Banking net income and macroeconomics, from multicollinearity to Granger causality using US data

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Abstract

We select six macroeconomic variables and study their relation with (aggregate) net banking income. The aggregate net banking income was reconstructed from US banking sector authorities' data. Usefulness may be twofold, it provides aggregate insight and the methodology can be replicated at bank institution level. We use standard tools such as linear regression analysis (to study multicollinearity) and Granger causality. The obtained results suggest a highly changing relation between all variables in time and an increase of causality and feedback relations after the 2008 crisis.

Keywords: Banking Net Income, Macroeconomics, Multicollinearity, Granger causality

JEL Classification: C22, E44, G21

1 Introduction

An important condition for economic strength is a stable banking system. Key factors for banking stability are capital, asset, funding and income structures (Altunbas, Manganelli, & Marques-Ibanez, 2011). We will focus on some aspects of one of these elements; income. Sources of determinants of banking income are usually classified in two types. Macroeconomic, general of the economic system; and idiosyncratic, particular to each institution. Numerical models test macroeconomic dynamics impact on some quantitative economic-financial measurement[1], in terms of position and evolution. The link between macroeconomic variables (MV) and the specific bank institution is usually done via a satellite model (Henry et al., 2013); which provides the connection between relevant MV

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[1]Typically balance sheet or income statement.

and the key bank indicator (usually a risk measure). In this framework, the explanatory power of each variable is relevant to individual institutions as determinant of their results (Hughes & Poi, 2015) since they provide information on what factors banking income depend.

Some constrains of this work may provide a stricter delimitation of the scope. We do not use explicit macro and satellite models, but preserve the structural relation between macro and micro (bank level) variables. Hence, consequences of the relations for concrete capital calculation, risk measurement and stress testing forms are out of the range of this work. Furthermore, we do not seek a most representative mix of variables that explains or is useful to predict in some optimal way [2]. For concreteness, significant variables are absent, notably bank level variables or MV such as foreign exchange rates or the yield curve. The aim is to analyse the form of structural long-term relations between the MV and the bank sector situation expressed via it income. These bounds do provide a simpler framework without loss of generality. In section 2 variables are presented, section 3 discusses stationarity. Section 4 examines a linear regression of net banking income as dependent variable considering structural breaks, heterocedasticity and multicollinearity. Section 5 build over the hints of section 4 analysing Granger causality between MV and its Granger causal relation with banking net income afterwards. The final part of section 5 studies the increase of causality and feedback relations. Section 6 concludes[3].

2 Election of variables

In this paper we want to describe the relation between banking income and some MV using usual econometric tools. We use annual data for macroeconomic series and net income of commercial banks of the United States[4]. Series have data from 1959 for all variables, but as we use some differentiated data for regression and analysis, the data frame employed covers from 1961 to 2017. The Federal Reserve of St. Louis is the source for all macroeconomic series[5]. For banking sector net income; we use Annual Reports of the Federal Deposit Insurance Corporation until 1980[6] and the Annual Statistic Digest of the Federal Reserve for the period 1981-1984[7]. From 1985 onwards data is taken from the Federal

[2] Functional forms and simplicity.

[3] Complete results of Granger causal relations between variables are shown in appendix A.

[4] All econometric tests of this work are performed with Eviews 9 with a significance level of 5% when not indicated otherwise, the analysis of the results of section 5 have been realized with Excel 2016.

[5] (U.S. Bureau of Economic Analysis, 1959-2017, (accessed July 11, 2018)), (Board of Governors of the Federal Reserve System (US), 1959-2017, (accessed July 11, 2018)a), (U.S. Bureau of Labor Statistics, 1959-2017, (accessed July 11, 2018)a), (U.S. Bureau of Labor Statistics, 1959-2017, (accessed July 11, 2018)b), (Board of Governors of the Federal Reserve System (US), 1959-2017, (accessed July 11, 2018)b), (Federal Reserve Bank of St. Louis, 1959-2017, (accessed July 11, 2018)).

