GREThA
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Économie Théorique et Appliquée

The international contagion of short-run interest rates during the Great Depression

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Cahiers du GREThA
n° 2015-11
April

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Abstract

The aim of this article is to clearly identify the mechanisms of the money market spillovers between the United States, the United Kingdom and France during the interwar period. To describe these mechanisms in detail, a BEKK model, in which we introduce a structural break, is adopted. Our analysis sheds new light on key historical issues: Was the crisis imported into the US? Did France set off interest rate volatility in the rest of the world during the thirties? Does the propagation process of interest rate volatility corroborate the "Golden Fetters" hypothesis?

Mots-clés : Contagion, Gold Exchange standard, interest rates

La contagion internationale des taux d'intérêt durant la grande dépression

Résumé

Le but de cet article est d'identifier les mécanismes de contagion de taux d'intérêt à 3 mois à l'œuvre entre la France, la Grande-Bretagne, les Etats-Unis, les trois principaux pays créditeurs durant l'entre-deux guerres. Afin de décrire ces mécanismes en détail, un modèle BEKK dans lequel est introduit une rupture structurelle est adopté. Notre analyse éclaire sous un jour nouveau trois questions historiques fondamentales : La crise de 1929 a-t-elle été importée aux Etats-Unis ? Est-ce la France qui a exporté ses volatilités de taux dans le reste du monde ? Est-ce que le processus de propagation de volatilité des taux accrédite ou non la thèse des « golden fetters » ?

Keywords: Contagion, Etalon Or, Taux d'intérêt

JEL: : **N12, N14, N22, N24, E4**

Reference to this paper: MAVEYRAUD Samuel (2015) The international contagion of short-run interest rates during the Great Depression, *Cahiers du GREThA*, n°2015-11.

http://ideas.repec.org/p/grt/wpegrt/2015-11.html.

Introduction[1]

This paper focuses on the contagion of interest rates before and during the Great Depression and addresses a key historical issue: Does the "Golden Fetters" hypothesis (Eichengreen, 1992) hold regarding the mechanisms of crisis contagion? To that end, we wonder whether the tensions in American money markets spread to English and French markets (or conversely), and whether Black Thursday modified the spillover mechanisms more dramatically than the breakdown of the Gold Exchange Standard itself.

To cast new light on these questions, a trivariate BEKK model (Baba et al. (1991)) has been adopted. This model reveals, in particular, the spreading mechanisms governing the evolution of variances and covariances in discrepancies between the 3-month Treasury Bond yields of France, Great Britain and the USA. The originality of our modeling lies in the introduction of a structural break in the equation of central tendency and equations of conditional variances and covariances (as proposed by Beirne et al., 2013). Taking this break into account helps to explicitly test the potential modifications of the spillover phenomenon between money markets.

We undertake this analysis in two steps. First, we consider the whole period, from 1921 to 1936, by introducing a structural break. The ensuing model, with its structural break, enables the nature of contagion throughout the whole period to be revealed, once the most relevant shock has been internalized. We have tested five equally important candidates in History in order to determine a break during this period: the triggering of the financial crisis in the US, the devaluation of the Pound in September 1931, the declaration of the Dollar inconvertibility in March 1933, the London Conference in June 1933 and the official devaluation of the Dollar in January 1934.

[1] Our thanks go to the participants in the Economic History and Cliometrics seminar (Sciences Po Lyon, November 18, 2013) for their comments and suggestions, and especially to Alexandru Minea and Annie Orsières. We are also very grateful to the participants in the "Cliometrics" sessions of the 31st Annual International Symposium on "Money, Banking and Finance" of the European Research Group (GdRE CNRS "Monnaie, Banque, Finance"), Sciences Po Lyon, 19-20 June 2014, and particularly to Price Fishback, Christopher Meissner, Pamfili Antipa, Olivier Damette, Eric Girardin, Anders Ogren and Jon Moen for their fruitful remarks and suggestions. We would like to thank the participants in the MMF Symposium, Durham, 17-19 September 2014 and, in particular, Jean-Bernard Chatelain, Peter Sinclair and George Bratsiotis for their precious comments. All errors and inadequacies remain ours.

Once having identified, with the maximum of likelihood, the most relevant break among the five candidates, we distinguish two sub-periods: before and after this break. The first goes from 1921m01 to 1929m06, the second one from 1930m06 to 1936m12. As is usual in this literature, we deliberately do not take into account the period around the structural break, in order to avoid turbulences and noises. Thus, we manage to assess appropriately the two sub-periods as two distinct periods, and then compare the dynamics of the relationship between interest rates[2] during those two periods.

This article is organized as follows: the first section recapitulates comparative studies of contagion using historical data; the second section surveys existing literature on contagion; the third one draws connections between the contagion mechanisms during the Great Depression and the "Golden Fetters" hypothesis; in the fourth section, we present data, methodology, and the econometric model (BEKK with a structural break); our findings are exposed in Section 5; discussion is driven in Section 6; the last section concludes.

1. Comparative studies of contagion based on historical data

Very few articles use elaborated tools to study contagion in historical perspective. The seminal paper in economic history which addresses the question of contagion in financial crises is that of Bordo and Murshid (2001). Two levels of analysis can be distinguished in their study: a comparison of contagion phenomena over time; a specific analysis of contagion over the Gold Exchange Standard (GES) period.

Bordo and Murshid's initial research goals are quite straightforward. They aim at comparing empirical data on contagion and crises from the past with modern episodes of contagion, in order to elaborate a more thorough explanation of present-day crises, but also in order to destroy

[2] The word interest rate must be understood as actuarial interest rate (a synonym for yield)

common misconceptions about the intensity and supposedly "exceptional" severity of modern crises. They want to obtain better insights into the most suitable economic and monetary regimes that would help to avoid favouring contagion across financial markets. They conduct this research not only to infer which economic policies and regulations could best suit a given country with its own particular monetary regime and economic situation, but also to find inspiration for policy suggestions to solve current crises.

To carry out their comparative analysis of contagion, Bordo and Murshid (2001) use the weekly data of NYSE-traded bonds emitted by the following countries: Argentina, Belgium, Brazil, Canada, Chile, Denmark, Finland, France, Germany, Italy, the Netherlands, Sweden, Switzerland, the UK and the US. Their purpose is to ascertain whether there are stronger market co-movements after turbulent periods (i.e. the financial crises at the end of the 19th century, the Great Depression of 1929, and the Asian crises of the 1990s). They examine, in six-month time frames, the evidence of increased cross-market correlations after a major shock. During the interwar, there seems to be stronger cross-market linkages, notably via a higher co-movement of bond prices after a shock. On the contrary, during the Mexican crisis at the beginning of the 1980s, and after the speculative attack against Thailand in 1997, Bordo and Murshid (2001) find no evidence of stronger cross-market co-movements, thus making it impossible to assert that contagion now is stronger than it was in the past.

When assessing for regional patterns of contagion, Bordo and Murshid (2001) note that, in the past, contagion patterns usually found their source in the UK, and were then propagated toward other European countries. Another common historical pattern of crisis transmission in the last century has been from the core European countries to the peripheral ones. This pattern of shock transmission seems to have remained unchanged in recent times.

Finally, Bordo and Murshid (2001) find that tangible cross-market co-movements have occurred in *both* tranquil and tumultuous periods, but they are not able to establish a strong case for contagion today.

Concerning the Great Depression itself, Bordo and Murshid (2001) provide a review of the basic facts which, they consider, characterized the 1929 contagion. The spread of contagion was manifested in two effects: first, price and output decreased all over the world, a series of decreases that led the US to stop foreign lending. Second, the depression was accompanied by the banking panics sparked off, not only in the US, but throughout the world.

They sum up the main features of the 1929 contagion, and adopt as their own the explanations provided by Eichengreen (1992), acknowledging that the "US-induced crisis notoriously experienced international propagation.[…] The depression spread through the channels of international gold flows, money supplies, and the capital flight" (Bordo and Murshid, 2001). Do they actually provide evidence that the "Golden Fetters" hypothesis which they endorse is the key to understanding contagion during the interwar period, and do their outcomes corroborate this view? One point remains unclear in their analysis: they provide evidence of intense co-movements in the aftermath of the 1929 crisis, notably after Sterling and the Dollar were devalued, respectively in 1931 and 1933. Why then should contagion be stronger only once the GES system has exploded and not before? Does this finding correctly reproduce the "Golden Fetters" hypothesis? This point needs further clarification (see Discussion, Section 3).

The second paper that deals with contagion over the interwar period is that of Accominotti (2011). Contagion is assessed via Principal Component Analysis performed on an Exchange Market Pressure index, sovereign bond spreads and stock market returns (1928-1936). The exchange market pressure (EMP) index, first introduced by Girton and Roper (1977), is built as a weighted average of the monthly changes in countries' international reserves and exchange rate volatilities. This index has been generalized by Eichengreen, Rose and Wyplosz (1995, 1996, July

1996) and built by Accominotti (2011) for the interwar period. Spreads on sovereign bonds traded in New York (monthly prices for 29 countries from January 1928 to February 1934) relative to the yield on long-term United States bonds, measure the default risk. Monthly series for stock market returns covering 14 national stock exchanges, from February 1928 to December 1936, are also included in the database. Accominotti's subsequent analysis of contagion consists in a Principal Component Analysis that explores the co-movements between the series of EMP index, spreads on sovereign bonds and stock market returns.

