A Revisit on the Role of Macro Imbalances in
the US Recession of 2007-2009:
Nonlinear Causality Approach

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Abstract
This study reexamines evidences from a recent study by Paul (2010) on the role of macro imbalances in the US recession of 2007-09. Paul (2010) ascribes the prolonged recession to the twin deficits; while we identify fiscal deficit as the problem.

Keywords: Macro imbalances, US recession, nonlinear Granger causality
1. Introduction

The US recession of 2007-09 has drawn considerable interest among researchers and policymakers. Attempts have been made to identify cause of this recession. Using vector Autoregressive (VAR) model, Paul (2010) has shown that trade deficits fiscal deficits have contributed to the lower interest rate and declining output during 1987-2009. He also shows that low interest rates caused low private saving, which contributed to the housing bubble. So, Paul (2010) concludes: low saving and twin deficits have caused the recession.1

Paul’s (2010) results are based on VAR model which can produce biased result in the presence of nonlinearity in the series. This paper is revisits Paul’s (2010) conclusions by implementing the nonlinear Granger causality a la Hiemstra and Jones (1994) (HJ henceforth), which is an improvement over Baek and Brock (1992) (BB henceforth).

2. Nonlinear Granger causality

Linear approach to causality test can have low power in the presence of nonlinearity. BB first proposed a nonparametric test to capture nonlinear causality that is not detected by standard Granger test. The test utilizes the correlation integrals, which is an estimator of spatial probabilities across time, based on the closeness of the points in hyperspace that detects the relation between two time series. Because the distribution of the test statistic is one-tailed, rejections of the null hypothesis are restricted to one-tail. HJ modified the BB test which has better small-sample properties and does not require the assumption of i.i.d. HJ in their Monte Carlo simulations show that the modified test is robust to structural breaks in the series and contemporaneous correlations in the errors of the VAR model used to filter out linear cross- and auto-dependence. Following HJ, let 

\[ P(X_t | I_{t-1}) \]

denote the conditional probability distribution of 

\[ tX \]

given the information set \[ I_{t-1} \], which consists of an \( L_X \)-length lagged vector of \( tX \), say \( X_{t-1:Lx} \), and an \( L_Y \)-length lagged vector of \( tY \), say \( Y_{t-1:Ly} \). In the test, a given pair of lags \( L_X \) and \( L_Y \) holds within the following relationship:

\[ H_0: F(X_t | I_{t-1}) = F(X_t | (I_{t-1} - Y_{t-1:Ly})) \] (1)

The null hypothesis of interest thus states that taking the vector of past Y-values out of the information set does not affect the distribution of current X-values. Adopting the notation used by HJ, we denote the \( m \)-length lead vector of \( X_t \) by \( X_t^m \). We summarize the vectors defined so far, for \( t \in Z \), as:

1 More comprehensive review on this aspect can be referred in Paul (2010) as this study is just a revisit of the evidence of Paul (2010) therefore, review has been avoided.
A revisit on the role of macro imbalances in the US recession

\[ X_t^m = (X_t, X_{t+1}, \ldots, X_{t+m-1}), \quad m = 1, 2, \ldots \]
\[ X_{t-L_t}^L = (X_{t-L_t}, X_{t-L_t+1}, \ldots, X_{t-L_t+L_t}), \quad L_t = 1, 2, \ldots \]
\[ Y_{t-L_t}^L = (Y_{t-L_t}, Y_{t-L_t+1}, \ldots, Y_{t-L_t+L_t}), \quad L_t = 1, 2, \ldots \] (2)

