CAUSALITY BETWEEN US ECONOMIC POLICY AND EQUITY MARKET UNCERTAINTIES: EVIDENCE FROM LINEAR AND NONLINEAR TESTS

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This paper examines the causal relationship between economic policy uncertainty (*EPU*) and equity market

uncertainty (*EMU*) in the US using linear and nonlinear Granger causality tests. We use daily data on the newly

developed indexes by Baker et al. (2013a) covering 1985:01:01 to 2013:06:14. Results from the linear causality tests

indicate strong bidirectional causality. We test for parameters stability, and find strong evidence of short run

parameter instability, thus invalidating any conclusion from the full sample linear estimations. Therefore we turn to

nonlinear tests. Using Hiemstra and Jones (1994), Diks and Panchenko (2006), and Kyrtsou and Labys (2006)

symmetric test, we observe a stronger predictive power from *EMU* to *EPU* than from *EPU* to *EMU*. Using the

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1

asymmetric version of Kyrtsou and Labys (2006) test, we find no evidence of positive predictive power from *EPU* to *EMU*. However, we find strong evidence of positive predictive power from *EMU* to *EPU* and only weak evidence of negative *EPU* causing *EMU*. Performing the causality test using the Sato et al. (2007) time-varying method, we find that the causality between *EPU* and *EMU* is not constant over time but rather time-varying. Hence, we implement a sub-sample bootstrap rolling window causality tests to fully account for the existence of structural breaks. Using the intensity plots of the *p*-values from this, we find evidence that *EPU* can help predict the movements in *EMU* only around 1993, 2004 and, 2006. However, we find strong evidence that *EMU* can help predict the movements in *EPU* throughout the sample period barring around 1998, 2003 and 2005. Further, the analysis of total effects based on the bootstrap sum of coefficients suggests a positive and stronger causal effect from *EMU* to *EPU* but smaller and insignificant causality from *EPU* to *EMU*. The implications of these findings for both investors and policy makers are provided.

I. Introduction

The inability of government to change existing policies frequently may likely delay investment decisions by economic agents such as firms and investors. The resulting policy-related economic uncertainty, referred to as economic policy uncertainty, may affect the entire economy for instance through loss of outputs and jobs, stimulation of equity market uncertainty and slow economic recovery after a recession. Over the past thirty years, a number of researches have focused on the effect of economic policy uncertainty on macroeconomic variables: economic growth, inflation, investment and employment (Bernanke 1983 Rodrik 1991; Aizenman and Marion 1993; Ali 2001; Hermes and Lensink 2001; Bloom et al. 2007; Bloom 2009; Bachmann et al. 2013; Baum et al. 2010; Jones and Olson 2013; Bhagat et al. 2013; Colombo 2013 among others). The general consensus is that policy uncertainty has a negative effect on economic growth and investment but a less clear-cut effect on inflation. However, the recent global financial crisis with accompanying volatility in the equity market has kindled a protracted and high-profile debate over the role of key economic policies. This has primarily surrounded the European debt crisis and the US fiscal cliff and debt ceiling concerns, but also includes debate over such other policies as healthcare and financial services regulation (Fishman et al. 2012). It is believed that the recent increasing focus on economic policy uncertainty undoubtedly suggests the role it plays in economic growth and equity volatility market (Fishman et al. 2012).

Further, many investors argue that recent equity volatility levels are as much about policy as economics and corporate earnings. According to Li et al. (forthcoming), "stock markets usually move swiftly and sharply in response to policy changes. Tax cuts, monetary easing or financial deregulation would send the stock markets soaring. On the contrary, quantitative easing withdrawal would send the stock markets crashing." However, Li et al. (forthcoming) noted that the extent to which the stock market would be impacted on by policy changes (whether good or bad) depends on the certainty about such policy changes. Hence, economic policies with increased certainty often release definite signals that stimulate positive investment reactions while economic policies with increased uncertainty always cast a huge shadow of doubts that stall the investment decisions. Taylor (2010) and Hoshi (2011) suggest that high policy uncertainty in relation to the resolution of large bankrupt financial institutions has worsened or prolonged the recent financial crisis in the U.S. Although, a common explanation for the disappointment in the US economic performance over the past five years is an increase in policy uncertainty, Hatzius et al. (2012) argued that the economy's poor performance has been caused by an exogenous increase in US policy uncertainty.

This study intends to contribute towards the study of the effects of economic policy uncertainty, focusing on its effect on the US equity market performance. Specifically, we examine the causal link between two interesting new indexes, the US economic policy uncertainty index and the equity market uncertainty index developed by Baker et al. (2012, 2013a). We consider both the direction and magnitude of the causal and reverse causal effects. The choice for the US is justified because it is the only country with these indexes. A number of studies have investigated the relationship between economic policy uncertainty and equity market uncertainty or volatility.[1] This is particularly so since Baker et al. (2012, 2013a) constructed the respective two new indexes. For instance, Gregory and Rangel (2012) find a strong positive correlation between the US economic policy uncertainty and the level of S&P 500 variance (equity volatility) across different maturities. Using their own estimate of the market's implied earnings growth and the economic policy uncertainty index, Mezrich and Ishikawa (2013) find that the current US economic policy uncertainty index is far higher than before 2007, and that implied long-term earnings growth in equities could be pushed down to around 0.2 percent due to substantial existence of economic policy uncertainty. Baker et al. (2013b) also observed that the greater frequency (40 percent) of the US policy-driven equity market jumps is triggered by higher economic policy uncertainty.

Antonakakis et al. (2013) examine the time-varying correlations between the US stock market returns (and volatility) and policy uncertainty, and find that increased stock market volatility increases policy uncertainty and dampens stock markets returns while increases in the volatility of policy uncertainty lead to negative stock market returns and increased uncertainty. Pástor and Veronesi (2012; 2013) also show that the uncertainty about government policy increases stock volatility and risk premia, especially in a weak economy. Lam and Zhang (2014) use the economic policy uncertainty index of Baker et al. (2013a) and find that it has little explanatory power for international equity returns. Alternatively, they construct two new measures of global policy uncertainty based on the ratings from international country risk guide, which captures the potential policy shock from government changes and the bureaucratic ability to reduce policy shocks, and find that both factors significantly affect equity returns in 49 countries from 1995 to 2006.

Majority of these studies consider the relationship between the two series simply using correlation analysis or visual plots. Further, none of the studies account for structural break which is evident in the data. More importantly is the complete absence of studies examining the causality between the two newly developed indexes.

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[1] We do not provide literatures on the relationship between economic policy uncertainty index and equity market returns. However, interested readers may consult Li et al., 2013 for a review.

Existence of a correlation or relationship may neither imply causality nor can reverse causality be inferred. Therefore, this study fills these gaps by considering the causal link between these two series. We confront the data with a wide range of causality tests. First we consider different versions of the linear Granger causality tests. Results from full-sample causality tests may be misleading if structural changes exists. Therefore, we perform parameter stability tests on the estimated full sample VAR. Subsequently, we test for causality using nonlinear methods of Hiemstra and Jones (1994), Diks and Panchenko (2006), and Kyrtsou and Labys (2006), Sato et al. (2007) time-varying causality and Balcilar et al. (2010) sub-sample rolling window bootstrap causality tests. This paper makes contribution to the existing literature. Firstly, it is the first study to the best of our knowledge to investigate the causal link between Baker et al. (2013a) economic policy uncertainty and equity market uncertainty indexes. Secondly, it takes potential structural breaks into account by using nonlinear and time varying causality methods instead of limiting the analysis to full-sample data that assumes that single causality holds in every time period.

Majority of the studies reviewed above implicitly assume that policy uncertainty is exogenous and hence attempt to find the effect of policy uncertainty on equity market uncertainty. However, policy uncertainty is likely to be endogenous to other factors that affect equity market uncertainty. For instance, policy uncertainty has been found to be higher during elections and hence financial and economic variables also tend to change more during these periods (Rodrik 1991; Bernhard and Leblang 2006; Bialkowski et al. 2008; Boutchkova et al. 2012; Julio and Yook, 2013). Moreover, the Baker et al. (2013a) economic policy uncertainty index spikes upward around US presidential elections (Figure 1). Other exogenous influences include debates over the stimulus package, the debt ceiling dispute, wars and financial crashes (Gulen and Ion 2013). This means that political decisions and other economic news may affect existing policies or introduction of new ones, hence providing some exogenous variation in policy risk over time. This potential endogeneity has implications for statistical analysis and result interpretations based on correlations and regressions that do not isolate the impact of policy uncertainty on economic activity from confounding variables, i.e separating first moment shocks from second moment shocks. This might lead to a case of omitted variable bias which arises if increases in policy uncertainty tend to occur at the same time as increases in national election or other economic news. To address this endogeneity concerns, some studies have found proxies as instrumental variables for policy uncertainty while others have included several other variables that capture expectations about future economic conditions (Gulen and Ion 2013; Julio and Yook 2013; Wang et al. 2014). Therefore, in order not to fall prey of ignoring the endogeneity of policy uncertainty, this study analyses both causal

and reverse causal effects, testing whether causality runs from policy uncertainty to economic market uncertainty as well as whether causality runs from the latter to the former.

The rest of the paper is organized as follows: the data and preliminary analysis is represented in section 2. Section 3 presents the empirical models and results. Section 4 concludes.