The author finds that global stress in the early 1930s was related to "a liquidity shortage on international capital markets, which culminated in the huge capital flow reversal of the year 1931. The geography of financial troubles at the beginning of the 1930s closely matched the distribution of countries between creditors and debtors. The crisis first propagated to the large importers of capital. With the huge liquidation of international investments, countries that were previously relying on foreign borrowing to finance their current account deficits fell victim of speculative attacks"[3]. By contrast, the largest creditors of the 1920s repatriated those capital flows home in the early 1930s, a situation which, according to Accominotti (2011), is the main characteristic of this period.

Unfortunately, neither that author nor Bordo and Murshid (2001) define what exactly they mean by contagion. In Bordo and Murshid (2001), the seminal paper in economic history which addresses the question of contagion in financial crises, the definition of contagion is only based on the correlation of prices. The three indicators estimated by Accominotti (2011) cannot be considered as relevant indicators of contagion: in particular, an increase in the spread of government bonds does not necessarily reveal an increase in contagion, but almost always signifies a higher risk premium.

[3] The same idea is repeated in a more recent Working Paper by Accominotti and Eichengreen (2013): The sharp increase in stock market volatility in the major financial centers that exported capital at the end of the 1920s is described as being at the origin of the decline in foreign lending to borrowing countries.

Since the definition of contagion remains vague or weak regarding the literature on the topic, none of these papers proposes a rigourous econometric approach of contagion (for instance, problems due to auto-regressive conditional heteroskedasticity on time series are not taken into account). Moreover, the direction of contagion is never statistically tested. In our article, we base our analysis of contagion on the most recent research on the subject in order to re-examine the "Golden Fetters" hypothesis.

2. Contagion: a survey

The existing literature includes many studies involving contagion, but scholars do not seem to agree on one generally accepted definition of this phenomenon. A recent paper on contagion gives up to eleven definitions of this concept (Forbes, 2012).

As Sebastian Edwards (2000) points out, the use of the term "contagion" in economics is relatively new, only dating back from the early 1990s. However, the word itself is not new: it stems from the Latin verb "contingere", which means "to come in contact with" or "to pollute" and, initially, the term was coined in the 14th century to describe a pathological phenomenon, i.e. the "transmission of a disease by direct or indirect contact". More exactly, in epidemiology, contagion designates the very vast - greater than originally expected - spread of a disease (Edwards, 2000).

The only clear-cut certitude found in the literature is that contagion cannot be assimilated to causality (Forbes, 2012). Whereas causality is a strong, direct link between two distinct phenomena, cause and effect, contagion is not as clear-cut and direct. Hence, these two phenomena should be differentiated.

Two main conceptions of contagion can be distinguished in the literature: a global and systemic definition, mainly based on macroeconomic aggregates, and another one founded on price movements.

The global and systemic approach

Eichengreen et al. (July 1996) define contagion as "a situation where the knowledge that there is a crisis elsewhere increases the probability of a domestic crisis".

Following this broad definition, several papers focus more on the extent of contagion than on its origin and direction, defending the idea that contagion can concern not only specific, correlated markets or countries, but that it can become global. According to a popular definition by Masson (1998, 1999), there exist three types of contagion mechanisms: "monsoonal effects", "spillover effects" - a term integrated in one of Forbes' latest papers (Forbes, 2012) and in several other pieces of literature - and residual contagion mechanisms. Whereas monsoonal effects focus on contagion in a group of countries stemming from a common cause, such as policies in common adopted wholesale by industrialized countries, spillover effects are crises that originate in one specific market or country and that then "may affect the macroeconomic fundamentals" [i.e. GNP, prices, the balance of payments situation, the level of unemployment] in another market or country (Masson, 1998). This type of contagion is also acknowledged by Kaminsky and Reinhart, who call it "fundamentals-based contagion" (Kaminsky and Reinhart, 2000). Finally, residual contagion phenomena are "those that cannot be identified with observable changes in macroeconomic fundamentals", i.e. the crises that originate in one country and spread to another because those countries, or the markets involved, are subject to "multiple equilibria", i.e. "self-fulfilling expectations" held by investors in the country or market involved (Masson, 1999). A similar, popular definition of contagion as a residual, negative effect has been adopted by Edwards (2000), who indicates three scales of shock propagation: global, that coming from one correlated country, and residual. He classifies residual contagion as being "all that exceeds market participants' expectations" (Edwards, 2000).

As mentioned by this author, it appears more useful to apply the notion of contagion to more restricted - and perhaps more quantifiable - phenomena.

The price movements approach: from interdependence to shift contagion

Kodres and Pritsker (2002) observe that contagion has been defined by some authors as any price movement, i.e. as "a price movement on one market resulting from a shock in another market", and hence can be assimilated to a spillover effect.

Moreover, this approach considers contagion as being a correlation between several markets, countries or groups of countries. In her survey of literature of the existing definitions of contagion, Kristin Forbes (2012) shows that contagion is used to refer to:

- a co-movement across several markets or countries. Morgenstern (1959) asserts that financial crises are likely to spread either simultaneously to several countries or in multiple phases, from those countries where the crisis started to the other, "peripheral" ones. It is implicit that contagion usually traces its roots in one market or country and then, at a later stage, spreads to another (or several other) markets or countries (Kindleberger and Aliber (2011));
- a phenomenon that needs multiple occurrences in order to exist. For example, in Boyer et al. (2006), contagion is described as the excess correlation between stock markets. Dungey et al. (2010) provide us with a more explicit definition, saying that contagion is the bunch of "effects of contemporaneous movements in asset returns across countries".

We see, then, that this literature is starting to admit the existence of contagion and its specificity, as compared to mere market interdependence or minor spillover phenomena. For instance, Forbes and Rigobon (2002) and Bordo and Murshid (2001) measure interdependence as cross-market correlations, and treat contagion as a stronger degree of cross-market correlation.

The first-ever work to mention "excess" is a 2003 article (Bae et al. (2003), cited by Forbes (2012), which defines contagion as the "exceedance events in a region that are not explained by covariates" - i.e. interest rates, volatility, exchange rates - but rather by severe shocks in excess or in defect of the "5th and 95th quantile of marginal return distribution" in equity indexes. This definition paves the way to quantifying excessive or abnormal contagious phenomena – and, hence, to defining contagion more accurately. Boyer et al. (2006) define contagion as the "excess

correlation" – i.e. a tangible increase in correlation of accessible and investable securities across financial markets – "between stock markets during periods of high volatility". A slightly more inclusive definition of contagion is that elaborated by Bekaert et al. (2014), according to which contagion is "the co-movement in excess of that implied by the factor model, i.e. above and beyond what can be explained by fundamentals taking into account their natural evolution over time".

Contagion, however, is not only "excess correlation": it also implies an alteration of the nature of the cross-market relationship, i.e. whether there is a change in market interdependence before and after the shock. Hence, some scholars advocate the use of the expression "shift contagion", which refers to a tangible shift in cross-market correlation after a shock in one single country, as opposed to interdependence, which is a mere "continuation of the same cross-market linkages that exist during more tranquil periods" (Forbes and Rigobon (2001)).

As Forbes and Rigobon (2001) point out, this notion of change is what distinguishes contagion from simple correlation: as such, the existence of a shift in market correlation before and after the contagious event is worth analyzing. Forbes and Rigobon (2001) aim at proving that the term contagion – or, in their words, "shift-contagion" - "implies that cross-market linkages are fundamentally different after a shock to one market". In order to do so, they analyze the correlation between the concerned markets before and after the shock: if their correlation has increased "significantly", then that episode can be classified as shift-contagion. Moreover, what characterizes shift-contagion is the fact that pre-crisis transmission mechanisms are different than transmission mechanisms that occur during or after the crisis. This implies that, during the crisis, contagion channels appear that would not exist in "normal" or more tranquil periods: hence, Forbes and Rigobon (2001) assert that shift contagion occurs when a crisis considerably transforms a market and its mechanisms by inducing a "structural shift". However, Forbes and

Rigobon (2001) fail to provide generally accepted indications as to the minimal increase or value range that need to be covered by contagion in order for it to be significant.

BEKK model with a structural break

Implementing a BEKK model with a structural break provides an accurate tool to measure contagion which, moreover, allows the limitations mentioned above to be overcome. First, it measures spillover effects via the transmission of volatility from one variable to another. Second, it identifies both the origin and direction of contagion. Third, the excess volatilities are taken into account in the variances-covariances equation, since the BEKK model is based on a GARCH approach. Fourth, the introduction of a structural break in the BEKK model enables shift contagion phenomena to be studied.

