correlation coefficients $r_{ij}^{(1)}$ and $r_{ij}^{(2)}$ were calculated based on the year's estimated covariance matrices $\mathbf{\Sigma}_1$ and $\mathbf{\Sigma}_2$, respectively.

INSERT TABLE 3 AROUND HERE.

Table 3 emphasizes with bold numbers the weighted correlation coefficients of 2010 of those pairs of the countries that saw an increase in their correlation coefficients from the year 2009. The first observation is that only for three country pairs the year 2012 correlation coefficient is greater than that of the year 2009, these pairs are Ireland-Greece, Ireland-Italy, and Spain-Italy. Even the Ireland-Italy 2010 coefficient is almost equal to the 2009 coefficient. Testing the equality would require us to be bale to fully identify the model so that we would know which of the B-matrix elements to restrict.

The second observation is that for both Greece and Portugal, their correlation coefficients with the other countries fall–even quite substantially–in 2010 from 2009. So, at least according to these results, Greece and Portugal become less integrated with the other member countries during the crisis. This decoupling suggests that the rising bond spreads in these two countries was not transmitted to the other countries. Rather, the results suggest towards a "flight-to-quality" effect; withdrawal of investments from Greece and Portugal in favor of the other countries. But, a rigorous testing of this claim, is not possible with our framework. Deducting an appropriate test is outside the scope of this paper but could be an interesting topic for the future research.

The explanation for the mostly decreasing weighted correlation coefficients is not so much a decrease in covariances between the countries but an increase in the countries variances during the crisis. Especially, in the high-volatility distribution of the the year 2012 with the covariance matrix $\mathbf{\Sigma}_2^{(2)} = \mathbf{W}_2\mathbf{\Psi}_2\mathbf{W}_2'$ all the countries have much higher variances than in the corresponding covariance matrix of the year 2009, that is $\mathbf{\Sigma}_2^{(1)} = \mathbf{W}_1\mathbf{\Psi}_1\mathbf{W}_1'$. So, our framework allows us to easily adjust the conditional correlation coefficients for possible, crisis-contingent heteroskedasticity in the error distributions.

In their influential paper, Forbes and Rigobon (2002) show that because crises usually lead into higher variances, conditional correlation coefficients that are not adjusted for this crisis-contingent heteroskedasticity might provide very biased results. Based on this result, they then show that the results of many of the earlier contagion studies that analyzed contagion with conditional correlation before and after a crisis were biased. But unlike Forbes and Rigobon who need to assume no endogeneity between the sample countries, our framework deals with endogeneity issues automatically. In addition, calculation of our weighted correlations coefficients do not require us take any stance which of the countries see their variance to increase, also this is indirectly taken care of by our model and we let the data to speak for itself.

5 Concluding remarks

In this paper we have developed a new and simple test for contagion. The test is based on the ide of augmenting the SVAR model of Favero and Giavazzi (2002) with an assumption concerning the distribution of the reduced form errors. The errors are assumed to follow a mixed-normal distribution. This allows us to employ the SVAR identification method proposed by Lanne and Lütkepohl (2010). The identification method uses non-normalities, not parameter restrictions as a source of the extra information that we need to identify our model. This way we are able to estimate the parameters of the instantaneous effects between the variables. In line with the prevailing empirical contagion literature, the main idea of our contagion test is to test whether these parameters are stable across the normal and crisis times. This approach constitutes a simple to apply test for contagion where the standard test theory of ML estimation is applicable.

In the empirical application of the paper, we test for contagion in the eurozone government bond spreads in 2009–2010. The sample includes five countries (Ireland, Greece, Spain, Italy and Portugal) whose ten years government bond spreads of Germany are considered. Null-hypothesis of no contagion is rejected. In addition, we calculate mixture probability weighted correlation coefficients for both of the years. According to these coefficients, the contagion effects seem quite complex and we are tempted to conclude that there were not any single source country of contagion. Such complex contagion effects–contagion also meaning any investors ”flight to quality”–are often forgotten in the empirical contagion literature.