A crucial claim made by Hiemstra and Jones (1994) without proof, states that the null hypothesis in equation (1) implies, for all \( \varepsilon > 0 \):

\[ P(\|X_t^m - X_t^m\| < \varepsilon \mid \|X_{t-L_t}^L - X_{t-L_t}^L\| < \varepsilon, \|Y_{t-L_t}^L - Y_{t-L_t}^L\| < \varepsilon) = P(\|X_t^m - X_t^m\| < \varepsilon \mid \|X_{t-L_t}^L - X_{t-L_t}^L\| < \varepsilon), \] (3)

where \( P(A \mid B) \) denotes the conditional probability of \( A \) given \( B \), and \( \|\cdot\| \) the maximum norms—a distance measure (here supremum norm), which for a \( d \)-dimensional vector \( x = (x_1, \ldots, x_d)^\top \) and is given by \( \|x\| = \sup_{j=1}^d |x_j| \). The probability on the left-hand side of equation (3) is the conditional probability that two arbitrary \( m \)-length lead vectors \( \{X_i\} \) (i.e., \( X_t^m \) and \( X_t^m \)) are within a distance \( \varepsilon \) of each other (\( \varepsilon \)-close), given the corresponding \( L_x \)-length lag vector of \( \{X_i\} \) (i.e., \( X_{t-L_t}^L \) and \( X_{t-L_t}^L \)) and \( L_y \)-length lag vector \( \{Y_i\} \) (i.e., \( Y_{t-L_t}^L \) and \( Y_{t-L_t}^L \)) are within \( \varepsilon \) of each other (or \( \varepsilon \)-close). The probability on the right-hand side (RHS) of equation (3) is the conditional probability that two arbitrary \( m \)-length lead/lag vectors of \( \{X_i\} \) (i.e., \( X_t^m \) and \( X_t^m \)) are within a distance \( \varepsilon \) for each other, given that the corresponding lagged \( L_x \)-length lag vectors of \( \{X_i\} \) being within a distance \( \varepsilon \) of each other; and two lag vectors of \( \{Y_i\} \) being within a distance \( \varepsilon \) of each other. Hence, non-Granger causality implies that the probability of two arbitrary lead vectors of length \( m \) are within a distance of \( \varepsilon \) of each other is the same, conditional upon the two lag vectors of \( \{X_i\} \) being within a distance \( \varepsilon \) of each other; and two lag vectors of \( \{Y_i\} \) being within a distance \( \varepsilon \) of each other. In other words, no Granger causality means that the probability of the lead vectors are within distance \( \varepsilon \) is the same whether we have information about the distance between \( \{Y_i\} \) lag vectors or not.

We can write the conditional probability expressed in equation (3) as ratios of joint probabilities. Assuming that \( C_1(m+L_x,L_y,\varepsilon)/C_2(L_x,L_y,\varepsilon) \) and \( C_3(m+L_x,\varepsilon)/C_4(L_x,\varepsilon) \) denote the ratio of joint probabilities corresponding to the left-hand side (LHS) and RHS of equation (3), the joint probabilities is written as:

\[ C_1(m+L_x,L_y,\varepsilon) = P(\|X_t^m+L_x - X_t^m+L_x\| < \varepsilon, \|Y_t^L - Y_t^L\| < \varepsilon), \]
\[ C_2(L_x,L_y,\varepsilon) = P(\|X_t^L - X_t^L\| < \varepsilon, \|Y_t^L - Y_t^L\| < \varepsilon), \]
\[ C_3(m+L_x,\varepsilon) = P(\|X_t^m\| < \varepsilon), \]
\[ C_4(L_x,\varepsilon) = P(\|X_t^L\| < \varepsilon). \]
The strict Granger non-causality condition in equation (3) can be written as follows:

\[
C_3(m + L_s, \varepsilon) = P \left( \|X_{t-L_s}^{m+L_s} - X_{s-L_s}^{m+L_s}\| < \varepsilon \right), \\
C_4(L_s, \varepsilon) = P \left( \|X_{t-L_s}^{L_s} - X_{s-L_s}^{L_s}\| < \varepsilon \right)
\]

For given values of \(m, L_s, L_t \geq 1\) and \(\varepsilon > 0\).