This regime-switching analysis is particularly appropriate for studying contagion over the interwar period, and also for testing its dominant explanation, the "Golden Fetters" hypothesis.

3. Contagion during the Great Depression and the "Golden Fetters" hypothesis

In this section, we recall the key features of the "Golden Fetters" hypothesis. According to Eichengreen (1992), two factors produced the stability of the pre-war Gold Standard (GS): credibility (commitment to par was not violated prior to 1914), and cooperation between central banks, which rendered the commitment an international one. This cooperative management by central bankers was quite different from the leader/follower approach of the "Theory of hegemonic stability" developed by Kindleberger (1973). Over the pre-war GS period, Eichengreen (1992) argues, the Bank of England acted in fact as an "international borrower of last resort" (not a lender, as defended by Kindleberger, 1973), and, was "hostage to international cooperation, reduced to dependence on the assistance of European central banks" (Eichengreen, 1992, p.8). During the interwar period, international cooperation collapsed, provoking the disappearance of one of the pillars of the pre-war GS. Eichengreen (1992) assesses this imperfect GS (Subsequently, the Gold Exchange Standard, GES), as being at the origin of the propagation

of the Great Depression worldwide. Capital flows were the *vector* of this crisis: "The asymmetry in the GS system under which countries in surplus can shift the burden of adjustment to countries in deficit, forcing them to deflate, was the last thing needed […] Monetary authorities outside the US were forced to respond vigorously to the decline in capital inflows if they wished to stay on the GS (p. 15)". Due to the commitment to gold, monetary and fiscal policies in the world remained restrictive, aggravating the contractionary effects on economic activity. Eichengreen highlights the fact that "governments hazarding expansionary initiatives were forced to draw back (p. 16)" (Britain in 1930; the US in 1931-33; Belgium in 1934). The trade-off was whether to defend the GES, or to renew with international cooperation to implement expansionary policies in the world. Under the GES, the lack of international cooperation precluded these initiatives. What "amplified this destabilizing impulse […] and gave rise to the great economic contraction? The answer lies in *the spread of financial instability* […] the bank failures and financial chaos that led to the liquidation of bank deposits (p. 18)". "Why didn't policy makers intervene to head off the collapse of their financial systems? They failed to do so because the GES posed an insurmountable obstacle to unilateral action. Containing bank runs required policy makers to inject liquidity into the banking system, but this could be inconsistent with the GS rules (p. 18)". After that, "realizing that convertibility might be compromised and that devaluation might cause capital losses on domestic assets, investors rushed to get their money out of the country […] the destabilizing linkages between domestic and international financial systems operated most powerfully where foreign deposits were more prevalent: Europe's banking systems were interconnected by a network of foreign deposits (p. 18)". *This is the vehicle through which contagion comes into play in Eichengreen's analysis*. European countries illustrate these mechanisms: disturbing revelations about the cover ratio (of gold reserves to notes and coins) in countries like Germany and Austria, accelerated capital flight and favored foreign deposit withdrawals. "Far from being a bulwark of financial stability, the GS was the main impediment to its maintenance (p. 19)". Domestic authorities could not fund the banking system without jeopardizing the GS rules.

Saving banks required international cooperation, which never materialized. This is why the author first designates the GES as the main cause of the Great Depression and then goes on, in a striking formula, to *assimilate the end of the Depression with the end of the GES* (Eichengreen (1992, p. 21)). Ultimately, unlike Kindleberger (1973) and Nurkse (1944), he asserted that countries that left Gold experienced economic recovery, whereas those remaining on Gold exacerbated their economic situation. Accordingly, "breaking the Golden Fetters" constituted the solution.

Our purpose here is to focus on the implications of contagion raised by the "Golden Fetters" hypothesis. Following Eichengreen (1992), Bordo and Murshid (2001) identify and retain one major contagion channel under the imperfect Gold Exchange Standard (GES), explicitly gold fluxes between countries and capital flights. They contend that their findings on contagion corroborate Eichengreen's "Golden Fetters" hypothesis. Their explanation relies on the absence of coordination between central banks during the GES, in which case the defense of parity should have implied a copycat of central banks in the use of interest rate. In order to respect par and without cooperation between Central Banks, each central bank had to monitor its domestic metallic holdings. In that respect, the best tool was the use of the discount rate. To control for this consequence, each central bank had no choice but to base its own discount rate on that of the others. In the absence of cooperation over the GES, the will to preserve metallic holdings in order to respect the gold par should have meant that each central bank had to adopt a follow-the-herd attitude. However, in case of currency attacks, if there had been efficient coordination, this would have led the central bank with a weak currency to raise its interest rate, while the central bank with a strong currency would have lowered its interest rate.

It is this specific link between the international contagion of interest rates and the "Golden Fetters" hypothesis that we want to test in the present paper. If the "Golden Fetters" hypothesis holds, then interest rate contagion should have been more pronounced under the GES than when it collapsed. This is because as soon as the GES was abandoned, each country was free to

pursue its own domestic goals, resulting in far more disconnected interest rates. Was this the case? This is the key historical issue we address in our analysis of the international contagion of short-term interest rates during the interwar period.

To this end, we have chosen to consider the three implications of Eichengreen's "Golden Fetters" thesis as sub-hypotheses, in order to assess their validity:

1) H1: "The financial crisis has been imported into the US" (Eichengreen, 1992). The BEKK model with a structural break will enable this issue to be decided.

2) H2: "France caused the disruption of the International Monetary System in the thirties" (Eichengreen, (1992)). Comparison of the two sub-periods (twenties versus thirties) will help to reveal whether or not contagion moved from France to other countries.

3) H3: "The GES was responsible for contagion" (which is *stricto sensu* the "Golden Fetters" hypothesis): The origin of the 1929 crisis is considered as indissociable from the unsustainable international monetary system (Gold Exchange Standard), with its presumed complete absence of central bank cooperation. Theoretically, such a lack of cooperation should have led to a stronger copycat policy of instrument rates while the GES was in operation, rather than after its breakdown. This was because, throughout the whole GES period, lack of cooperation resulted in competition for gold species between issuing institutions; this, in turn, led each central bank to copy its rate on that of its "partners/competitors". After the breakdown of the GES, each central bank was assumed to act independently, so interest rate contagion should have been less patent. We propose to test the validity of H3 through two indicators derived from our BEKK model:

a) We test contagion with five distinct structural breaks[4]: the triggering of the financial crisis (end of 1929), September 1931 (devaluation of the Pound), March 1933 (suspension of the Dollar convertibility), June 1933 (London conference), January 1934 (official

[4] These breaks are the most significant ones over the considered period. For reasons of space, tests that enable the presence of structural breaks in 1931, 1933, 1934 to be rejected, are not presented here, but are available upon request. It should be noted that for these dates, the BEKK model never converges, which means that these dates cannot be considered as indicating relevant structural breaks.

devaluation of the Dollar). Our purpose is to identify which structural break constitutes the defining moment of the period. Finding that one of the dates corresponding to the breakdown of the GES is more statistically significant than the financial crisis of 1929 would, undoubtedly, support the "Golden Fetters" hypothesis (H3). We contend, however, that an additional condition is required.

b) By counting the number of scenarios of absence of contagion during the two sub-periods, we are able to characterize the plausibility of Eichengreen's statement: if there were more scenarios of non-contagion in the thirties than in the twenties, H3 would be corroborated; conversely, more scenarios of non-contagion in the twenties than in the thirties would weaken H3.

4. Data and methodology

The data used here refer to the 3-month Treasury Bond interest rates in the USA, Great Britain and France, based on a monthly frequency. The considered period goes from 1921M01 to 1936M12 (Figure 1). The database has been elaborated by Pierre Villa.

[Insert Figure 1]

The choice of these data is driven by the two following considerations.

First, the three-month government bond yields incorporate the effects of monetary policy: by means of open market policies, central banks could purchase or sell Treasury bonds in function of their particular objectives.

Second, our choice of the three countries is based on the outcomes of Accominotti (2011), who indicates that the crisis that came from debtor countries was provoked by creditor countries repatriating their capital home. In the present article, we further assess the role played in the contagion phenomena by the three main creditor countries' use of strictly similar short-run interest rates.

When considering the possible specifications required to model the co-movements of spreads, we finally decided to adopt a BEKK model. We immediately discarded the VECH model of Bollerslev et al. (1988) as it was extremely unwieldy (more than 70 coefficients to evaluate in a trivariate framework); moreover, that model could generate time series on conditional variances featuring negative values. We also rejected the DVECH model (Diagonal VECH) which imposes prior restrictions on the structure of coefficient matrices: the conditional variance of the interest rate of a given country is dependent on its own past values and the innovation square related to them. The number of coefficients to be evaluated is certainly considerably reduced, thereby making estimation easier, but it then becomes impossible to test the reality of many schemes of influence between the volatilities of various variables, precisely because that model imposes its own pre-defined scheme. As for the dynamic correlations DCC scheme (Engle (2002) and Tse and Tsui (2002)), this is not suitable for tests on hypotheses relating to propagation phenomena because, like the VECH model, it does not guarantee the positivity of the values calculated from conditional variances.