[6] (Federal Deposit Insurance Corporation (U.S.), 1959-1980).

[7] (Board of Governors of the Federal Reserve System (U.S.), 1981-1984).

Financial Institutions Examination Council Annual Reports[8]. Since 1985 until 2008 these reports have yearly data each calendar midyear; we take the semi-sum of a year with the next one to obtain data for end December. Two advantages result from using net income form all commercial banks; the analysis can be done at aggregate sector level and be replicated at institution level and any intermediate grouping. Next we present briefly each variable.

Banking sector net income: *Income* is an indicator of the soundness of bank financial evolution and had been used to evaluate sensitiveness to adverse macroeconomic scenarios (Coffinet, Lin, & Martin, 2009).

GNP: We use *GNP* (Gross National Product) following the Saint Louis formulation, which is a standard measure used in empirical investigations (Hafer et al., 1982). GNP is a suitable measure in this case because it includes income regardless of whether it is earned by nationals within national borders or derived from foreign source. Banking net income also incorporates income derived from foreign sources.

M2: We adopt *M2* as money quantity measure since commercial banks operate with (deposits and some financial instruments) M2 to generate income. The statement that changes in the expansion of money quantity may be related to changes in banking income is equivalent to assert, following Milton Friedman, that the relevant variable is the deviation of anticipated growth rates of money quantity (De Vroey, 2016).

We also work with nominal variables as a change in money quantity does not necessarily derive in changes of prices or GNP. Changes of money velocity (Wicksell, 1978) may also occur, especially due to the large period and Δt we use. If money demand is not stable and undergoes unpredictable shifts, velocity cannot be forecasted and the quantity of money may not be tightly linked to aggregate spending (income) (Galí & Gertler, 2007). In particular, technological change in transactions (financial innovations) affects money velocity (Judd & Scadding, 1982). Substitutes for money are regularly emerging which may have an impact on velocity[9]; recently Lucas et al. incorporate money market deposit accounts in his definition of money (Lucas Jr & Nicolini, 2015).

Inflation: *Inflation* measures accumulation of aggregate price changes; higher (moderate) inflation may lower default rates of non-adjustable instruments[10].

Unemployment: *Unemployment rate* is one of key MV determining net banking income; e.g. higher unemployment leads to higher default risk (Foglia, 2008).

Interest rate: We employ a *short term interest rate*. Hall uses this kind of rate even for investment decision (Hall, Sims, Modigliani, & Brainard, 1977) considering the argument that the difference of a long-term interest rate is reflected in the short-term rate. This make possible to cover a broad market that use different interest rates.

Spread: The Moody's Seasoned Baa Corporate Bond Yield Relative to Yield on 10-Year Treasury Constant Maturity Spread is employed because of the con-

[8](Federal Financial Institution Examination Council (U.S.), 1985-2017).

[9]Agents may substitute instruments that have different velocity than the new ones.

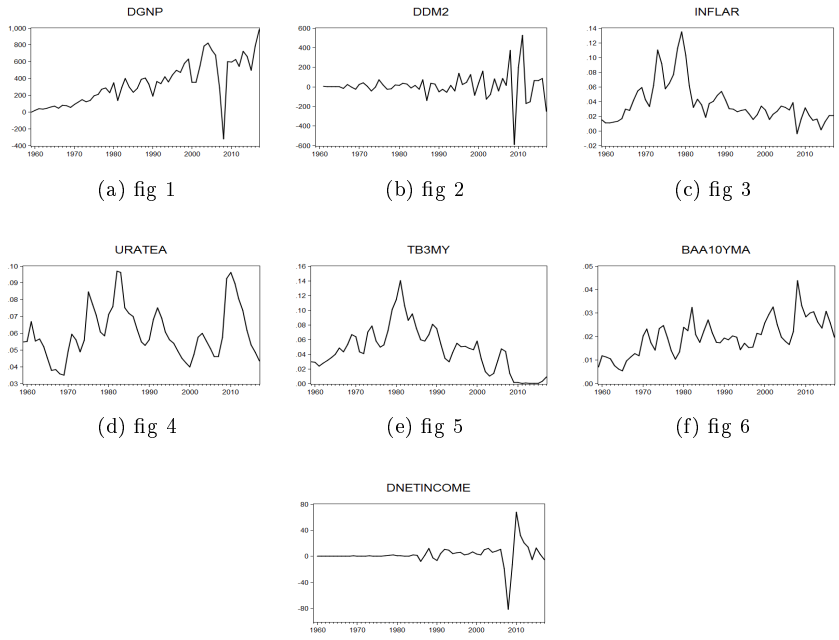
[10]Whereas high inflation may lead to the disappearance of long term loan markets (Heymann & Leijonhufvud, 1995).