Our test still relies on predefined normal and crisis periods. Although for many recent crises more or less clear data cut-off points can be pointed out, in some cases, our test might be susceptible for the selected crisis periods. There are several ways to extend our model framework. One would be to consider some more fat tailed error distributions than a mixed normal distribution; a mixed t-distribution could be one candidate. Furthermore, Lanne and Lütkepohl (2010) show how to apply their identification/estimation method to vector error correction model (VECM). Because many financial variables in levels show evidence of unit roots, extending our test to VECM framework might be fruitful.

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Table 1: Restricted model: Parameter estimates

Explanatory variable	**Dependent variable** Ireland	Greece	Spain	Italy	Portugal
Constant	0.003	0.052^{**}	0.000	0.022	-0.009
	(0.022)	(0.026)	(0.015)	(0.012)	(0.018)
Ireland(-1)	0.990^{***}	-0.006	0.004	0.003	-0.003
	(0.009)	(0.011)	(0.006)	(0.005)	(0.008)
Greece(-1)	0.007	0.992^{***}	0.007	0.004	0.019^{***}
	(0.008)	(0.010)	(0.005)	(0.004)	(0.007)
Spain(-1)	-0.025	0.104^{***}	0.951^{***}	-0.009	-0.045
	(0.035)	(0.04)	(0.022)	(0.018)	(0.030)
Italy(-1)	0.012	-0.091^{**}	0.011	0.970^{***}	0.038
	(0.032)	(0.037)	(0.022)	(0.018)	(0.027)
Portugal(-1)	0.009	-0.005	0.004	0.001	0.968^{***}
	(0.021)	(0.025)	(0.013)	(0.011)	(0.018)
Matrix W	**Col. 1**	**Col. 2**	**Col. 3**	**Col. 4**	**Col. 5**
Row 1	0.028^{***}	0.006	0.048^{***}	0.042^{***}	-0.023^{***}
	(0.006)	(0.072)	(0.013)	(0.008)	(0.006)
Row 2	0.027^{***}	0.061^{***}	-0.007	0.045^{***}	-0.026^{***}
	(0.009)	(0.014)	(0.100)	(0.009)	(0.006)
Row 3	0.013^{***}	-0.001	0.000	0.046^{***}	0.018^{***}
	(0.002)	(0.004)	(0.004)	(0.003)	(0.005)
Row 4	0.009^{***}	-0.001	0.000	0.035^{***}	-0.027^{***}
	(0.002)	(0.003)	(0.004)	(0.003)	(0.004)
Row 5	0.043^{***}	-0.006	0.007	0.024^{***}	-0.020^{***}
	(0.004)	(0.013)	(0.010)	(0.005)	(0.004)
Matrix Ψ	ψ_1	ψ_2	ψ_3	ψ_4	ψ_5
	24.212^{***}	12.499^{***}	10.867^{***}	3.998^{***}	0.849^{***}
	(4.091)	(1.955)	(1.578)	(0.630)	(0.134)
Mixture prob.	γ				
	0.658^{***}				
	(0.032)				

NOTE:
Standard errors obtained from the inverse Hessian of the log-likelihood function.
(**) / (***) indicates statistical significance at 5 % / 1 % significance level.
The log-likelihood function gets value 5154.8.

Table 2: Unrestricted model: Parameter estimates

Explanatory variable	**Dependent variable** Ireland	Greece	Spain	Italy	Portugal
Constant	0.001	0.047^{**}	-0.005	0.013	-0.027
	(0.020)	(0.021)	(0.014)	(0.012)	(0.018)
Ireland(-1)	0.997^{***}	-0.009	0.002	0.001	0.005
	(0.009)	(0.011)	(0.006)	(0.005)	(0.009)