Finally, the BEKK model was chosen, because (i) it is the only model that can test the hypothesis of the appropriate propagation scheme of volatilities, (ii) without having to estimate too many coefficients, (iii) while guaranteeing the positivity of the values calculated from conditional variances. However, as we want to test H3 by identifying the defining moment of the period, we introduce a structural break into the model.

Writing conventions for propagation schemes of volatilities and modeling.

A variety of propagation schemes based on the monthly levels of the 3-month interest rates (Y1, Y2, Y3) and, above all, on their volatilities, can be found in the 3 zones (USA, Great Britain and France). A scatter plot analysis confirms that a positive correlation prevails between the 3-month interest rates (Figure 2).

[Insert Figure 2]

As the unit root tests implemented on the monthly series systematically confirm level stationarity (Table 1), we have retained monthly levels of interest rates.

[Insert Table 1]

Consequently, the implemented BEKK model will focus on the dynamics of the monthly levels of interest rates and their associated second-order moments.

The BEKK model enables the simultaneous modeling of conditional expectations, variances and covariances of the short-run interest rates. It allows for a fairly easy testing of different propagation schemes of volatility between the three zones and, unlike the standard VECH model, the estimated coefficients provide, in all circumstances, positive conditional variances.

The unrestricted reference model M1 explicitly allows for volatility propagation schemes in all directions between the three zones. The model consists of two systems of equations, S1 and S2. Conditional expectations, variances and covariances are specified as:

$$\text{S1} \qquad \mathbf{Y_t = \Delta_t \alpha + [I - \Delta_t] \beta + \phi Y_{t-1} + \varepsilon_t}$$

$$\text{S2} \qquad \mathbf{H_t = C' \Delta_t C + D' [I - \Delta_t] D + A' \varepsilon_t \Delta_t \varepsilon_t' A + B' \varepsilon_t [I - \Delta_t] \varepsilon_t' B + G' H_{t-1} \Delta_t G + F' H_{t-1} [I - \Delta_t] F}$$

where Δ_t is an identity matrix from the date of the structural break and a null matrix before this date, and **I** is the identity matrix of dimension 3. The matrices $\boldsymbol{\alpha}$, **C**, **A** and **G** (respectively $\boldsymbol{\beta}$, **D**, **B** and **F**) are the matrices of prevailing coefficients after (respectively before) the date of the structural break. The restrictions made on the matrix components $\boldsymbol{\beta}$, **D**, **B** and **F,** when we move from the propagation scheme « RRR » to another scheme, can easily be deduced from the restrictions mentioned in a non-exhaustive way in Appendix 1.

The first system, S1, depicts the conditional expectations of the short-run interest rates Y_{jt} ($j \in \{1, 2, 3\}$). For the sake of simplicity, each equation has been indicated in an AR(1) form.

As for the S2 system, it models the 3 conditional variances and the 3 conditional covariances $h_{ij,t}$ where $(i, j) \in \{1, 2, 3\}$).

It should be noted that the $\mathbf{H}_t$ matrix of conditional variances and covariances is symmetric, which is not the case, however, of matrices **C**, **A** and **G (**respectively **D**, **B** and **F**). The g_{ij} and f_{ij} coefficients (resp. a_{ij} and b_{ij}) determine the degree of dependence of the conditional variance $h_{jj,\,t}$ of Y_j on date t toward the lagged conditional variance $Y_{ii,\,t-1}$ of Y_i (resp. toward the lagged squared innovation $\varepsilon_{i,t-1}^2$). Consequently, a restriction of nullity on these coefficients ($a_{ij} = b_{ij} = g_{ij} = f_{ij} = 0$) suggests the absence of propagation of the volatility from i toward j. Many diffusion schemes can be considered between conditional volatilities, with each being bound up with particular restrictions on some coefficients of matrices **A**, **G**, **B** and **F**. Some prior conventions are useful to describe these diffusion schemes. To begin with, on the basis of the three monetary zones (1 = USA, 2 = Great Britain, 3 = France) three pairwise relationship can be observed (1, 2), (1, 3) and (2, 3). These pairwise relationships can take four alternate forms:

a) Total absence of propagation between the two zones, a scheme indicated by the letter N (No contagion). N(1,2) depicts the absence of any volatility propagation between zone 1 and 2.
b) Reciprocal propagation, identified by the letter R[5].
c) Univocal propagation of the first component of the couple toward the second is indicated by the letter U[6].
d) Inverted univocal propagation of the second component of the couple toward the first one; the letter I identifies this scheme[7].

[5] R(1, 2) suggests a recursive scheme of propagation 1 ↔ 2.
[6] U(1, 2) means that there is one scheme of propagation between zones 1 and 2 working in the sense 1 →2.

The description of the global diffusion scheme between the three pairs (1, 2), (1, 3) and (2, 3) will take the form of a triplet, whose components are chosen from the set {N, R, U, I}. For instance, the total absence of diffusion mechanisms between any of the three monetary pairs is described by the triplet N(1, 2), N(1, 3), N(2, 3) or, in short, « NNN » if we admit that the first component of the triplet always refers to the couple (1,2), the second to the couple (1,3), and the third one to the couple (2, 3)[8]. Therefore, the model of reference M1 is also, according to these conventions, the model « RRR ». Each possible restriction of the model RRR relates to a specific propagation scheme, and has been tested using a Wald test.

The set of possible schemes is mentioned in the appendix (A1); it sums up, for each scheme, the corresponding restrictions on the components of the initial matrices **A**, **B**, **G** and **F**.

Calibration and coefficient estimations

The « RRR » model is estimated using the method of maximum likelihood. Assuming normality of the joint distribution ε_{1t}, ε_{2t} and ε_{3t}, the likelihood of the t^{th} observation for a set of coefficient $\Theta = \{\alpha, \Phi, \mathbf{C}, D, A, B, G \text{ and } F\}$ is:

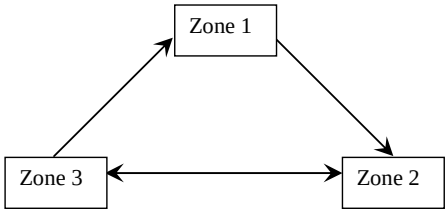
$$L_t = \frac{1}{2\pi}\left|H_t\right|^{-1/2} \exp^{-\hat{\varepsilon}_t' H_t^{-1} \hat{\varepsilon}_t}$$

Where $|H_t|$ is the determinant of $\mathbf{H}_t$ and $\hat{\varepsilon}_t = \begin{bmatrix} \hat{\varepsilon}_{1,t} \\ \hat{\varepsilon}_{2,t} \\ \hat{\varepsilon}_{2,t} \end{bmatrix}$ is the residuals matrix stemming from ε.

The log-likelihood of the whole sample is then:

[7] I(1, 2) enables a 1 ← 2-type of diffusion scheme to be to identified.

[8] For example, UIR suggests the existence of a scheme such as:



$$\text{LogL} = \sum_{t=1}^{T}\left(-\frac{1}{2}\text{Log}\left(\left|H_t\right|\right)-\text{Log}\left(2\pi\right)-\hat{\varepsilon}_t\,'\,H_t^{-1}\hat{\varepsilon}_t\right)$$

The numerical resolution of the optimization problem needs an appropriate choice of the first guests of the coefficients. To that end, we start by evaluating a univariate GARCH model for each of the three variations Y_{jt} ($j \in \{1, 2, 3\}$) of interest rates. On the basis of the resulting estimated coefficients and residuals, the *first guests* for matrices $\boldsymbol{\alpha}$, $\boldsymbol{\Phi}$, **C**, D, **A, B**, F and **G** are calibrated[9].

In the same way, the conditional covariance time series are initially set as:

$$h_{ij,\,t} = \text{Cov}(\hat{\varepsilon}_{i,\,t},\, \hat{\varepsilon}_{j,\,t})\ \forall\ i, j, t$$

while the initial conditional variances time series of the BEKK model correspond to their estimated counterpart in the univariate models.

5. Findings

The unrestricted model RRR is evaluated for the whole period. The structural break introduced in the BEKK model has been selected by using the maximum of likelihood among five candidates: contrary to the hypothesis of Accominotti (2011) and Eichengreen (1992), the triggering of the financial crisis of 1929 surpasses by far the other four dates, which correspond to the breakdown of the GES. None of these other four dates, which characterize the 1931-1934 "Golden Fetters" hypothesis, gives significant results regarding contagion schemes.

Estimation of the possible schemes of contagion is presented in Table 2.

[Insert Table 2]

[9] Calibration of the initial coefficient values is here only described for the case of the system with no structural break. Calibration of the model with a structural break does not present, conceptually, any additional difficulties except for the fact that it requires, even for the same spread, the estimation of two univariate Garch models for each of the two sub-periods separated by the date-event of 1929m11.