Let \(\{X_t\}\) and \(\{Y_t\}\) be the actual realization of the process and \(I(A, B, \varepsilon)\), an indicator function that takes the value 1 if the vector A and B are within a distance \(\varepsilon\) of each other; and zero otherwise. Also consider that the properties of the supremum norm allow us to inscribe \(\varepsilon\), so that the estimates of the correlations integrals in equation (5) can be expressed as:

\[
Cl(m + L_s, L_t, \varepsilon, n) = \frac{2}{n(n-1)} \sum \sum I \left( X_{t-L_s}^{m+L_s}, X_{t-L_s}^{m+L_s} \right) \cdot I \left( Y_{t-L_t}^{L_s}, Y_{t-L_t}^{L_t} \right), \\
C_2(L_s, L_t, \varepsilon, n) = \frac{2}{n(n-1)} \sum \sum I \left( X_{t-L_s}^{L_s}, X_{t-L_s}^{L_s} \right) \cdot I \left( Y_{t-L_t}^{L_s}, Y_{t-L_t}^{L_t} \right), \\
C_3(m + L_s, \varepsilon, n) = \frac{2}{n(n-1)} \sum \sum I \left( X_{t-L_s}^{m+L_s}, X_{s-L_s}^{m+L_s} \right) \\
C_4(L_s, \varepsilon, n) = \frac{2}{n(n-1)} \sum \sum I \left( X_{t-L_s}^{L_s}, X_{s-L_s}^{L_s} \right)
\]

For \(t, s = \max(L_s, L_t) + 1, ..., T - m + 1; n = T + 1 - m - \max(L_s, L_t)\).

Assuming that \(X_t^m\) and \(Y_t^m\) are strictly stationary and meet the required mixing conditions as specified in Denker and Keller (1983); and under the null hypothesis \(Y_t^m\) does not strictly Granger cause \(X_t^m\), the test statistic \(T\) is asymptotically normally distributed. That is,

\[
T = \left( \frac{Cl(m + L_s, L_t, \varepsilon, n) - C_3(m + L_s, \varepsilon, n)}{C_2(L_s, L_t, \varepsilon, n) - C_4(L_s, \varepsilon, n)} \right) \sim N \left( 0, \frac{1}{\sqrt{n}} \sigma^2(m, L_s, L_t, \varepsilon) \right),
\]

\[
\sigma^2(m, L_s, L_t, \varepsilon) = \frac{C_2(L_s, L_t, \varepsilon, n)}{C_2(L_s, L_t, \varepsilon, n) - C_4(L_s, \varepsilon, n)}
\]
where, \( n = T + 1 - m - \max(L_x, L_y) \), and \( \sigma^2() \) is the asymptotic variance of the modified BB test statistic.\(^2\) Based on this asymptotic results and one-sided critical values, the null is rejected if the test statistic in equation (6) is too large. To test for nonlinear Granger causality between \( \{X_t\} \) and \( \{Y_t\} \); test statistic in equation (6) is applied to the estimated residual series from the bivariate VAR model. In this case, the null hypothesis is: \( \{Y_t\} \) does not nonlinearly strictly Granger cause \( \{X_t\} \), and equation (6) holds for all \( m, L_x, L_y \geq 1 \) and \( \varepsilon > 0 \). By removing a linear predictive power from a linear VAR model, any remaining incremental predictive power of one residual series for another can be considered nonlinear predictive power (see Baek and Brock, 1992). A significant test statistic in equation (6) suggests that lagged values of \( Y \) help to predict \( X \), whereas a significant negative value suggest knowledge of the lagged value of \( Y \) confounds the prediction of \( X \). For this reason, the test statistic in equation (6) should be evaluated with right-tailed critical values when testing for the presence of Granger causality. Using Monte Carlo simulations Hiemstra and Jones (1993) find that the modified Baek and Brock (1992) test has remarkably good finite sample size and power properties against a variety of nonlinear Granger causal and non-causal relations.