II. Data and preliminary analysis

To examine the causality between economic policy uncertainty (*EPU*) and equity market uncertainty (*EMU*) in the United States, this study obtain daily *EPU* and *EMU* indexes from the Economic Policy Uncertainty Index website (http://www.policyuncertainty.com), newly introduced by Baker et al. (2013a). The data covers the 1985:01:01 to 2013:06:14 period. The end-point is pragmatic and was the final data point available at the time of writing. To measure policy-related economic uncertainty for the US, Baker et al. (2013a) construct an index from three underlying components, namely, newspaper coverage of policy-related economic uncertainty, the number and projected revenue effects of federal tax code provisions set to expire in future years and disagreement among economic forecasters about policy relevant variables as a proxy for *EPU*. To measure equity market uncertainty, Baker et al. (2013a) construct a news-based index which is based on the count of articles that reference 'economy' or 'economic', and 'uncertain' or 'uncertainty'' and one of 'stock price', 'equity price', or 'stock market' in 10 major U.S. newspapers, scaled by the number of articles in each month and paper. This news-based equity index is highly corrected with the widely used market-based equity volatility index (VIX) (Baker et al. 2013a). All the original data is processed by taking natural logarithms, to correct for potential heteroscedasticity and dimensional difference between series.

Figure 1a shows the plot of the original series. The scale on the left axis pertains to the policy uncertainty index while the scale on the right axis pertains to the equity market uncertainty. We also present the log transformed series in Figure 1b. Although, the two series exhibit high volatility as expected, they look quite stationary. The figures show *EPU* and *EMU* jumps corresponding to several prominent events, and much elevated levels of policy uncertainty since the 2007-09 recession. In particular, there are spikes associated with 1987 and 1998 stock market crashes, tight presidential elections, wars, 2001 September 11 attacks, contentious budget battles, and major policy decisions and battles during and after the recent global recession. Overall, there seems to be some co-movement

6

between the series. [2] To examine whether there is lead-lag relationship between EPU and EMU, we plot the corresponding 365-day moving average as shown in Figures 1c and 1d. From this figures there is no apparent way to decipher which variable is leading which. Figures may be used to make inferences about the lead-lag relationship; however they cannot provide a scientific proof of causality. Research in general is often based on scientific evidence. Hence, there is need for formal causality test. This study therefore proceeds with formal causality testing using various approaches as stated earlier.

[INSERT FIGURE 1]

Prior to investigating Granger causality, we test for the stationarity of the data using the using the Z_α unit root test of Phillips (1987) and Philips and Perron (1988) (PP), Augmented Dickey Fuller (ADF) test and MZ_α test of Ng and Perron (NP) (2001)[3]. We conduct the test with two specifications: intercept only and both trend and intercept. The results show that the two series (*EPU* and *EMU*) are I(0), meaning they are stationary. Hence, for subsequent analysis, we use the series in their natural logs.

[INSERT TABLE 1]

III. Empirical procedures and results

Following the above preliminaries the study now proceeds with the investigation of the causal connection between equity market uncertainty (*EMU*) and economic policy uncertainty (*EPU*) in the US . The null hypothesis is Granger non-causality between the two series. Granger non-causality occurs when the information set on the first variable (e.g., equity market uncertainty (*EMU*)) does not improve the prediction of the second variable (e.g., economic policy uncertainty (*EPU*)) over and above the predictive capacity of the information in the second variable. The two null hypotheses at stake are (a) that *EPU* does not Granger cause *EMU* and (b) that *EMU* does not Granger cause *EPU*. We use both linear and non-linear models for testing causality. This is because the linear test is only sensitive to causality in the conditional mean and may not be sufficient to detect nonlinear effects on the conditional distribution (Baek and Brock 1992). Hiemstra and Jones (1994) also noted that traditional linear Granger causality

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[2] We find a positive but low correlation (0.26) between EPU and EMU. Correlation simply shows whether there is a positive or negative association or comovement between two series, without showing which series leads the other, Moreover, any evidence of correlation may be due to other confounding factors. Therefore correlations are not sufficient to make causal inferences.

test have low power in detecting certain kinds of nonlinear relations. Higher order structure, such as conditional heretoskedasticity, is also often ignored (Diks and Panchenko 2005, 2006). In view of this, nonparametric approaches are appealing because they place direct emphasis on prediction without imposing a certain functional form.

First we use the classical linear Granger causality testing. Next, we also account for heteroscedasticity due to volatility clustering in our data as is evident in Figure 1. To take account of possible conditional heteroscedasticity of unknown form (Cheung and Ng 1996), we employ a popular heteroscedasticity-consistent covariance matrix estimator (HCCME) developed by MacKinnon and White (1985), known in the literature as HC3 estimator, for robustifying the classical linear Granger causality test. An alternative way to improve the performance of the classical Granger causality test in the presence of heteroscedasticity is to use a fixed design wild bootstrap procedure as in Hafner and Herwartz (2009). The wild bootstrap has been shown to yield reliable finite sample inference even when applied to data that are homoscedastic (Gonçalves and Kilian 2004). Therefore, we use the wild bootstrap method in addition to the *HCCME*. The technical details of the various linear models are provided in appendices 1 to 3.

The results from the linear Granger causality are presented in Table 2. The upper most panel reports the results from the classical Granger test, the middle panel reports the tests with the heteroscedasticity-robust variance covariance matrix and the lower panel reports the results from the wild-bootstrap procedure. The optimal lag length based on the Schwarz Information Criteria (SIC) test is eight (8) for the variables in their log-levels forming a VAR. In all the three versions of the linear tests, the null hypotheses are rejected at 1 percent, thus providing evidence in favour of bidirectional causality over the full sample. This implies the existence of a feedback system where *EMU* and *EPU* react to each other. In other words, movements in the *EPU* index can be significantly predicted by movements in the *EMU* index and vice versa.

[INSERT TABLE 2]

In the standard Granger causality testing, the full-sample is used for estimation. The assumption is that parameters of the VAR model used in testing are constant over time. However, when the underlying full-sample time series have structural changes, this assumption is probably violated. The results from the full sample causality would become invalid (Balcilar and Ozdemir 2013). Therefore, we test for parameter stability of the VAR results reported

in Table 2 using four different tests. We use the *Sup-F*, *Ave-F* and *Exp-F* tests developed by Andrews (1993) and Andrews and Ploberger (1994) to investigate the stability of the short-run parameters.

However, it is noted that when the underlying variables in levels are cointegrated, the VAR model in first differences is misspecified unless it allows for error-correction. Therefore, we use the *Lc* tests of Nyblom (1989) and Hansen (1992) to investigate the long-run parameters stability. If the series are I(1), the Nyblom-Hansen Lc test also serves as a test of cointegration (Balcilar et al. 2010). To avoid the use of asymptotic distributions, the p-values are obtained from a bootstrap approximation to the null distribution of the test statistics, constructed by means of Monte Carlo simulation using 2000 samples generated from a VAR model with constant parameters. The Sup-F, Ave-F and Exp-F tests needs to be trimmed at the ends of the sample. Following Andrews (1993) we trim 15 percent from both ends and calculate these tests for the fraction of the sample in [0.15, 0.85].

The results from the parameter stability tests are reported in Table 3. The first three rows of Table 3 report tests statistics for short-run parameter stability, starting with the *EMU* equation in the first two columns and followed by the *EPU* equation and the overall VAR system in turn. In row 1 the *Sup-F* statistic reports the test of parameter constancy against a one-time sharp shift in parameters. This is followed in rows 2 and 3 by two test statistics *Ave-F* and *Exp-F*, which assumes that parameters follow a martingale process, and test against the possibility that the parameters might evolve gradually.[4] The final test reported in Table 3 is the L_c test for the stability of the parameters for the *EPU* and *EMU* equations.

Starting with the L_c tests, the final row of Table 3 indicates significant evidence of parameter instability in both the *EMU* and *EPU* equations. Turning now to the first three rows of Table 3 where the sequential *Sup-F*, *Ave-F*, and *Exp-F* tests are reported. We find evidence of parameter instability in both equations and for the VAR as a whole. The evidence in Table 3 suggests both one-time shifts as well as a gradual evolution of the parameters in the *EMU-EPU* VAR. Parameter instability of the kind identified here would undermine traditional Granger causality tests of the connection between equity uncertainty and policy uncertainty. Hence, one would expect that the Granger causality tests to be sensitive to sample period changes in this case.

[INSERT TABLE 3]

Accordingly we proceed to investigate the association between *EMU* and *EPU* with nonlinear, time varying VAR and bootstrap rolling window Granger causality tests. Various nonparametric tests have been proposed in the

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[4] The Ave-F and Exp-F are both optimal tests as shown by Andrews and Ploberger (1994).

literature. The most prominent one perhaps is developed by Hiemstra and Jones (1994), which is a modified version of Baek and Brock (1992). An alternative nonlinear model is that proposed by Diks and Panchenko (2005, 2006) who show that the relationship tested by Hiemstra and Jones (1994) is not generally compatible with Granger causality leading to the over rejection of the null hypothesis. Hence, we use both the Hiemstra-Jones (1994) and Diks-Panchenko (2006) nonlinear causality tests. In addition, we also employ the Kyrtsou and Labys (2006) symmetric and asymmetric nonlinear approach. We also use Sato et al. (2007) time varying causality as well as Balcilar et al. (2010) sub-sample bootstrap rolling window causality to account for time variation in the relationship between the series. The technical details of the various nonlinear models are provided in appendices 4 to 8.