In Table 3, we have ranked restrictions of M1 model via decremental values of type I errors: the most acceptable restrictions appear at the top of the list. We found 10 scenarios of possible contagion schemes, with a probability of occurrence superior to 80%. We have also decided to reject 18 scenarios with a probability of occurrence inferior to 15%.

In order to select the most probable scheme of contagion, Table 3 recapitulates the number of scenarios whose probability of occurrence is higher than 80% or inferior to 15%. For each pair of countries, we retain (respectively, we reject) the scheme observed the largest number of times, with a probability superior to 80% (respectively, inferior to 15%).

[Insert Table 3]

We obtain the following results:

- an inverted propagation process between the 3-month interest rates of the USA and France
- an inverted or reciprocal propagation process between the USA and Great Britain
- an inverted propagation process between France and Great Britain

Moreover, according to Table 3, we can reject the total absence of propagation between the zones throughout the whole period.

Having determined that the structural break occurred in November 1929, we distinguish two sub-periods, in order to compare the dynamics of the relationship between interest rates before and after this structural break. The first sub-period corresponds, then, to the 1920s, and the second to the 1930s. This allows us to analyze the extent to which Black Thursday has modified the propagation schemes of interest rates.

The ranked restrictions of M1 model (Table 4) offer quite different results than those obtained for the reference period.

[Insert Table 4]

At the 80% level, 42 contagion schemes could be the good candidates and, unlike the whole period, we cannot reject any propagation scheme at a 15% level.

Table 5 indicates that the most probable scheme of propagation is the following: a reciprocal propagation process from the 3-month interest rate of the USA to the 3-month interest rate of Great Britain, and reciprocal propagation between the interest rates of the USA and France and also between France and Great Britain.

[Insert Table 5]

The ranked restrictions of M1 model for the 1930m06-1936m1 period (Table 6) indicate that 6 scenarios of possible contagion schemes with a probability of superior to 80% occur, and that 27 schemes with a probability of occurrence inferior to 15% should be rejected.

[Insert Table 6]

Table 7 indicates that the following scheme may well prevail during the second sub-period:

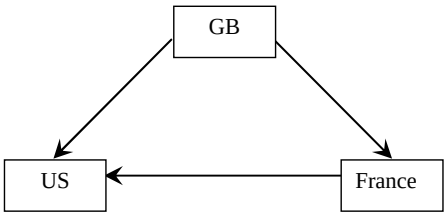
- there is a propagation scheme from France to Great Britain
- there is reciprocal propagation between the 3-month interest rates of Great Britain and the USA
- There is a univocal propagation scheme between the 3-month interest rates of France and the USA.

[Insert Table 7]

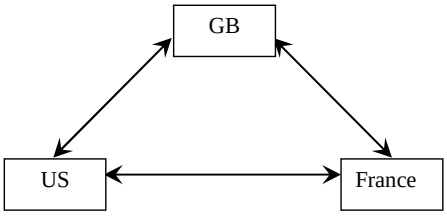
To summarize our results, we can reasonably assume that the following contagion scheme prevailed throughout the interwar period and its associated sub-periods (Figure 3).

Figure 3: The most probable schemes of propagation

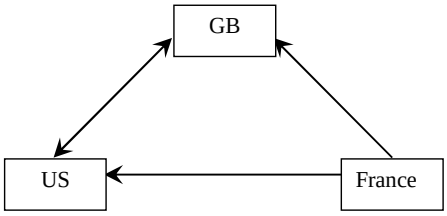
(a) The most probable scheme of contagion throughout the interwar period



(b) The most probable scheme of contagion throughout the 1920s



(c) The most probable scheme of contagion throughout the 1930s



6. Discussion

Our results shed new light on the "Golden Fetters" thesis. We recall here that the aim of the present article was to test the implications of the "Golden Fetters" hypothesis as three sub-hypotheses, in order to assess their validity:

1) Regarding H1 (proposal: "the financial crisis has been imported into the US", Eichengreen, 1992), the BEKK model with structural break (see Figure 3) corroborates Eichengreen's view: the US suffered from the contagion that originated in Europe. We find that Great Britain and France were at the origin of changes in the volatility of US interest rates. We can not, therefore, reject the idea that the financial crisis was imported into the US.

2) H2: (proposal: "France caused the disruption of the International Monetary System in the thirties", Eichengreen, 1992). Comparison of the two sub-periods (twenties versus thirties)

indicates contrasted dynamics of contagion. By retaining the real propagation mechanisms at the 1% error level, the twenties seem marked by perfect interaction between the three countries: over this period, recursion (mutual contagion) stands out as dominant. Conversely, in the thirties, the contagion moves from France both to the UK and to the US. The volatility of French interest rates controls both the UK and the US rates: At issue, according to Eichengreen (1992), the "policy of species" of the Bank of France, which drained the gold species in order to maintain the gold parity of its currency. Our econometric results tend to support hypothesis (H2): we can not reject the possibility that, in the thirties, the sources of interference between interest rates might have come from the "mercantilist" policy of the Bank of France. Nonetheless, this finding should not be over-interpreted. It is, admittedly, one of the most probable schemes of contagion, but not the only one. We have also detected the presence of reciprocal propagation between GB and the USA.

3) H3: Was the GES ultimately responsible for contagion? This strong claim, advanced by Eichengreen (1992), implies that the origin of the 1929 crisis lies in an unsustainable international monetary system (Gold Exchange Standard), characterized by the presumed complete absence of central bank cooperation: this lack of cooperation should, logically, lead to a stronger copycat policy of instrument rates under the GES than after its break.

a) By mobilizing the BEKK model with a structural break, we first determined which date revealed the most significant structural break. We tested contagion mechanisms with five plausibly distinct structural breaks: the triggering of the financial crisis of 1929, September 1931 (devaluation of the Pound), March 1933 (suspension of the Dollar convertibility), June 1933 (London conference), January 1934 (official devaluation of the Dollar). Using the method of maximum likelihood to order our results, we have found that only 1929 marked a significant break in the transmission mechanism of interest rate volatility. The change in the transmission of shocks of

interest rate volatility appears at the end of the year 1929, clearly as a result of the financial crisis, not of the progressive collapse of the GES. This result undermines the thesis of the "Golden Fetters", because none of the key dates of the breakdown of the GES appears to be significant here.

b) We then counted the number of scenarios of absence of contagion in the two sub-periods: under the hypothesis of non-cooperation between central banks over the GES period, as explained above, the transmission of interest rate volatility should be strong (with each central bank mapping its interest rates on its "partners/competitors"). Yet, we have found that the scenario of no link between interest rates (complete absence of contagion) was rejected 30 times during the period 1930 to 1936, while we can not reject it for the period 1921-1929. This means that the absence of contagion is more characteristic of the twenties than the thirties, which is in contradiction with H3. We have also found that the scenario of absence of contagion could be accepted (with a probability of 80%) for 18 episodes over the twenties, against only two episodes over the thirties. Both the rejection and acceptance of scenarios of absence of contagion indicate that H3 should be reconsidered.

Conclusion: Does the "Golden Fetters" hypothesis hold?

Our study shows somewhat contrasting results for the effects of contagion effects during the GES period. Our outcomes suggest that the disappearance of the GES did not have the effects implicitly assumed by Eichengreen (1992), and that the absence of contagion was more probable in the twenties than in the thirties. If one follows the conclusions of Eichengreen (1992), then contagion due to the GES should have been stronger in the twenties than in the thirties. We

provide empirical evidence here that goes against this scenario. We show that, in the mechanism of short-run interest rate contagion, the key date was the triggering of the financial crisis, not the steps leading to the collapse of the GES. However, the other two propositions drawn from the work of Eichengreen (1992) resist our analysis: the assumption of the financial crisis being imported in the US can not be ruled out; a possible French source in the transmission of short run interest rate shocks can not be ignored, but it is not the only source. Ultimately, it emerges from our results that two of the scenarios proposed by Eichengreen (1992) are possible (H1 and H2, although not entirely in the case of H2), with the third one (H3) remaining far less plausible.