3. Data analysis and results interpretation

Prior to formally checking for nonlinear Granger causality, the Brock, Dechert, and Scheinkman (BDS) test formally tests if the data are characterized by nonlinearities.\(^3\) The BDS approach essentially tests for deviations from identically and independently distributed (i.i.d.) behavior in time series. Results show that the vast majority of the BDS statistics are statistically significant, suggesting significant nonlinearities in the univariate time series.\(^4\) Values for the lead length \( m \), the lag lengths \( L_x \) and \( L_y \), and the distance measure \( \varepsilon \) must be selected in prior to implementing the Baek and Brock (1992) test. This is sharp contrast with the linear causality test where we do not have any well developed methods for choosing optimal lag lengths and distance measure. Following Hiemstra and Jones (1994) we set the lead length at \( m = 1 \) and set \( L_x = L_y \) for all cases. The common lengths of \( (1 - 5) \) lags and a common distance measure of \( \varepsilon = 1.5\sigma \) (\( \sigma \) denotes the standard deviation of the series)\(^5\) is used. In the results focus on p-values for the modified BB test as this enables us to compare them.

\(^2\) The asymptotic variance is estimated using the theory of U-statistic for weakly dependent processes (Denker and Keller, 1983). For detailed derivation of the variance see the appendix in Hiemstra and Jones (1994).

\(^3\) We are thankful for Prof. Paul for sharing the data used in his paper. Data source for related variables can be found in his paper, and to Mr. Panchenko for providing me the codes for the analysis.

\(^4\) Correlation and descriptive statistics are presented in the working paper version of the study and can be found in Tiwari (2011). Results of the BDS test are available upon request.

\(^5\) In estimation we also considered \( \varepsilon = 0.5\sigma \) and \( 1.0\sigma \); but without any qualitative differences.
with the empirical p-values obtained using the re-sampling procedure. The empirical p-values account for estimation uncertainty in the residuals of the VAR model used in the modified BB test which makes the results more reliable.\footnote{Baek and Brock (1992) (BB) suggest that their test could spuriously reject the null hypothesis of Granger non-causality from the presence of non-stationarity due to structural breaks in the data and heteroskedasticity [see Diks and Panchenko (2005, 2006) for the effect of conditional heteroskedasticity]. Granger non-causality test does not identify the underlying source of causality which may be due to structural breaks in the data (BB; Andersen, 1996), differential reaction to information flow, proxied by volatility (Ross, 1989), or some combination. To test if results are period-sensitive, experiment with sub-periods is possible leaves very small sample in both periods which can misleading. Since modified BB test uses residuals of the VAR model, not the original untreated observations, inference may be erroneous due to unaccounted estimation uncertainty. Randles (1984) points out that in the above noted situation the potential difference in outcome is not reflected in the test statistics. To avoid this, we use a re-sampling scheme by incorporating parameter estimation uncertainty. We use the test statistics of the modified BB, but using the re-sampling procedure of Diks and DeGoede (2001) to determine the empirical p-values of nonlinear Granger causality tests. The test statistics $T_i$ is given in equation (6).} Diks and DeGoede (2001) conducted several experiments to determine the best randomization procedure for obtaining empirical p-values. They found that the best finite sample properties of the tests are obtained when only the causing series are bootstrapped in the analysis. As such this methodology has been adopted in this paper. Specifically, we used the stationary bootstrap of Politis and Romano (1994) to preserve potential serial dependence in the causing series. The re-sampling scheme\footnote{The re-sampling procedure imposes a more restrictive null hypothesis of conditional independence. However, the test detects the deviations from the null in the direction of Granger causality. Let N denote the length of the series and PS is the stationary bootstrap switching probability. We start a new bootstrapped sequence from a random position in the initial series selected from the uniform distribution between 1 and N. With probability 1–PS, the next element in the bootstrapped sequence corresponds to the next element in the initial series. With probability PS we randomly select an element from the initial sequence and put it as the next element in the bootstrapped sequence. The procedure continues until we obtain a bootstrapped sequence of length N. To ensure stationarity of the bootstrapped sequence, we connect the beginning and the end of the initial sequence.} which is robust with respect to parameter estimation uncertainty is implemented as follows:

1. Estimate a parametric model and obtain the fitted values of the conditional mean and the estimated residuals.
2. Resample the residuals in such a way that satisfies the null hypothesis.\footnote{The re-sampling scheme is heavily drawn from Francis et al. (2010).}
3. Create artificial series using the fitted values and the re-sampled residuals.
4. Further, re-estimate the model using the artificial data and obtain new series of the residuals.
5. Compute test statistics $T_i$ for the artificial residuals.