Table 4 reports the results from Hiemstra and Jones (1994) nonlinear Granger causality test based on the residual from the bivariate VAR. Following Hiemstra and Jones (1994), we set the value for the lead length of $m = 1$, the common lag lengths $(Lx = Ly)$ of 1 to 8 and a common scale parameter of $e = 1.5\sigma$, where $\sigma = 1$ denotes the standard deviation of the standardized time series test statistic. The standardized test statistic, denoted by TVAL, is asymptotically distributed N(0,1) under the null hypothesis of nonlinear Granger noncausality. The results in Table 4 indicate that the null hypothesis that *EPU* does not Granger cause *EMU* is rejected at 1 and 5 percent significance level, respectively for the 4th and 5th lags only. Analogously, the null hypothesis that *EMU* does not Granger cause *EPU* is rejected at 1 percent for lags 6, 7 and 8. Overall, the Hiemstra and Jones (1994) test provides evidence in favour of bidirectional nonlinear causality between *EMU* and *EPU* though this occurred at uncommon lags. The evidence is also stronger for causality from *EMU* to *EPU* than the reverse.

[INSERT TABLE 4]

Turning now to the results from the Diks and Panchenko (2006) nonlinear Granger causality test. The p-values of the test statistics are reported in Table 5. The results suggest evidence of bidirectional nonlinear causality between *EMU* and *EPU* for all the common lag lengths used in conducting the test. However, looking at the levels of significant, it is observed that *EMU* has stronger predictive power for *EPU* than does *EPU* for *EMU.* The evidence suggests that the *EMU* can be more helpful in predicting movements in the *EPU* index.

[INSERT TABLE 5]

The next nonlinear Granger causality we consider is that developed by Kyrtsou and Labys (2006). Our parameter prior selection is presented in Table 6. Our optimal integer delay variables for the causality from EPU

index to EMU index (τ_1), and for the causality from *EMU* index to *EPU* index(τ_2) as selected by SIC are set to 7 and 10, respectively. We also set the power of the lagged values of *EPU* index (c_1) and *EMU* index (c_2), respectively to 2 and 1.

[INSERT TABLE 6]

The results for both symmetric and asymmetric causality are presented in Table 7. From Table 7, we observe a strong evidence of bidirectional causality between *EPU* index and *EMU* index. Whether the direction of changes in the studied series has a significant effect on their causal relationships can be examined by using the asymmetric version of the Kyrtsou–Labys test. In order to do so, we demeaned both series since they contain only positive values. We remark no evidence that positive values of *EPU* index cause *EMU* index. Nevertheless, negative values of the former series significantly cause the latter only at 10% level. Moreover, we observe that only positive values of *EMU* index cause *EPU* index with strong evidence.

[INSERT TABLE 7]

We also conduct time-varying Granger causality tests developed by Sato et al. (2007). We implemented a dynamic Granger causality test (i.e., we test whether the Granger causality between two time series is time-invariant or not), as well as time-varying Granger causality test (i.e., we test for one variable does not cause the other versus one variable causes the other at least at one point in time). The results from the dynamic Granger causality are presented in the upper panel of Table 8. Interestingly, the null hypotheses that the causality from *EPU* to *EMU* and the causality from *EMU* to *EPU* are constant over time are both rejected. Turning now to the time-varying version of the Sato et al. (2007) test as reported in the lower panel of Table 8, we reject the null hypothesis of no causality in favour of the existence of strong time varying bidirectional causality between *EPU* and *EMU*. These findings support the results from the parameter stability test.

[INSERT TABLE 8]

The analysis so far points to the fact the causality between *EPU* and *EMU* cannot be constant. It therefore becomes important to see at which specific periods there is causality from one to the other as well as determine the magnitude and direction of impact. We now turn to the rolling sub-sample causality testing using the residual based

modified-LR causality tests with the null hypothesis that that *EPU* does not Granger cause *EMU* and vice versa. The bootstrap p-values of LR-statistics are estimated from the VAR models in Eq. (1) using the rolling sub-sample data. We set the maximum lags to 8 for a window of 60 and use SIC to choose lags for each window separately. After trimming 60-days observations from the beginning of the full sample, these rolling estimates move from 1985:03:02 to 2013:06:14. We present both the intensity and kernel density plots of the p-values for each sub-sample. Besides, the magnitude of the total effect of *EPU* on *EMU* and that of *EMU* on *EPU* are also calculated and presented.

Figure 2 shows the intensity plot of the bootstrap p-value of the *LR*-statistics for testing the hypothesis that *EPU* does not Granger cause *EMU* while Figure 3 shows the same plot for the hypothesis that *EMU* does not Granger cause *EPU*. These figures are based on counting the p-values falling in a grid of 1 year length in the horizontal axis and 0.1 on the vertical axis. From Figure 2, the p-values of testing that *EPU* does not Granger cause *EMU* have concentrations scattered everywhere. There are only three periods when the intensity is below 0.10. These are around 1993, 2004 and 2006. This shows that *EPU* has predictive power for *EMU* only for these few periods. On the other hand, Figure 3 indicates that the p-values of testing that *EMU* does not Granger cause *EPU* concentrate heavily below 0.10, almost uniformly from 1985 to 2013. There are minor exceptions around 1998, 2003 and 2005. These results point to a stronger evidence of causality from *EMU* to *EPU* over most of the periods. We also present the density plots in Figures A1 and A2. They show where the predictive power is concentrating from a nonparametric estimation. Based on the density plots, we find no evidence that *EPU* can help predict *EMU*, and very strong evidence that *EMU* has predictive power for *EPU*.

[INSERT FIGURE 2]

[INSERT FIGURE 3]

The strong causality from *EMU* to *EPU* can be linked to a number of important events that have strong financial and market connection. These include the 1987 stock market crash, 1997 Asian crisis, the 1997–2000 dot-com bubble, the 9/11 terrorist destruction of the World Trade Center's Twin Towers, the stock market crash of 2000–2002, the stock-market scandals of early 2002 (WorldCom, Enron etc), Lehman Brother's collapse in 2008 due

to the continuing subprime mortgage crisis, 2007-2009 global financial crisis and the 2011 debt ceiling debate. These findings suggest that the stock market uncertainty would increase uncertainty about economic policy in the US. The few cases where *EPU* hold predictive power for *EMU* may be associated with a number of events as well. The unanticipated election outcome that saw Bill Clinton as the winner in late 1992 will hold explanation to the around 1993 effect. The 2004 period effect may reflect the expiration of accelerated capital depreciation allowances. The Federal Reserve somewhat surprising move from a cycle of increasing interest rates, to a cycle of flat rates between June 2004 to August 2007 may also have influenced investors decisions and hence the equity market uncertainty.

Further, we consider the magnitude of the total effect of *EPU* on *EMU* and that of *EMU* on *EPU*. The bootstrap estimates of the sum of the rolling coefficients that *EPU* (*EMU*) does not have significant effect on *EMU* (*EPU*) is 0.0257 (0.0510) with the lower and upper 90% confidence bounds of -0.0121 and 0.0647 (0.0411 and 0.0612). These results show that *EMU* has a larger, positive and significant impact on *EPU* at the 10 percent level. However, *EPU* has smaller but insignificant impact on *EMU*.[5]

IV. Conclusion

Using a new economic policy uncertainty (*EPU*) and equity market uncertainty (*EMU*) indexes from Baker et al. (2013a), we investigate the causality between the two series using US daily data from 1985 to 2013. Empirical results based on the full-sample classical linear causality, heteroscedasticity-consistent covariance matrix estimator and wild bootstrap versions of the linear test, indicate a bi-directional causality between the two series. We conduct parameter stability tests on the full sample standard Granger tests and find that the short run relationship between *EPU* and *EMU* for the US is unstable over the sample period. Therefore, we also examine causality using various nonlinear Granger causality tests. While the Hiemstra and Jones (1994) nonlinear tests suggest evidence of bidirectional causality at higher but uncommon lags, the Diks and Panchenko (2006) nonlinear test suggest evidence of bidirectional causality at all common lags. Using the Kyrtsou–Labys (2006) nonlinear symmetric and asymmetric tests, we observe evidence of bidirectional causality with the symmetric tests while the asymmetric tests indicates that only positive values of *EMU* index cause *EPU* index with strong evidence while only negative values of the *EPU* significantly cause the *EMU* but only at 10% level.

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[5] We also apply the Hafner and Herwartz (2006) causality in variance test and found that the volatility in the equity uncertainty more strongly affects economic uncertainty. Specifically, in terms of volatility: *EMU* ≠> *EPU*: LM-stat: 486.9296 (p-value=0.000000); *EPU* ≠> *EMU*: LM-stat: 10.36887 (p-value=0.005603).