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Figure 1: The 3-month interest rates of France, Great Britain and the USA (1921M01-1936M12)

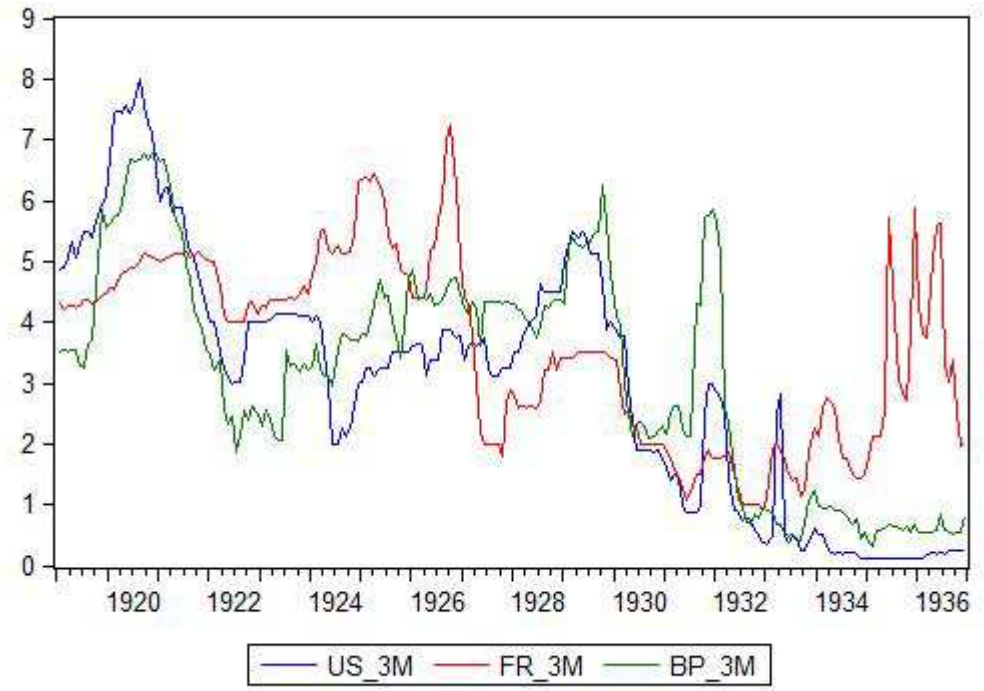

Figure 2: The correlation between the 3-month interest rates of France, Great Britain and the USA (1921M01-1936M12)

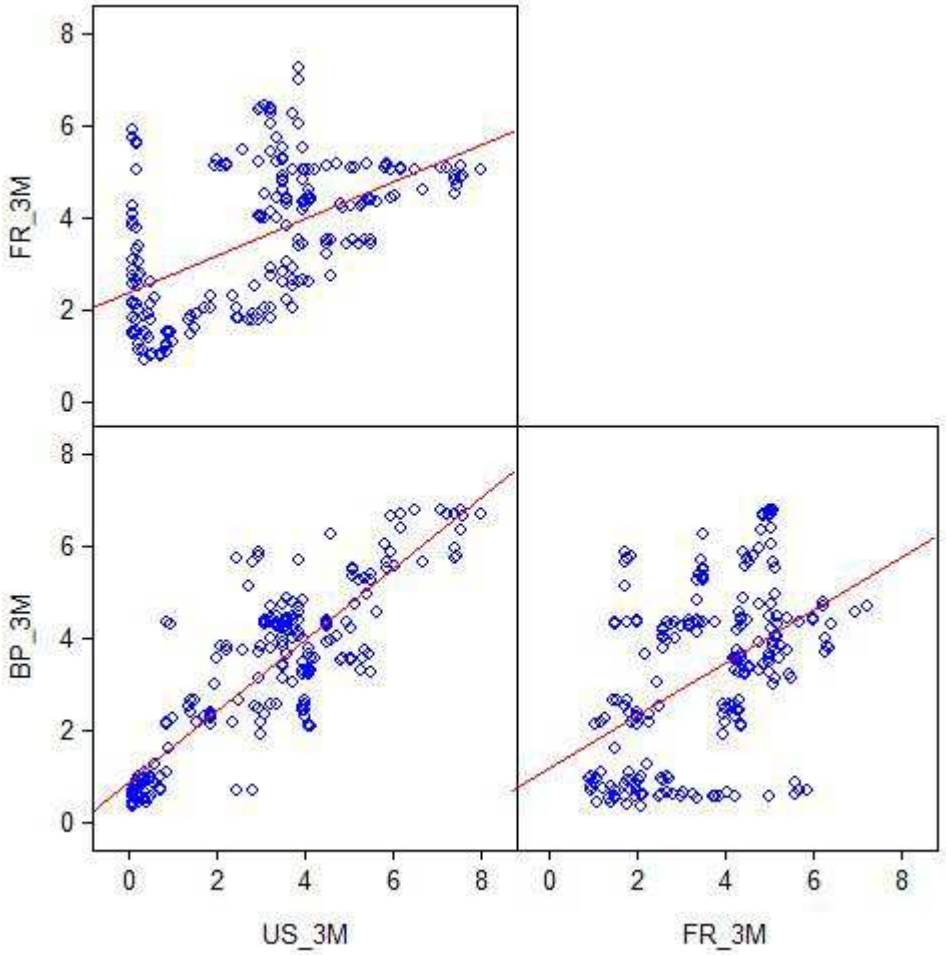

Table 1. Unit-root tests

	3-month interest rates			
Period: 1921M01-1936M12	1921m01-1929m06		1930m06-1936m12	
	t-Statistic	Prob.	t-Statistic	Prob.
Im, Pesaran and Shin W-stat	-1.78	0.04	-2.09	0.01

The number of lags is automatically determined on the basis of the Min(SIC) criterion. The Prob. column indicates the risk threshold from which it becomes possible to reject H0.

Table 2. Propagation schemes of volatilities for 3-month interest rates (1921m01-1936m12) Break on 1929M11: Wald restriction tests

1–2USA↔ FRA	1–3 USA↔GB	2–3 FRA↔GB	Wald Stat	% P-val
R	R	I	0.493137	97.42
I	R	U	2.434867	96.47
I	I	I	4.871141	96.21
R	R	U	0.631324	95.95
I	I	U	5.023761	95.72
I	R	I	3.165346	92.36
I	I	R	3.682484	88.46
R	I	I	3.856801	86.98
I	R	R	1.296504	86.2
R	I	U	4.498451	80.96
I	U	R	5.639672	68.75
R	R	N	5.783262	67.15
I	U	U	9.838523	63.01
R	I	R	3.016483	55.51
R	U	U	7.175579	51.78
R	U	I	7.669387	46.64
N	I	R	11.79841	46.2
I	N	R	12.2358	42.69
U	U	I	12.37814	41.58
N	I	I	16.64276	40.91
R	U	R	4.085821	39.45
I	U	I	12.67004	39.35
U	R	R	4.173033	38.31
U	R	I	8.74197	36.45
R	N	R	8.877864	35.27
N	R	R	9.061661	33.71
I	N	I	17.87961	33.1
U	U	N	18.38672	30.17
U	I	R	9.532098	29.94
U	R	U	9.537282	29.9
U	R	N	14.07094	29.62
R	N	I	14.15778	29.07
I	N	U	18.72683	28.31
R	N	U	14.28863	28.27
R	U	N	14.30124	28.19
U	I	I	14.30569	28.16
N	R	I	14.37414	27.75
N	N	I	23.24544	27.69
N	U	I	19.44487	24.63
U	U	R	10.40044	23.8
N	N	R	19.8765	22.58
U	U	U	15.30949	22.49
U	N	I	20.39068	20.31

N	U	R	16.1511	18.44
U	N	R	16.92651	15.24
N	I	U	22.33523	13.27
N	R	U	17.49013	13.21
N	U	U	24.99994	6.98
U	I	U	20.54314	5.75
I	I	N	27.06526	4.08
N	N	U	32.75582	3.59
I	R	N	22.34018	3.39
I	U	N	29.80802	1.9
U	N	U	29.89013	1.86
R	I	N	24.74377	1.61
N	U	N	36.46109	1.36
N	R	N	31.84416	1.05
N	I	N	39.32954	0.61
U	I	N	37.46111	0.18
I	N	N	44.15522	0.14
N	N	N	50.93498	0.11
R	N	N	40.9662	0.06
U	N	N	49.05617	0.03

Table 3. Selection of propagation schemes based on Wald restriction tests (1921m01-1936m12) - Break on 1929M11

Rejected schemes at a 15% level

	1–2USA↔ FRA	1–3 USA↔GB	2–3 FRA↔GB	Total
R	2	3	0	5
U	4	3	5	12
I	4	5	0	9
N	7	6	12	25

Most probable schemes (P-value >80%)

	1–2USA↔ FRA	1–3 USA↔GB	2–3 FRA↔GB	Total
R	3	5	2	10
U	0	0	3	3
I	6	4	4	14
N	0	0	0	0

Table 4. Propagation schemes of volatilities for 3-month interest rates (1921m01-1929m06): Wald restriction tests