By repeating the bootstrap N-times and calculating test statistic $T_i$ for each bootstrap $i=1…N$, we obtain empirical distribution of the test statistics under the null. Further, to obtain the empirical p-values of the test we compare the test statistics computed from the initial data $T_o$ with the test statistics under the null $T_i$:

$$p = \frac{\sum_{i=0}^{N} \#(T_o \leq T_i)}{N + 1},$$
A revisit on the role of macro imbalances in the US recession

where, #(·) denotes the number of events in the brackets. The test rejects the null hypothesis in the direction of nonlinear Granger causality whenever T_o is large. For the bootstrapping the number of bootstraps is set at N=99.\(^9\) The bootstrap switching probability PS is set to 0.05. The results based on the bootstrapped empirical p-values of non-linear Granger causality analysis are reported in the following Table 1.

Table 1: Results of nonlinear Granger causality

<table>
<thead>
<tr>
<th>Null hypothesis about Granger causality</th>
<th>Lag1</th>
<th>Lag2</th>
<th>Lag3</th>
<th>Lag4</th>
<th>Lag5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fiscal deficit does not cause Fed rate</td>
<td>0.25</td>
<td>0.21</td>
<td>0.38</td>
<td>0.36</td>
<td>0.32</td>
</tr>
<tr>
<td>Trade deficit does not cause Fed rate</td>
<td>0.91</td>
<td>0.54</td>
<td>0.70</td>
<td>0.28</td>
<td>0.44</td>
</tr>
<tr>
<td>Fed rate does not cause saving rate</td>
<td>0.26</td>
<td>0.11</td>
<td>0.18</td>
<td>0.18</td>
<td>0.28</td>
</tr>
<tr>
<td>Fiscal deficit does not cause GDP growth</td>
<td>0.81</td>
<td>0.73</td>
<td>0.27</td>
<td>0.18</td>
<td>0.61</td>
</tr>
<tr>
<td>Trade deficit does not cause GDP growth</td>
<td>0.37</td>
<td>0.14</td>
<td>0.09</td>
<td>0.11</td>
<td>0.12</td>
</tr>
<tr>
<td>Trade deficit does not cause fiscal deficit</td>
<td>0.10</td>
<td>0.06</td>
<td>0.25</td>
<td>0.22</td>
<td>0.28</td>
</tr>
<tr>
<td>Fiscal deficit does not cause Trade deficit</td>
<td>0.62</td>
<td>0.25</td>
<td>0.22</td>
<td>0.23</td>
<td>0.32</td>
</tr>
<tr>
<td>Trade deficit does not cause saving rate</td>
<td>0.18</td>
<td>0.62</td>
<td>0.60</td>
<td>0.42</td>
<td>0.16</td>
</tr>
<tr>
<td>Fed rate does not cause Fiscal deficit</td>
<td>0.01</td>
<td>0.32</td>
<td>0.50</td>
<td>0.54</td>
<td>0.27</td>
</tr>
<tr>
<td>Saving rate does not cause trade deficit</td>
<td>0.46</td>
<td>0.44</td>
<td>0.63</td>
<td>0.14</td>
<td>0.23</td>
</tr>
<tr>
<td>Fiscal deficit does not cause saving rate</td>
<td>0.24</td>
<td>0.70</td>
<td>0.60</td>
<td>0.26</td>
<td>0.03</td>
</tr>
<tr>
<td>Saving rate does not cause fiscal deficit</td>
<td>0.06</td>
<td>0.62</td>
<td>0.62</td>
<td>0.72</td>
<td>0.35</td>
</tr>
<tr>
<td>Fed rate does not cause trade deficit</td>
<td>0.76</td>
<td>0.93</td>
<td>1.00</td>
<td>0.96</td>
<td>0.95</td>
</tr>
<tr>
<td>Saving rate does not cause Fed rate</td>
<td>0.15</td>
<td>0.42</td>
<td>0.15</td>
<td>0.07</td>
<td>0.32</td>
</tr>
<tr>
<td>GDP growth does not cause fiscal deficit</td>
<td>0.01</td>
<td>0.08</td>
<td>0.21</td>
<td>0.09</td>
<td>0.24</td>
</tr>
<tr>
<td>GDP growth does not cause trade deficit</td>
<td>0.52</td>
<td>0.11</td>
<td>0.07</td>
<td>0.74</td>
<td>0.79</td>
</tr>
</tbody>
</table>