Using the Sato et al. (2007) time-varying Granger causality tests, we show that the causality between *EPU* and *EMU* is not constant over time but rather time-varying. Therefore, we extend our analysis by fully taking structural breaks into account using the bootstrap rolling window approach proposed by Balcilar et al. (2010). The bootstrap rolling window approach allows the causal relationship between series to be time-varying, instead of assuming that a permanent causal relationship holds over the whole period. Using the intensity plots of the bootstrap p values from the rolling testing approach, we observe that *EPU* has predictive power for *EMU* only for the 1993, 2004 and, 2006 sub-periods while *EMU* has predictive power for *EPU* almost at all sub-periods except for 1998, 2003 and 2005 sub-periods. Finally, our bootstrap residual-based total effects test based on sum of coefficients suggest a positive and stronger significant effect of *EMU* on *EPU* but smaller and insignificant predictive power from *EPU* to *EMU*. Our findings provide vital implications for policy makers and investors. First, the uncertainties surrounding the US equity market in recent years may be largely attributed to other factors (e.g declined expectations for economic growth) other than the economic policy uncertainty at least based on the time varying tests which takes into full account structural changes and regime switches . This is not to say that economic policy uncertainty does not matter for equity market uncertainty. Definitely, the need for the U.S. government to reduce uncertainty about economic policy in order to reduce potential risks in the stock market and hence increase investor confidence cannot be over stressed. However, the weak causal effect of *EPU* on *EMU* in this study simply shows that there are other fundamental factors that accounts for much of the movement in the US stock market other than economic policy uncertainty. This result is consistent with that of Li et al (forthcoming) who found a weak causal relationship between *EPU* and stock returns for China and India, thus concluding that robust economic growth coupled with favourable economic conditions, such as trade surplus and abundant capital inflows, dominates the stock market performance in China and India. Secondly, the strong in-sample predictive power of *EMU* for *EPU*, indicates that both soaring and crashing stock market performance may increase uncertainty about economic policies. Therefore, reducing stock market uncertainties for enhanced economic policy, investor confidence and overall economic growth is important. Future research may test if stock market uncertainty and economic policy uncertainty have out-of-sample forecasting ability for each other.

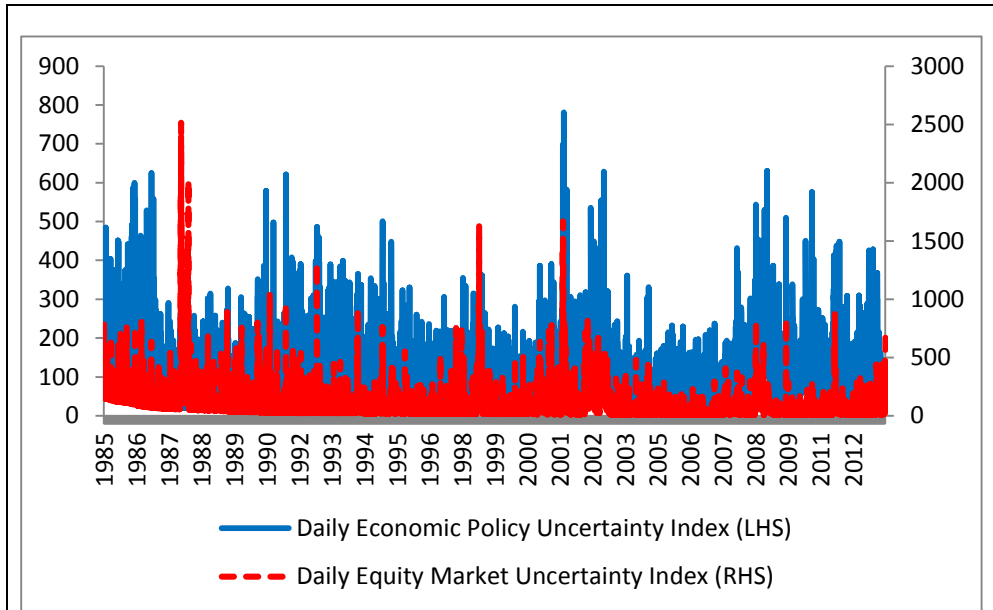**Figure 1. Economic policy uncertainty and equity market unceratinty**

Figure 1a. Policy uncertainty index and equity uncertainty index
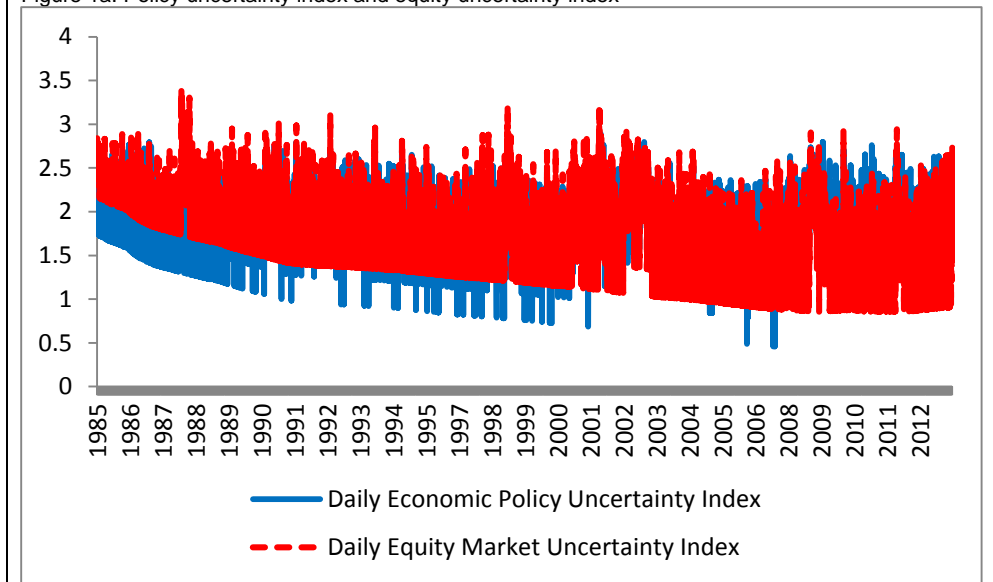

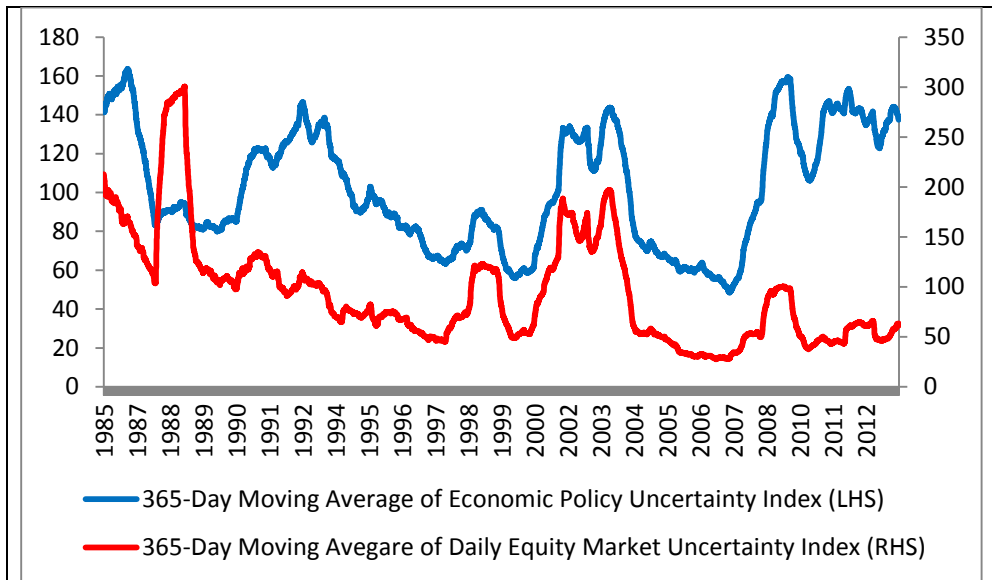Figure 1b. Policy uncertainty index and equity uncertainty index in logs