1–2USA↔ GB	1–3 USA↔FRA	2–3 GB↔FRA	Wald Stat	% P-val
I	N	R	0.10	100.00
N	I	R	0.18	99.99
I	I	U	0.17	99.99
N	I	I	0.57	99.98
I	U	R	0.06	99.96
I	I	R	0.06	99.96
R	N	R	0.09	99.91
N	R	I	0.46	99.83
U	I	I	0.47	99.82
I	I	I	0.47	99.82
I	R	U	0.13	99.80
N	R	R	0.14	99.75
U	U	R	0.15	99.75
U	I	R	0.16	99.71
R	I	U	0.16	99.70
U	U	I	0.57	99.68
U	R	U	0.21	99.51
U	R	N	0.70	99.46
R	U	R	0.01	99.31
I	R	R	0.02	99.17
I	N	U	1.57	99.14
R	I	R	0.04	98.22
I	R	I	0.41	98.20
R	R	U	0.04	98.02
R	I	I	0.43	98.01
U	R	I	0.45	97.78
R	U	I	0.51	97.25
I	U	U	1.42	96.49
R	N	U	1.43	96.37
R	R	N	0.62	96.04
U	U	U	1.55	95.59
U	R	R	0.11	94.85
U	N	R	1.80	93.73
U	N	U	3.02	93.28
U	N	I	3.62	88.99
U	I	U	2.49	86.98
R	U	U	1.33	85.55
N	N	U	5.58	84.94
N	N	R	4.27	83.17
I	U	I	2.91	82.02
R	R	I	0.40	81.95
I	N	I	4.43	81.64
N	N	I	6.45	77.64

R	N	I	3.33	76.70
U	U	N	5.04	75.36
N	U	U	5.29	72.65
N	I	U	5.36	71.80
N	U	I	5.38	71.68
R	U	N	3.73	71.32
N	U	R	4.07	66.79
U	I	N	5.98	64.95
N	R	U	5.01	54.22
I	R	N	5.26	51.12
U	N	N	9.33	50.09
R	I	N	5.76	45.06
I	I	N	7.83	44.99
N	N	N	12.04	44.29
N	U	N	10.15	42.77
N	I	N	10.18	42.45
N	R	N	8.60	37.74
I	U	N	8.64	37.38
I	N	N	10.83	37.10
R	N	N	9.08	33.52

Table 5. Selection of propagation schemes based on Wald restriction tests (1921m01-1929m06)

Rejected schemes at a 15% level

	1–2USA↔ GB	1–3 USA↔FRA	2–3 GB↔FRA	Total
R	0	0	0	0
U	0	0	0	0
I	0	0	0	0
N	0	0	0	0

Most probable schemes (P-value >80%)

	1–2USA↔ GB	1–3 USA↔FRA	2–3 GB↔FRA	Total
R	11	12	14	37
U	13	9	13	35
I	12	11	13	46
N	6	10	2	18

Table 6. Propagation schemes of volatilities for 3-month interest rates (1930m06-1936m12): Wald restriction tests

1–2GB↔ FRA	1–3 GB↔US	2–3 FRA↔US	Wald Stat	% P-val
R	R	N	0.087257	99.91
R	R	I	0.05316	97.38
R	R	U	0.053929	97.34
I	R	N	1.779036	93.89
I	R	I	1.297166	86.19
I	R	U	1.541508	81.93
N	R	U	3.417805	75.49
R	U	I	1.972047	74.09
N	R	N	5.526478	70.01
I	I	U	4.098324	66.34
R	I	U	2.496221	64.53
R	U	N	4.255086	64.22
U	R	N	4.453052	61.56
I	R	R	1.099971	57.7
U	R	U	2.995865	55.85
R	U	U	3.225842	52.08
N	R	R	3.39723	49.37
N	R	I	5.43646	48.92
I	I	R	3.575559	46.65
I	U	N	8.054405	42.82
R	U	R	1.859954	39.46
R	N	R	4.252468	37.29
U	R	I	4.424572	35.16
U	U	I	6.836071	33.63
R	I	R	2.227796	32.83
U	U	N	9.742889	28.35
I	U	I	7.677891	26.27
I	U	U	7.731055	25.85
N	U	N	12.52656	25.14
N	U	I	10.35847	24.08
N	I	U	10.40154	23.8
U	R	R	2.994214	22.38
U	U	R	5.861161	20.98
R	N	U	8.484417	20.47
N	U	U	11.56852	17.15
U	U	U	9.163999	16.46
N	U	R	9.81462	13.27
I	N	U	12.54106	12.86
U	I	U	10.08558	12.11
I	U	R	7.45553	11.37
N	I	R	10.38553	10.93
N	N	U	17.67896	6.06
I	N	R	12.06443	6.05

U	N	R	12.22209	5.72
N	N	R	15.91356	4.36
U	N	U	16.10408	4.09
U	I	R	10.0646	3.94
R	N	I	14.16441	2.79
R	I	N	14.47251	2.48
I	I	N	19.09318	1.44
R	I	I	13.02208	1.12
N	I	N	23.70769	0.84
I	I	I	17.52413	0.75
R	N	N	21.34454	0.63
U	I	N	22.60246	0.39
U	N	I	22.80762	0.36
N	I	I	23.4974	0.28
N	N	N	31.86781	0.15
U	I	I	21.82563	0.13
U	N	N	29.01136	0.12
I	N	N	29.06539	0.12
N	N	I	31.31883	0.05
I	N	I	28.92165	0.03

Table 7. Selection of propagation schemes based on Wald restriction tests (1930m06-1936m12)

Rejected schemes at a 15% level

	1–2GB↔ FRA	1–3 GB↔US	2–3 FRA↔US	Total
R	4	0	7	11
U	8	2	4	14
I	7	11	8	26
N	8	14	8	30

Most probable schemes (P-value >80%)

	1–2GB↔ FRA	1–3 GB↔US	2–3 FRA↔US	Total
R	3	6	0	9
U	0	0	2	2
I	3	0	2	5
N	0	0	2	2

Appendix 1. Propagation schemes of interest rate volatilities, and corresponding restrictions on A, B, G and F components (BEKK model with structural break)