Continued on next page

Table 2 – continued from previous page

Greece(-1)	0.008	1.006***	0.009	0.006	0.024***
	(0.008)	(0.009)	(0.005)	(0.004)	(0.008)
Spain(-1)	−0.029	0.059	0.954***	−0.010	−0.006
	(0.032)	(0.038)	(0.023)	(0.019)	(0.034)
Italy(-1)	0.000	−0.066**	0.020	0.982***	0.032
	(0.030)	(0.030)	(0.021)	(0.017)	(0.026)
Portugal(-1)	0.007	−0.020	−0.003	−0.005	0.938***
	(0.021)	(0.024)	(0.014)	(0.011)	(0.020)
Matrix$\mathbf{W}_1$	**Col. 1**	**Col. 2**	**Col. 3**	**Col. 4**	**Col. 5**
Row 1	0.035***	−0.022	−0.012	0.047***	0.034**
	(0.013)	(0.021)	(0.012)	(0.011)	(0.016)
Row 2	0.029	0.027	0.012	0.034***	0.018
	(0.015)	(0.018)	(0.009)	(0.007)	(0.012)
Row 3	0.003	0.007**	0.003	0.007	0.052***
	(0.004)	(0.003)	(0.005)	(0.015)	(0.004)
Row 4	0.004	0.005	0.011	0.039***	0.018
	(0.003)	(0.004)	(0.007)	(0.006)	(0.013)
Row 5	0.006	0.009	−0.017**	0.041***	0.024
	(0.006)	(0.006)	(0.008)	(0.008)	(0.014)
Matrix$\mathbf{W}_2$	**Col. 1**	**Col. 2**	**Col. 3**	**Col. 4**	**Col. 5**
Row 1	0.075***	−0.002	0.019	0.027**	−0.002
	(0.008)	(0.025)	(0.016)	(0.011)	(0.014)
Row 2	0.049**	0.105***	−0.034	0.038**	−0.004
	(0.023)	(0.025)	(0.066)	(0.017)	(0.017)
Row 3	0.023***	0.020	0.032**	0.021	−0.041***
	(0.007)	(0.02)	(0.014)	(0.015)	(0.014)
Row 4	0.016***	0.016	0.029***	0.026***	−0.001
	(0.006)	(0.018)	(0.011)	(0.008)	(0.013)
Row 5	0.062***	0.029	0.054**	−0.043***	0.016
	(0.011)	(0.038)	(0.024)	(0.016)	(0.016)
Matrix $\mathbf{\Psi}_1$	$\boldsymbol{\psi}_1$	$\boldsymbol{\psi}_2$	$\boldsymbol{\psi}_3$	$\boldsymbol{\psi}_4$	$\boldsymbol{\psi}_5$
	18.318***	13.616***	5.15***	2.183***	1.171***
	(4.232)	(2.989)	(1.182)	(0.560)	(0.355)
Matrix $\mathbf{\Psi}_2$	$\boldsymbol{\psi}_1$	$\boldsymbol{\psi}_2$	$\boldsymbol{\psi}_3$	$\boldsymbol{\psi}_4$	$\boldsymbol{\psi}_5$
	16.577***	8.489***	6.41***	2.131***	1.173**
	(3.657)	(2.169)	(1.467)	(0.501)	(0.575)
Mixture prob.	γ_1				
	0.769***				
	(0.036)				
Mixture prob.	γ_2				
	0.725***				
	(0.049)				

NOTE:
Standard errors obtained from the inverse Hessian of the log-likelihood function.
(**) / (***) indicates statistical significance at 5 % / 1 % significance level.
The log-likelihood function gets value 5346.2.

Table 3: Mixture probability weighted correlation coefficients

	Greece 2009 (2010)	Spain 2009 (2010)	Italy 2009 (2010)	Portugal 2009 (2010)
Ireland	0.49 (0.41)	0.44 (**0.61**)	0.57 (**0.58**)	0.69 (0.58)
Greece	–	0.64 (0.43)	0.78 (0.40)	0.65 (0.31)
Spain	–	–	0.47 (**0.78**)	0.64 (0.45)
Italy	–	–	–	0.77 (0.55)

The bold 2010 numbers indicate an increase from 2009.
The weighted correlation coefficient between countries i and j was calculated in the following way: $r_{ij}^{(w)} = \gamma \cdot r_{ij}^{(1)} + (1-\gamma) \cdot r_{ij}^{(2)}$, where γ is the estimated mixture probability of the period, and $r_{ij}^{(1)}$ and $r_{ij}^{(2)}$ are correlation coefficients based on the periods estimated covariance matrices $\mathbf{\Sigma}_1$ and $\mathbf{\Sigma}_2$, respectively.

Figure 1: Ten years government bond spreads over Germany, 2009–2010 (daily data)