Figure 1c. Moving average of policy uncertainty index and equity uncertainty index (levels)
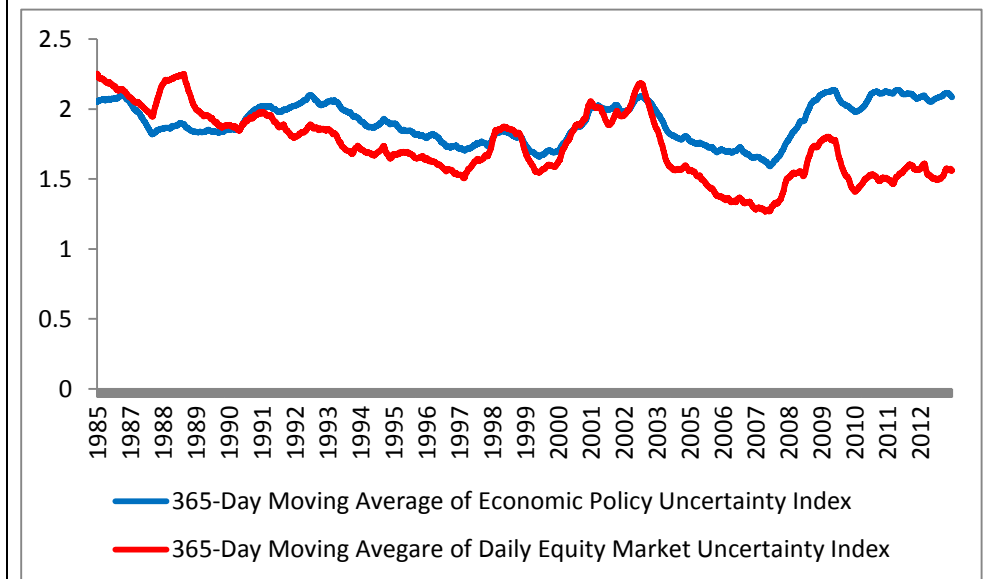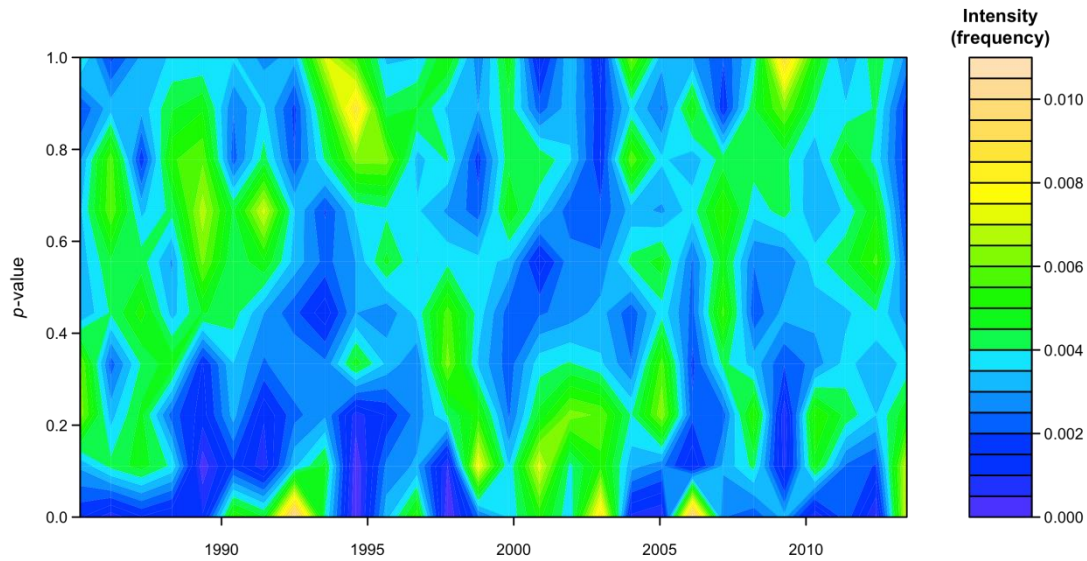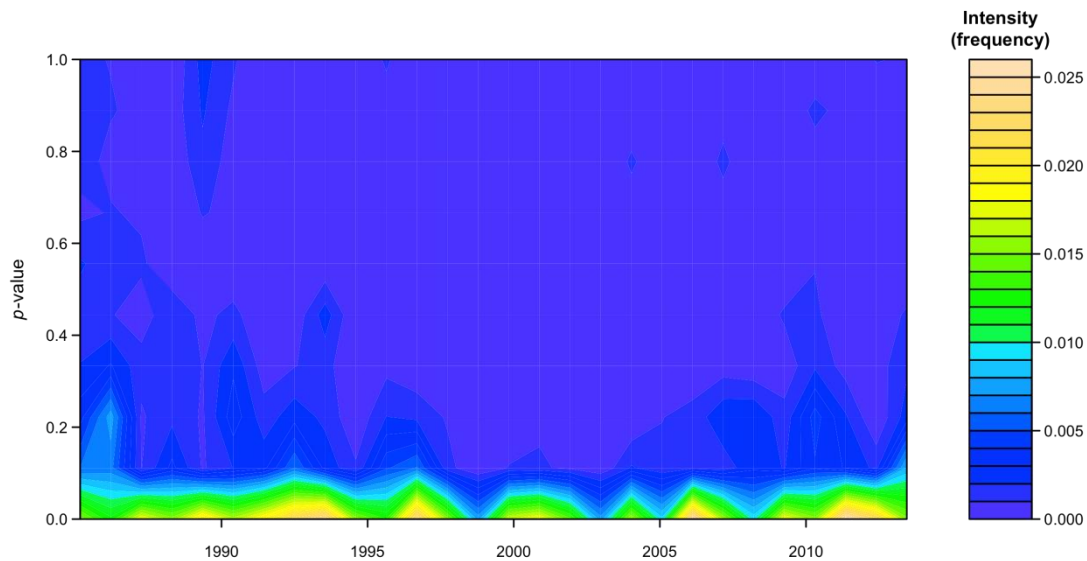

Figure 1d. Moving average of policy uncertainty index and equity uncertainty index (logs)

Figure 2. Intensity plot of p-value for testing economic policy uncertainty does not Granger cause stock market volatility

Figure 3. Intensity plot of p-value for testing stock market volatility does not Granger cause economic policy uncertainty

Table 1. Unit root testing

		Trend and Intercept	Intercept	Conclusion
	ADF	-13.705***	-12.899***	
EPU	PP	-97.716***	-97.656***	**I(0)**
	NP	-4.99***	-3.654***	
EMU	ADF	-6.838***	-6.836***	**I(0)**

PP	-150.881***	-150.889***
NP	-4.44***	-4.482***

Notes: *** indicates significance at the 1% level.

Table 2. Results from linear causality tests

Classical Granger Causality test

Hypothesis	p-value
EPU ⇏ EMU	2.2473×10^{-08a}
EMU ⇏ EPU	0.000^a

Granger causality tests with the heteroscedasticity-robust variance covariance matrix

Hypothesis	p-value
EPU ⇏ EMU	5.194×10^{-05a}
EPU ⇏ EMU	0.000^a

Granger causality tests with the wild-bootstrap procedure

Hypothesis	p-value
EPU ⇏ EMU	0.003^a
EPU ⇏ EMU	0.000^a

Notes: This table reports the p-values of the Granger causality tests. [a] indicates the rejection of the null hypothesis of absence of causality at the 1% level.

Table 3. Parameter stability tests

	EMU Equation		*EPU* Equation		VAR (8) System	
	Statistics	Bootstrap *p*-value[a]	Statistics	Bootstrap *p*-value[a]	Statistics	Bootstrap *p*-value[a]
Sup-F	243.38	<0.01	417.76	<0.01	644.43	<0.01
Ave-F	104.60	<0.01	275.73	<0.01	381.96	<0.01
Exp-F	114.22	<0.01	203.04	<0.01	316.40	<0.01
Lc	12.45	<0.01	34.71	<0.01		

Notes: *, **, and *** denote significance at 10, 5 and 1 percent, respectively. [a]*p*-values are calculated using 2000 bootstrap repetitions.

Table 4. Hiemstra and Jones (1994) nonlinear causality test

	H$_0$: EPU $\nRightarrow$ EMU			H$_0$: EMU $\nRightarrow$ EPU	
Lags	CS	TVAL	Lags	CS	TVAL
1	-0.1882	-18.8810	1	-0.5186	-52.0237
2	-0.1271	-12.7553	2	-0.5265	-52.8226
3	-0.0517	-5.1948	3	-0.5921	-59.4024
4	-0.0199	1.9967[b]	4	-0.8317	-83.4289
5	0.2898	29.0757[a]	5	-1.4940	-149.8658
6	-160.6963	-16119.3726	6	463.0408	46447.4101[a]
7	-0.6315	-63.3553	7	1.3329	133.7093[a]
8	-0.5153	-59.6967	8	0.6342	63.6236[a]

Notes: CS and TVAL are respectively the difference between the two conditional probabilities, and the standardized test statistic. "Lags" denote the number of lags in the residual series used in the test. [a] and [b] indicate the rejection of the null hypothesis of absence of causality at the 1% and 5% levels, respectively.

Table 5. Diks and Panchenko nonlinear causality test

$Lx = Ly$	H$_0$: EPU $\nRightarrow$ EMU	H$_0$: EMU $\nRightarrow$ EPU
1	0.0000[a]	0.0000[a]
2	0.0002[a]	0.0000[a]
3	0.0051[a]	0.0000[a]
4	0.0783[b]	0.0000[a]
5	0.0991[b]	0.0000[a]
6	0.0010[a]	0.0000[a]
7	0.0025[a]	0.0000[a]
8	0.0067[a]	0.0000[a]

Notes: This Table reports the p-values of the Diks-Panchenko causality tests. [a] and [b] indicate the rejection of the null hypothesis of absence of causality at the 1% and 10% levels.

Table 6. Parameter-prior selection in the M-G model

τ_1	τ_2	c_1	c_2
7	10	2	1

Notes: This table reports the results for the parameter-prior selection. τ_1 and τ_2 are the optimal integer delay variables for the causality from policy index to equity index, and for the causality from equity index to policy index, respectively. c_1 and c_2 are the power of the lagged values of policy index and equity index, respectively.

Table 7. Kyrtsou-Labys nonlinear causality test

Relation ($A \rightarrow B$)	F-statistic	Probability
$EPU \rightarrow EU$	8.2228	0.0041
$EU \rightarrow EPU$	2988.2	0.0000
$EPU^{+} \rightarrow EU$	1.9490	0.1627
$EU \rightarrow EPU^{+}$	30.6654	0.0000
$EPU^{-} \rightarrow EU$	3.1822	0.0745
$EU \rightarrow EPU^{-}$	584.0920	0.0000
$EU^{+} \rightarrow EPU$	41.8094	0.0000
$EPU \rightarrow EU^{+}$	3.4596	0.0629
$EU^{-} \rightarrow EPU$	0.0246	0.8753
$EPU \rightarrow EU^{-}$	0.0035	0.9526

Notes: we consider the null hypothesis that A does not cause B.