Scheme	Corresponding restrictions
RRR	No restriction
III	A1(2) = A1(3) = A2(3) = G1(2) = G1(3) = G2(3) = B1(2) = B1(3) = B2(3) = F1(2) = F1(3) = F2(3) = 0
IIN	A1(2) = A1(3) = A2(3) = A3(2) = G1(2) = G1(3) = G2(3) = G3(2) = B1(2) = B1(3) = B2(3) = B3(2) = F1(2) = F1(3) = F2(3) = F3(2) = 0
IIR	A1(2) = A1(3) = G1(2) = G1(3) = B1(2) = B1(3) = F1(2) = F1(3) = 0
IIU	A1(2) = A1(3) = A3(2) = G1(2) = G1(3) = G3(2) = B1(2) = B1(3) = B3(2) = F1(2) = F1(3) = F3(2) = 0
INI	A1(2) = A1(3) = A3(1) = A2(3) = G1(2) = G1(3) = G3(1) = G2(3) = B1(2) = B1(3) = B3(1) = B2(3) = F1(2) = F1(3) = F3(1) = F2(3) = 0
INN	A1(2) = A1(3) = A3(1) = A2(3) = A3(2) = G1(2) = G1(3) = G3(1) = G2(3) = G3(2) = B1(2) = B1(3) = B3(1) = B2(3) = B3(2) = F1(2) = F1(3) = F3(1) = F2(3) = F3(2) = 0
INR	A1(2) = A1(3) = A3(1) = G1(2) = G1(3) = G3(1) = B1(2) = B1(3) = B3(1) = F1(2) = F1(3) = F3(1) = 0
INU	A1(2) = A1(3) = A3(1) = A3(2) = G1(2) = G1(3) = G3(1) = G3(2) = B1(2) = B1(3) = B3(1) = B3(2) = F1(2) = F1(3) = F3(1) = F3(2) = 0
IRI	A1(2) = A2(3) = G1(2) = G2(3) = B1(2) = B2(3) = F1(2) = F2(3) = 0
IRN	A1(2) = A2(3) = A3(2) = G1(2) = G2(3) = G3(2) = B1(2) = B2(3) = B3(2) = F1(2) = F2(3) = F3(2) = 0
IRR	A1(2) = G1(2) = B1(2) = F1(2) = 0
IRU	A1(2) = A3(2) = G1(2) = G3(2) = B1(2) = B3(2) = F1(2) = F3(2) = 0
IUI	A1(2) = A3(1) = A2(3) = G1(2) = G3(1) = G2(3) = B1(2) = B3(1) = B2(3) = F1(2) = F3(1) = F2(3) = 0
IUN	A1(2) = A3(1) = A2(3) = A3(2) = G1(2) = G3(1) = G2(3) = G3(2) = B1(2) = B3(1) = B2(3) = B3(2) = F1(2) = F3(1) = F2(3) = F3(2) = 0
IUR	A1(2) = A3(1) = G1(2) = G3(1) = B1(2) = B3(1) = F1(2) = F3(1) = 0
IUU	A1(2) = A3(1) = A3(2) = G1(2) = G3(1) = G3(2) = B1(2) = B3(1) = B3(2) = F1(2) = F3(1) = F3(2) = 0
NII	A1(2) = A2(1) = A1(3) = A2(3) = G1(2) = G2(1) = G1(3) = G2(3) = B1(2) = B2(1) = B1(3) = B2(3) = F1(2) = F2(1) = F1(3) = F2(3) = 0
NIN	A1(2) = A2(1) = A1(3) = A2(3) = A3(2) = G1(2) = G2(1) = G1(3) = G2(3) = G3(2) = B1(2) = B2(1) = B1(3) = B2(3) = B3(2) = F1(2) = F2(1) = F1(3) = F2(3) = F3(2) = 0
NIR	A1(2) = A2(1) = A1(3) = G1(2) = G2(1) = G1(3) = B1(2) = B2(1) = B1(3) = F1(2) = F2(1) = F1(3) = 0
NIU	A1(2) = A2(1) = A1(3) = A3(2) = G1(2) = G2(1) = G1(3) = G3(2) = B1(2) = B2(1) = B1(3) = B3(2) = F1(2) = F2(1) = F1(3) = F3(2) = 0
NNI	A1(2) = A2(1) = A1(3) = A3(1) = A2(3) = G1(2) = G2(1) = G1(3) = G3(1) = G2(3) = B1(2) = B2(1) = B1(3) = B3(1) = B2(3) = F1(2) = F2(1) = F1(3) = F3(1) = F2(3) = 0
NNN	A1(2) = A2(1) = A1(3) = A3(1) = A2(3) = A3(2) = G1(2) = G2(1) = G1(3) = G3(1) = G2(3) = G3(2) = B1(2) = B2(1) = B1(3) = B3(1) = B2(3) = B3(2) = F1(2) = F2(1) = F1(3) = F3(1) = F2(3) = F3(2) = 0
NNR	A1(2) = A2(1) = A1(3) = A3(1) = G1(2) = G2(1) = G1(3) = G3(1) = B1(2) = B2(1) = B1(3) = B3(1) = F1(2) = F2(1) = F1(3) = F3(1) = 0
NNU	A1(2) = A2(1) = A1(3) = A3(1) = A3(2) = G1(2) = G2(1) = G1(3) = G3(1) = G3(2) = B1(2) = B2(1) = B1(3) = B3(1) = B3(2) = F1(2) = F2(1) = F1(3) = F3(1) = F3(2) = 0
NRI	A1(2) = A2(1) = A2(3) = G1(2) = G2(1) = G2(3) = B1(2) = B2(1) = B2(3) = F1(2) = F2(1) = F2(3) = 0
NRN	A1(2) = A2(1) = A2(3) = A3(2) = G1(2) = G2(1) = G2(3) = G3(2) = B1(2) = B2(1) = B2(3) = B3(2) = F1(2) = F2(1) = F2(3) = F3(2) = 0
NRR	A1(2) = A2(1) = G1(2) = G2(1) = B1(2) = B2(1) = F1(2) = F2(1) = 0
NRU	A1(2) = A2(1) = A3(2) = G1(2) = G2(1) = G3(2) = B1(2) = B2(1) = B3(2) = F1(2) = F2(1) = F3(2) = 0
NUI	A1(2) = A2(1) = A3(1) = A2(3) = G1(2) = G2(1) = G3(1) = G2(3) = B1(2) = B2(1) = B3(1) = B2(3) = F1(2) = F2(1) = F3(1) = F2(3) = 0
NUN	A1(2) = A2(1) = A3(1) = A2(3) = A3(2) = G1(2) = G2(1) = G3(1) = G2(3) = G3(2) = B1(2) = B2(1) = B3(1) = B2(3) = B3(2) = F1(2) = F2(1) = F3(1) = F2(3) = F3(2) = 0
NUR	A1(2) = A2(1) = A3(1) = G1(2) = G2(1) = G3(1) = B1(2) = B2(1) = B3(1) = F1(2) = F2(1) = F3(1) = 0
NUU	A1(2) = A2(1) = A3(1) = A3(2) = G1(2) = G2(1) = G3(1) = G3(2) = B1(2) = B2(1) = B3(1) = B3(2) = F1(2) = F2(1) = F3(1) = F3(2) = 0
RII	A1(3) = A2(3) = G1(3) = G2(3) = B1(3) = B2(3) = F1(3) = F2(3) = 0
RIN	A1(3) = A2(3) = A3(2) = G1(3) = G2(3) = G3(2) = B1(3) = B2(3) = B3(2) = F1(3) = F2(3) = F3(2) = 0
RIR	A1(3) = G1(3) = B1(3) = F1(3) = 0
RIU	A1(3) = A3(2) = G1(3) = G3(2) = B1(3) = B3(2) = F1(3) = F3(2) = 0
RNI	A1(3) = A3(1) = A2(3) = G1(3) = G3(1) = G2(3) = B1(3) = B3(1) = B2(3) = F1(3) = F3(1) = F2(3) = 0

RNN	A1(3) = A3(1) = A2(3) = A3(2) = G1(3) = G3(1) = G2(3) = G3(2) = B1(3) = B3(1) = B2(3) = B3(2) = F1(3) = F3(1) = F2(3) = F3(2) = 0
RNR	A1(3) = A3(1) = G1(3) = G3(1) = B1(3) = B3(1) = F1(3) = F3(1) = 0
RNU	A1(3) = A3(1) = A3(2) = G1(3) = G3(1) = G3(2) = B1(3) = B3(1) = B3(2) = F1(3) = F3(1) = F3(2) = 0
RRI	A2(3) = G2(3) = B2(3) = F2(3) = 0
RRN	A2(3) = A3(2) = G2(3) = G3(2) = B2(3) = B3(2) = F2(3) = F3(2) = 0
RRU	A3(2) = G3(2) = B3(2) = F3(2) = 0
RUI	A3(1) = A2(3) = G3(1) = G2(3) = B3(1) = B2(3) = F3(1) = F2(3) = 0
RUN	A3(1) = A2(3) = A3(2) = G3(1) = G2(3) = G3(2) = B3(1) = B2(3) = B3(2) = F3(1) = F2(3) = F3(2) = 0
RUR	A3(1) = G3(1) = B3(1) = F3(1) = 0
RUU	A3(1) = A3(2) = G3(1) = G3(2) = B3(1) = B3(2) = F3(1) = F3(2) = 0
UII	A2(1) = A1(3) = A2(3) = G2(1) = G1(3) = G2(3) = B2(1) = B1(3) = B2(3) = F2(1) = F1(3) = F2(3) = 0
UIN	A2(1) = A1(3) = A2(3) = A3(2) = G2(1) = G1(3) = G2(3) = G3(2) = B2(1) = B1(3) = B2(3) = B3(2) = F2(1) = F1(3) = F2(3) = F3(2) = 0
UIR	A2(1) = A1(3) = G2(1) = G1(3) = B2(1) = B1(3) = F2(1) = F1(3) = 0
UIU	A2(1) = A1(3) = A3(2) = G2(1) = G1(3) = G3(2) = B2(1) = B1(3) = B3(2) = F2(1) = F1(3) = F3(2) = 0
UNI	A2(1) = A1(3) = A3(1) = A2(3) = G2(1) = G1(3) = G3(1) = G2(3) = B2(1) = B1(3) = B3(1) = B2(3) = F2(1) = F1(3) = F3(1) = F2(3) = 0
UNN	A2(1) = A1(3) = A3(1) = A2(3) = A3(2) = G2(1) = G1(3) = G3(1) = G2(3) = G3(2) = B2(1) = B1(3) = B3(1) = B2(3) = B3(2) = F2(1) = F1(3) = F3(1) = F2(3) = F3(2) = 0
UNR	A2(1) = A1(3) = A3(1) = G2(1) = G1(3) = G3(1) = B2(1) = B1(3) = B3(1) = F2(1) = F1(3) = F3(1) = 0
UNU	A2(1) = A1(3) = A3(1) = A3(2) = G2(1) = G1(3) = G3(1) = G3(2) = B2(1) = B1(3) = B3(1) = B3(2) = F2(1) = F1(3) = F3(1) = F3(2) = 0
URI	A2(1) = A2(3) = G2(1) = G2(3) = B2(1) = B2(3) = F2(1) = F2(3) = 0
URN	A2(1) = A2(3) = A3(2) = G2(1) = G2(3) = G3(2) = B2(1) = B2(3) = B3(2) = F2(1) = F2(3) = F3(2) = 0
URR	A2(1) = G2(1) = B2(1) = F2(1) = 0
URU	A2(1) = A3(2) = G2(1) = G3(2) = B2(1) = B3(2) = F2(1) = F3(2) = 0
UUI	A2(1) = A3(1) = A2(3) = G2(1) = G3(1) = G2(3) = B2(1) = B3(1) = B2(3) = F2(1) = F3(1) = F2(3) = 0
UUN	A2(1) = A3(1) = A2(3) = A3(2) = G2(1) = G3(1) = G2(3) = G3(2) = B2(1) = B3(1) = B2(3) = B3(2) = F2(1) = F3(1) = F2(3) = F3(2) = 0
UUR	A2(1) = A3(1) = G2(1) = G3(1) = B2(1) = B3(1) = F2(1) = F3(1) = 0
UUU	A2(1) = A3(1) = A3(2) = G2(1) = G3(1) = G3(2) = B2(1) = B3(1) = B3(2) = F2(1) = F3(1) = F3(2) = 0

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