Note: This table reports parametric bootstrap p-values for the standard Baek and Brock (1992) nonlinear Granger causality test (equation-6). The number of lags on residuals used is one. All tests are applied to the unconditional unstandardized residuals. The lead length, m=1, and distance measure, \(\varepsilon\)=1.5. Bold are significant.

It is evident from Table 1 that fiscal deficit and trade deficit do not Granger-cause Fed rate; fiscal deficit does not Granger cause GDP growth and trade deficit; Fed rate does not Granger cause saving rate and trade deficit. However, trade deficit Granger case GDP growth and fiscal deficit; fiscal deficit Granger cause saving rate and saving rate Granger fiscal deficit and fed rate and GDP growth Granger cause both trade deficit and fiscal deficit.

The findings contrast with those of Paul (2010). He found that fiscal and trade deficit Granger-cause Fed rate and argued that high fiscal and trade deficit lowers the Fed rate. This implies that macroeconomic imbalances indirectly contributed to the cheap interest rates.

\(^9\) B=99 is the smallest commonly suggested number of bootstrap replications (see Davidson and MacKinnon, 2000). Due to computational limitations we were unable to increase N, which may cause low power for our tests.
monetary policy which led to the housing bubble and ultimately caused the financial crisis. We argue that there might be any number of reasons working towards a cheap monetary policy in the US; the least of which is the two imbalances Paul (2010) cites. Further, Paul (2010) found that Fed rate Granger-cause saving rate. So, he concluded that Fed rate was responsible for the falling saving rates and thus the failure to pay for the home, leading to lower equity, higher leverage, higher risk and an ever growing bubble in the housing market. The findings of this paper do not lend any support to Paul. Paul’s (2010) finds that the twin deficit Granger-cause GDP growth and to output decline. We find that the trade deficit is the factor causing output decline, not the fiscal deficit. In addition to that I also find that GDP growth is also causing twin deficit that implies that GDP growth has increased the burden of trade deficit and fiscal deficit. Further, Paul (2010) found that twin deficits show bidirectional Granger causality. By contrast, we find that trade deficit Granger-cause fiscal deficit while fiscal deficit does not. Although research shows that fiscal deficit and saving rate Granger cause each other i.e., fiscal deficit and savings rate reinforce each other, we also found that Fed rate Granger cause fiscal deficit i.e., cheap monetary policy has been the cause of high fiscal deficit. We did not find evidence trade deficit to lower saving rates or that savings were helped by increased trade deficit.

4. Conclusions

This paper revisits the findings by Paul (2010) who identifies trade and fiscal deficit as the factors behind the cause of the great recession of 2007-09 in the US, the worst since the Great Depression. By applying the Granger causality, Paul (2010) argues that the twin deficits have contributed to lowering the interest rate and output decline over the period of 1987-2009. Paul (2010) results are suspect as he did not check the stationarity property of the series in applying Granger causality. Using more refined methods appropriate to the context, we did not find any evidence to support for the twin deficit hypothesis, rather it is the trade deficit which has lowered the GDP growth. Further, it is not the low interest rate which caused low savings but it is low rate of savings which caused the low rate of interest rate which caused boom and that contributed to the housing bubble. Low saving led to the failure by the borrowers to cushion the debt. The housing bubble is related to fiscal deficit. Low fed rate (interest rate) and GDP growth rate, and high saving rate and trade deficit have contributed to high fiscal deficit, whereas high fiscal deficits has increased the saving rate. Finally, increased savings has lowered the interest rate (i.e., cheap monetary policy) which has been the cause of housing bubble.
References


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