Table 8. Sato et al. (2007) time-varying test

Dynamic Granger causality test	
Hypothesis	P-value
EPU $\nRightarrow$ EMU	0.0000[a]
EMU $\nRightarrow$ EPU	0.0000[a]

Time-varying Granger causality test	
Hypothesis	P-value
EPU $\nRightarrow$ EMU	0.0000[a]
EMU $\nRightarrow$ EPU	0.0000[a]

Notes: − The dynamic Granger causality test allows to test whether the Granger causality between two time series is time-invariant or not (i.e., H0: The causality from X to Y is constant over time vs. H1: The causality from X to Y is not constant over time). − The time-varying Granger causality test examines the following hypotheses: H0: X does not cause Y vs. H1: X causes Y at least at one point in time. − Values in table are p-values. [a] indicates the rejection of the null hypothesis of absence of causality at the 1% level.

Appendices:

A1. The classical linear Granger causality testing

Granger (1969) defines causality between two stationary series in terms of predictability. Suppose x_t and y_t of length n are *EMU* and *EPU*, respectively. Testing for causal relations between the two series involves estimating a p-order linear vector autoregressive model, VAR(p), as follows:

$$\begin{bmatrix} y_t \\ x_t \end{bmatrix} = \begin{bmatrix} \alpha_1 \\ \alpha_2 \end{bmatrix} + \begin{bmatrix} \phi_{11,1} & \phi_{12,1} \\ \phi_{21,1} & \phi_{22,1} \end{bmatrix} \begin{bmatrix} y_{t-1} \\ x_{t-1} \end{bmatrix} + \begin{bmatrix} \phi_{11,p} & \phi_{12,p} \\ \phi_{21,p} & \phi_{22,p} \end{bmatrix} \begin{bmatrix} y_{t-p} \\ x_{t-p} \end{bmatrix} + \begin{bmatrix} \varepsilon_{1t} \\ \varepsilon_{2t} \end{bmatrix}, \tag{A1}$$

where $e_t = (e_{1t}, e_{2t})\prime$ is a white noise process with zero mean and covariance matrix Σ and p is the lag order of the process. In the empirical section, the Schwarz Information Criteria (SIC) is used to select the optimal lag order p. α_1 and α_2 are constants and $\phi's$ are parameters. In this setting, the null hypothesis that *EMU* does not Granger cause *EPU* can be tested by imposing zero restrictions $\phi_{12,i} = 0$ for $i = 1,2,..p$. In other words, *EMU* does not contain predictive content, or is not causal, for *EPU* if we do not the joint zero restrictions under the null hypothesis:

$$H_0^{EPU} : \phi_{12,1} = \phi_{12,2} = ... = \phi_{12,p} = 0, \tag{A2}$$

Analogously, the null hypothesis that *EPU* does not Granger cause *EMU* implies that we can impose zero restrictions $\phi_{21,i} = 0$ for $i = 1,2,..p$. In this case, *EPU* does not contain predictive content, or is not causal, for *EMU* if we do not reject the joint zero restrictions under the null hypothesis:

$$H_0^{EMU} : \phi_{21,1} = \phi_{21,2} = ... = \phi_{21,p} = 0, \tag{A3}$$

In either case, the rejection of non-Granger causality means that the movement in one series can be predicted by the other series. If only the hypothesis in either Eq. (2) or Eq. (3) is rejected, then there is a unidirectional causality. In the case that both hypotheses in Eq. (2) and Eq. (3) are rejected, the evidence points to bidirectional causality, which in this context implies a feedback system where *EMU* and *EPU* react to each other. It is also possible that neither of the two hypotheses are rejected implying that neither of the two variables has predictive content for the other.

A2. Heteroscedasticity-consistent covariance matrix estimator

The HCCME is given by:

$$HC3 : \hat{\Omega} = diag(\hat{\varepsilon}_i^2 / (1 - h_i)^2), \tag{A4}$$

where $\hat{\varepsilon}_i$ are the estimated residuals from a VAR(p) model and h_i is the ith diagonal hat matrix. The HC3 estimator appears to have better performance in small samples. A more extensive study of small sample behavior was carried out by Long and Ervin (2000) which arrive at the conclusion that the HC3 estimator provides the best performance in small samples as it gives less weight to influential observations.

A3. Wild bootstrap procedure

The wild bootstrap procedure is set up as follows:

1. Estimate the VAR(p) model and obtain the Wald statistic for non-causality as described by Hafner and Herwatz (2009).

2. Estimate the restricted VAR(p) model and obtain the estimated parameter values and the restricted residuals $\hat{\varepsilon}_i$.

3. Form a bootstrap sample of t observations, $\varepsilon_t^* = \hat{\varepsilon}_i \eta_t$, where η_t are a sequence of random variables with zero mean and unit variance being also independent of the variables occurring in VAR model. The pseudo disturbances η_t are generated using the Rademacher distribution

$$\eta_t = \begin{cases} -1 & with\ probability\ \pi = 0.5 \\ +1 & with\ probability\ 1 - \pi \end{cases}$$

4. Estimate the VAR(p) model for each artificial series and compute the Wald statistic in order to obtain the empirical distribution under the null hypothesis.

5. Repeat previous steps 1000 times to form a bootstrapping distribution. The p-value (p_b) of the test can be obtained as the proportion of the number of times the Wald test is smaller than the bootstrapped-Wald test.

6. Reject the null if p_b is smaller than the chosen significance level.

A4. Hiemstra and Jones (1994) Nonlinear Causality Test

Hiemstra and Jones (1994) proposed a nonparametric statistical method for detecting nonlinear causal relationships based on the correlation integral. To define nonlinear Granger causality, assume that there are two strictly and weakly dependent time series $\{X_t\}$ and $\{Y_t\}$, $t = 1,2,3,...,T$. Let m-length lead vector of X_t be designated by $\mathbf{X}_t^m$, and the Lx-length and Ly-length vectors of X_t and Y_t, respectively, by $\mathbf{X}_{t-Lx}^{Lx}$ and $\mathbf{Y}_{t-Ly}^{Ly}$. For given values of m, Lx and $Ly \geq 1$ and for all $e > 0$, $\{Y_t\}$ does not strictly Granger $\{X_t\}$ if:[6]

$$P(\left\| \mathbf{X}_t^m - \mathbf{X}_s^m \right\| < e \left\| \mathbf{X}_{t-Lx}^{Lx} - \mathbf{X}_{s-Lx}^{Lx} \right\| < e, \left\| \mathbf{Y}_{t-Ly}^{Ly} - \mathbf{Y}_{s-Ly}^{Ly} \right\| < e)$$
$$= P(\left\| \mathbf{X}_t^m - \mathbf{X}_s^m \right\| < e \left\| \mathbf{X}_{t-Lx}^{Lx} - \mathbf{X}_{s-Lx}^{Lx} \right\| < e), \tag{A5}$$

where $P(\cdot)$ denotes probability and $\|\cdot\|$ denotes the maximum norm. Eq. (5) states that the conditional probability that two arbitrary m-length lead vectors of $\{X_t\}$ are within distance e, given that the corresponding lagged Lx-length lag vectors of $\{X_t\}$ are e-close, is the same as when one also conditions on the Ly-length lag vectors $\{Y_t\}$ of being e-close.

A test based on Eq. (5) can be implemented by expressing the conditional probabilities in terms of the corresponding ratios of joint probabilities:

$$\frac{C1(m+Lx, Ly, e)}{C2(Lx, Ly, e)} = \frac{C3(m+Lx, e)}{C4(Lx, e)}, \tag{A6}$$

where $C1$, $C2$, $C3$ and $C4$ are the correlation integral estimator of the joint probabilities which are discussed in detail by Hiemstra and Jones (1994). With an additional index n, Hiemstra and Jones (1994) show that, under the assumption that $\{X_t\}$ and $\{Y_t\}$ are strictly stationary, weakly dependent, if $\{Y_t\}$ does not strictly Granger cause $\{X_t\}$ then,

---

[6] Strict Granger causality relates to the past of one time series influencing the present and future of another time series (Hiemstra and Jones 1994).

$$\sqrt{n}\left(\frac{C1(m+Lx,Ly,e,n)}{C2(Lx,Ly,e,n)}\right)-\left(\frac{C3(m+Lx,e,n)}{C4(Lx,e,n)}\right)\overset{a}{\sim} N(0,\sigma^2(m,Lx,Ly,e)), \tag{A7}$$

where $n=T+1-m-\max(Lx,Ly)$. See the appendix of Hiemstra and Jones (1994) for both definition and an estimator of $\sigma^2(m,Lx,Ly,e)$. One-sided (right-tailed) critical values are used, based on this asymptotic result, rejecting when the observed value of the test statistic in Eq. (7) is too large.

To test for nonlinear Granger causality between $\{X_t\}$ and $\{Y_t\}$, the test in Eq. (7) is applied to the estimated residual series from the bivariate VAR model. The null hypothesis is that Y_t does not nonlinearly strictly Granger cause X_t, and Eq. (7) holds for all m, Lx, $Ly\geq 1$ and $e>0$. By removing linear predictive power from a linear VAR model, any remaining incremental predictive power of one residual series for another can be considered as nonlinear predictive power (Baek and Brock 1992).

A5. Diks and Panchenko (2006) Nonlinear Causality Test

Diks and Panchenko (2005, 2006) argue that their test reduces the risk of over rejection of the null hypothesis of noncausality, observed in the Hiemstra and Jones (1994) widely used test. In this line, Dicks and Panchenko (2006) introduced a new nonparametric test for Granger non-causality which avoids this by replacing the global test statistic by an average of local conditional dependence measures. On the basis of these arguments, we employ both Hiemstra and Jones (1994) and Diks and Panchenko (2006) nonlinear Granger causality tests in this study.

Suppose that $X_t^{l_X}=(X_{t-\ell_X+1},\ldots,X_t)$ and $Y_t^{l_Y}=(Y_{t-\ell_Y+1},\ldots,Y_t)$ are the delay vectors - where ℓ_X, $\ell_Y\geq 1$. The null hypothesis of $X_t^{l_X}$ contain any additional information about Y_{t+1} is specified as:

$$H_0 = Y_{t+1}\Big|(X_t^{l_x};Y_t^{l_Y}) \sim Y_{t+1}\Big|Y_t^{l_Y}, \tag{A8}$$

The null hypothesis becomes a statement about the invariant distribution of the $(\ell_X+\ell_Y+1)$-dimensional vector $W_t=(X_t^{l_X},Y_t^{l_X},Z_t)$, where $Z_t=Y_{t+1}$. If we ignore the time index and we assume that $\ell_X=\ell_Y=1$, the distribution of Z - given that $(X,Y)=(x,y)$ - is the same as that of Z - given $Y=y$. In other words, X and Z are independent

conditionally on $Y = y$ for each fixed value of y, so the joint probability density function $f_{X,Y,Z}(x,y,z)$ and its marginals must satisfy the following relationship:

$$\frac{f_{X,Y,Z}(x,y,z)}{f_Y(y)} = \frac{f_{X,Y}(x,y)}{f_Y(y)} \cdot \frac{f_{X,Z}(y,z)}{f_Y(y)} \qquad (A9)$$

Diks and Panchenko (2006) show that the restated null hypothesis implies:

$$q \equiv E[f_{X,Y,Z}(X,Y,Z)f_Y(Y) - f_{X,Y}(X,Y)f_{Y,Z}(Y,Z)] = 0, \qquad (A10)$$

where $\hat{f}_W(W_i)$ is a local density estimator of a d_W-variate random vector W at W_i, defined by $\hat{f}_W(W_i) = (2\varepsilon_n)^{-d_W}$ $(n-1)^{-1}\sum_{j,j\neq i}I_{ij}^W$, where $I_{ij}^W = I(\|W_i - W_j\| < \varepsilon_n)$, $I(\cdot)$ the indicator function and ε_n the bandwidth, which depends on the sample size n.

The test statistic, which is a scaled sample version of q in Eq. (10), is simplified as:

$$T_n(\varepsilon_n) = \frac{n-1}{n(n-2)} \cdot \sum_i (\hat{f}_{X,Z,Y}(X_i,Z_i,Y_i)\hat{f}_Y(Y_i) - \hat{f}_{X,Y}(X_i,Y_i)\hat{f}_{Y,Z}(Y_i,Z_i)) \qquad (A11)$$

where T_n consist of a weighted average of local contributions $\hat{f}_{X,Y,Z}(X_i,Y_i,Z_i)\hat{f}_Y(Y_i) - \hat{f}_{X,Y}(X_i,Y_i)\hat{f}_{Y,Z}(Y_i,Z_i)$, which tend to zero in probability under the null hypothesis.

Diks and Panchenko (2006) prove that if $\varepsilon_n = Cn^{-\beta}(C > 0, \frac{1}{4} < \beta < \frac{1}{3})$ for one lag that the test statistic in Eq. (11) satisfies the following:

$$\sqrt{n}\frac{(T_n(\varepsilon_n)-q)}{S_n} \xrightarrow{D} N(0,1) \qquad (A12)$$

where $\xrightarrow{D}$ denotes convergence in distribution and S_n is an estimator of the asymptotic variance of $T_n(\cdot)$.

A6. The Kyrtsou and Labys (2006) nonlinear approach

Kyrtsou and Labys (2006) introduced the bivariate noisy Mackey-Glass (hereafter "M-G") model defined as follows

$$X_t = \alpha_{11} \frac{X_{t-\tau_1}}{1 + X_{t-\tau_1}^{c_1}} - \gamma_{11} X_{t-1} + \alpha_{12} \frac{Y_{t-\tau_2}}{1 + Y_{t-\tau_2}^{c_2}} - \gamma_{12} Y_{t-1} + \varepsilon_t \quad \varepsilon_t \to N(0,1), \tag{A13}$$

$$Y_t = \alpha_{21} \frac{X_{t-\tau_1}}{1 + X_{t-\tau_1}^{c_1}} - \gamma_{21} X_{t-1} + \alpha_{22} \frac{Y_{t-\tau_2}}{1 + Y_{t-\tau_2}^{c_2}} - \gamma_{22} Y_{t-1} + \vartheta_t \quad \vartheta_t \to N(0,1),),$$

where $t = \tau, ..., N, \quad \tau = \max(\tau_1, \tau_2)$ and $X_0,..., X_{\tau-1}, Y_0,... Y_{\tau-1}$ are given. The α_{ij} and γ_{ij} are parameters to be

estimated, τ_i are integer delays, and c_i are constants which can be chosen via prior selection. In this respect, the

best delays, τ_1 and τ_2, are selected on the basis of likelihood ratio tests and the Schwarz criterion. Different values

for τ and c can change dramatically the dynamic behaviour of the process. As pointed by Kyrtsou and Labys

(2007) the multivariate transformation of the model does not modify its dynamic properties in a univariate context.

Kyrtsou and Labys (2006) are the first to highlight Granger causality testing in this nonlinear setting by

finding nonlinear positive feedback in the relationships between commodity prices and US inflation. Later, this

nonlinear Granger causality testing was well explained in Hristu-Varsakelis and Kyrtsou (2008) and Hristu-

Varsakelis and Kyrtsou (2010).

The model in Eq. (13) is more appropriate than a simple VAR in case where dependency structures of

time series are more complicated and cannot be taken into account by vector autoregressions. The M-G-based

causality test is similar to the linear Granger causality test, except that the models fitted to the series are M-G

processes. This test is performed by estimating the M-G model parameters under no constraint with ordinary least

squares. To examine whether Y causes X, another M-G model is estimated under the constraint $\alpha_{12} = 0$ that

reflects our null hypothesis. Such a constraint arises from the fact that when Y has a significant nonlinear effect on

the current value of X in the model M-G, α_{12} must be significantly different from zero. Let $\hat{\omega}_t$ and $\hat{v}_t$ be the

residuals obtained respectively by the unconstrained and constrained best-fit M-G models. Thus, the corresponding

sums of residuals squares can be defined as $S_u = \sum_{t=1}^{T} \hat{\omega}_t^2$ and $S_c = \sum_{t=1}^{T} \hat{v}_t^2$. Recall that $n_u = 4$ is the number of free

parameters in the M-G model and on the other side $n_c = 1$ is the number of parameters required to be zero when estimating the restricted model. Obviously, the test statistic follows a Fisher distribution as

$$S_F = \frac{(S_c - S_u)/n_c}{S_u/(T-n_u-1)} \rightarrow F(n_c, T-n_u-1), \tag{A14}$$

where S_F is the test statistic.

What we have just presented is called the Kyrtsou-Labys "symmetric" version of the causality between X and Y. The "asymmetric" version of Kyrtsou-Labys test can be implemented by conditioning for positive or negative values of the causing series. Note that, since both series contain only positive values, we use demeaned data for this part of the analysis. To keep the matters tractable, suppose that we test, in Eq. (13), whether nonnegative returns in the series X cause the series Y. In this case, an observation (X_t, Y_t) is included in the regression model only if $X_{t-\tau_1} > 0$. The same restricted set of observations is used to compute the model corresponding to the null hypothesis, i.e., $\alpha_{21} = 0$. The procedure is then repeated with the order of the series reversed. That is, one can test whether positive returns in Y cause X and again with the subset of nonnegative returns. Note that conditioning in terms of causing series sign is not the only way to carry out an asymmetric causality. The sign conditioning is frequently chosen because it offers many advantages in practical relevance. Moreover, the nonpositivity, or respectively nonnegativity is not the only possible conditioning way as one can consider other events such as start/end of the week, price movement thresholds.

A7. Sato, Morettin, Arantes and Amaro (2007) time-varying causality analysis

The VAR used in Granger causality testing is an adequate approach only in cases when the processes to be modeled are stationary, i.e., the property of the models (expectation, variance, auto/cross-correlations) are invariant in time. These restrictions are not valid in many cases, since the system dynamics in real datasets exhibit changes depending on external factors (e.g., crisis, governmental interventions, and multinational agreements). In this line, Sato et al. (2007) have introduced a time-varying vector autoregressive modelling, by considering the model parameters as functions of time.

The time-varying vector autoregressive model (Sato et al., 2007) for a multivariate time series $x_{t,T} = (x_{1t,T}, x_{2t,T}, ..., x_{st,T})'$, where s is the dimension and T is the number of observations, is given by

$$x_{t,T} = u(t/T) + \sum_{l=1}^{p} A_l(t/T)x_{t-l,T} + \varepsilon_{t,T},\tag{A15}$$

where $\varepsilon_{t,T}$ is an error vector of independent random variables with zero mean and covariance matrix $\Sigma(t/T)$, $u(t/T)$ is the vector of intercepts and $A_l(t/T)$ are the autoregressive coefficients matrices with $l = 1,2, ..., p$.

The time-varying vector autoregressive model is an extension of the conventional VAR model. In this model, each VAR coefficient is described as a function of time. Here, we proposed to decompose these functions by using the B-splines decomposition (Eilers and Marx 1996) because it's less restrictive than the wavelets.

By using the B-splines time-function decomposition approach, the multivariate time-varying autoregressive model can be represented as:

$$x_t = \sum_{k=0}^{\infty} u_k \psi_k(t) + \sum_{l=1}^{p} \sum_{k=0}^{\infty} A_k^{(l)} \psi_k(t) x_{t-1} + \varepsilon_t,\tag{A16}$$

where $\psi_k(t)$ are B-splines functions (obs: $\psi_0(t) = 1$, constant for all t, u_k are vectors and $A_k^{(l)}$ ($l = 1,2, ..., p; k = 0,1,2, ...$) are matrices containing the B-splines expansion coefficients.

The basic idea of the estimation of the time-varying VAR is to represent the decomposition of the intercept and autoregressive time-functions as an approximation using finite linear combination of B-splines functions. In other words, each intercept and autoregressive function is described as a linear combination of M B-splines functions. By using this expansion, the model is approximated by a linear model with finite parameters, given by

$$x_t = \sum_{k=0}^{M} u_k \psi_k(t) + \sum_{l=1}^{p} \sum_{k=0}^{M} A_k^{(l)} \psi_k(t) x_{t-1} + \varepsilon_t,\tag{A17}$$

and the parameters of this model (which are the B-splines expansion coefficients) can then be estimated by using the least squares method in a linear multiple regression, similarly to the estimation of the conventional VAR models.

Then, the time-varying Granger causality test can be carried out by testing whether there is at least one autoregressive time-function from y_{it} to y_{lt} which is different from zero at least in one time point[7].

A8. Sub-sample Bootstrap Rolling Window Causality Approach

Generally, standard causality test statistics for joint parameter restriction and standard asymptotic properties include the Wald, Likelihood ratio (*LR*) and Lagrange multiplier (*LM*) statistics. With non-stationary data, as is typical in macroeconomic studies, these tests may not have standard asymptotic distributions (Toda and Phillips 1993, 1994). To address the problems of non-stationary underlying data, Toda and Yamamoto (1995) proposed a modified Wald test by estimating an augmented VAR model with I(1) variables to obtain standard asymptotic distribution for the Wald test. However, Mantalos and Shukur (1998) and Shukur and Mantalos (2000, 2004) have shown that the modified Wald test does not have correct size in small and medium size samples using Monte Carlo simulations. Hence, it is suggested that an improvement (in terms of power and size) can be achieved by using residual based bootstrap (*RB*) method critical values.

Further, the excellent performance of the *RB* method over the standard asymptotic tests, regardless of integration order or whether the series are cointegrated or not, has been confirmed in a number of Monte Carlo simulation studies (Shukur and Mantalos 2000; Hacker and Hatemi-J 2006; Balcilar et al. 2010). In light of this we also use the bootstrap *RB* based modified-*LR* statistics proposed by Balcilar et al. (2010) to examine the causality between *EPU* and *EMU* in the US. The starting point of the bootstrap *RB* based modified-*LR* Granger causality is Eq. (1) and the corresponding hypotheses in Eqs. (2) and (3).

Although the presence of structural changes can be detected beforehand and the estimations can be modified to address this issue using several approaches, such as including dummy variables and sample splitting, such an approach introduces pre-test bias. To overcome the parameter non-constancy and avoid pre-test bias, the rolling window sub-sample Granger causality tests, based on the modified bootstrap test is implemented.[8]

The rolling window estimators, also known as fixed-window estimators, are based on a changing subsample of fixed length that moves sequentially from the beginning to the end of sample by adding one observation at the end of the sample while dropping one at the start. Specifically, given a fixed-size rolling window including *l* observations, the full-sample is converted to a sequence of *T-l* subsamples, that is, τ-*l*+1, τ-*l*,..., *T*, for τ = *l*, *l*+1, ..., *T*.

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[7] Further technical details about the estimation of the time-varying VAR and hypothesis testing can be found in Sato et al. (2007).
[8] The technical details of the bootstrap test are explained in the appendix of Balcilar et al. (2010).

The *RB* based modified-*LR* causality is then applied to each subsample, instead of estimating a single causality test for full sample. Possible changes in the causal links between *EPU* and *EMU* for US are intuitively identified by calculating the bootstrap p-values of observed LR-statistic rolling through *T-l* sub-samples. More importantly, the magnitude of the effect of *EPU* on *EMU* as well as that of *EMU* on *EPU* is also assessed in this study. The effect of *EMU* on *EPU* is defined as the mean of all the bootstrap estimates, that is, $N_b^{-1}\sum_{k=1}^{P}\hat{\phi}_{12,k}^{*}$, where N_b equals the number of bootstrap repetitions. Analogously, the effect of *EPU* on *EMU* is calculated as the mean of all the bootstrap estimates, that is $N_b^{-1}\sum_{k=1}^{P}\hat{\phi}_{21,k}^{*}$. The estimates $\hat{\phi}_{12,k}^{*}$ and $\hat{\phi}_{21,k}^{*}$ are the bootstrap least squares estimates from the VAR in Eq. (1) estimated with the lag order of *p* determined by the SIC. The 90-percent confidence intervals are also calculated, where the lower and upper limits equal the 5th and 95th quantiles of each of $\hat{\phi}_{12,k}^{*}$ and $\hat{\phi}_{21,k}^{*}$, respectively.

Figure A1. Kernel density of p-value for testing economic policy uncertainty does not Granger cause stock market volatility
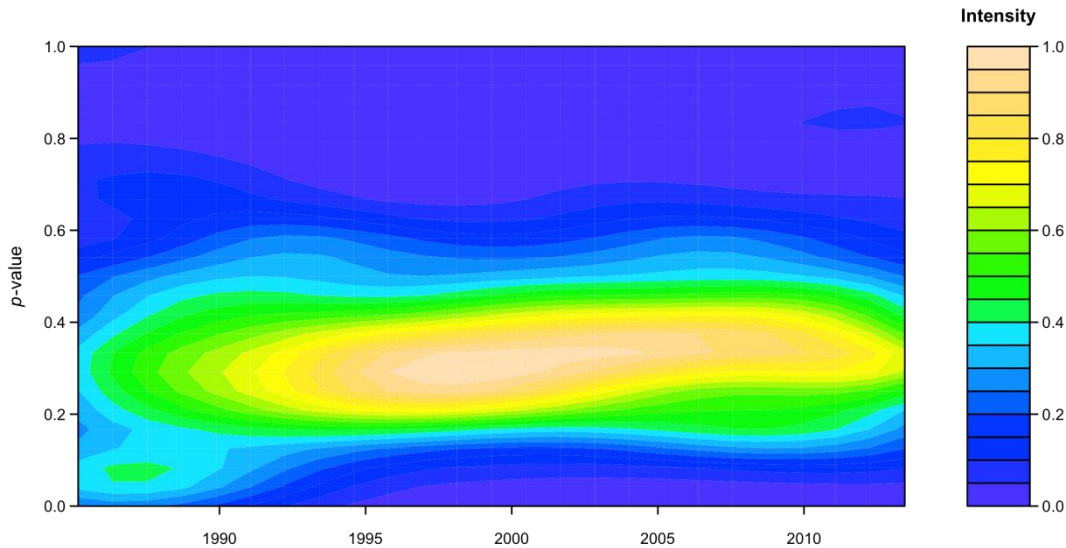
Figure A2. Kernel density of p-value for testing stock market volatility does not Granger cause economic policy uncertainty
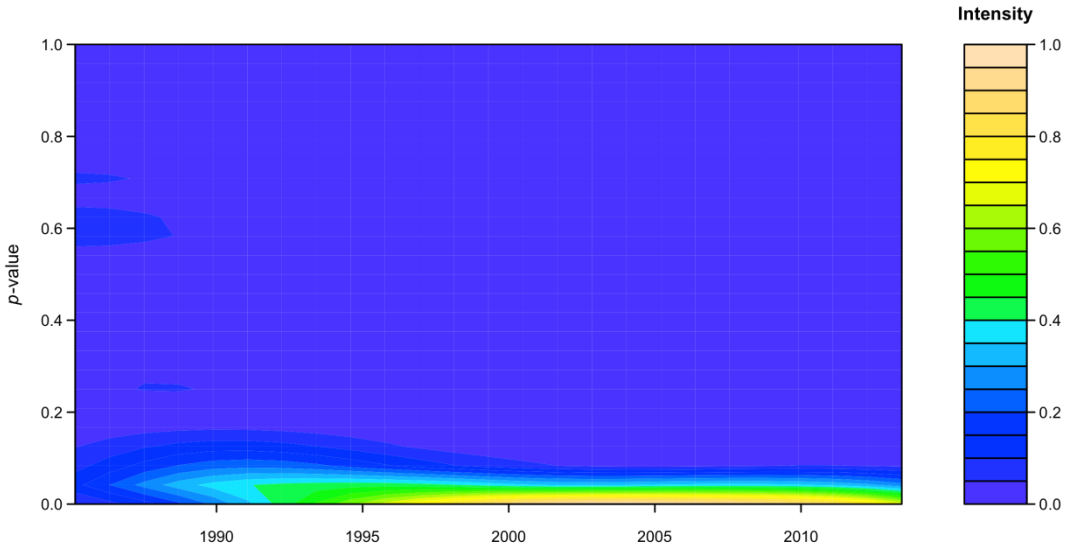
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