nection of *spreads* to macroeconomics and financial markets; e.g., as leading indicator of investment and output declines (Gilchrist, Ortiz, & Zakrajsek, 2009).

For variables expressed as rate (other than inflation) we take an annual average over monthly data, as income of the interval (year) is influenced not just by the first or last (monthly) value of the involved variable. For variables quantified in money units, we do not use logarithmic transformations since there are large percentage changes involved and the Taylor approximation of the logarithmic function does not perform with acceptable errors (Nielsen, 2008).

2.1 Graphs

Figure 1 shows the time evolution of selected variables, where *dgnp* is used for the first difference of annual GNP, *ddm*2 represent the annual second difference of M2, $inflar$ stays for annual inflation, $tb3my$ is the annual average of the annualized 3 mouth Treasury Bill rate, *uratea* is the annual average of the unemployment rate, $baa10yma$ is the annual average of the spread between a 10 year corporate bond and 10-year Treasure Constant Maturity; *dnetincome* is the first difference of annual aggregate net commercial banking income[11].



(a) fig 1 (b) fig 2 (c) fig 3

(d) fig 4 (e) fig 5 (f) fig 6

(g) fig 7

Figure 1: Selected macro variables and net income for commercial banks. Billions USS dollar for dgnp, ddm2, dnetincome; rate for others

[11] For variables, upper and lower case are used indistinctly in this paper.

Variables *dnetincome* and *dgnp* are already first differences, in order to reach the stationarity of the variables which is discussed in section 3.

3 Stationarity

Tests of stationarity for all variables presented in subsection 2.1 have been realized. In particular a test for unit root that allows a structural break was selected for $inflar$ and $tb3my$. Results are presented in table 1.

Table 1: Unit Root tests

	Stationarity		$\mathbf{H_0}: \exists$ unit root
Variable	Structural Break	Linear Function with	p-value
DDM2	no	Intercept	0
BAA10YMA	no	Intercept	0.0032
URATEA	no	Intercept	0.0088
DGNP	no	Intercept and Trend	0.0004
INFLAR	Break Specification: Intercept and trend Break Type: Additive outlier Break Date: 1982	Intercept and Trend	< 0.01
TB3MY	Break Specification: Intercept and trend Break Type: Additive outlier Break Date: 1980	Intercept and Trend	< 0.01
DNETINCOME	no	Intercept	0.0122

Since the seminal work of Perron (Perron, 1989) the presence of structural breaks and it relation to unit root testing has been an active field of investigation (Perron, 2017). Casini et al. (Casini & Perron, 2018) provides an overview of methods that are of direct usefulness in practice. In our case this test is pertinent considering the rather long period involved and changes in the economic structure that occurred along the way, such as the abandonment of the gold standard in 1971, the financial liberalization of the 1980s and the great recession of 2008. For $ddm2$, $baa10yma$, $ureatea$, $dgnp$ and $dnetincome$ we use a standard augmented Dickey-Fuller test. For $inflar$ and $tb3my$ we perform an augmented Dickey-Fuller unit root test that allow a breaking point[12]. We use additive outlier due to the shock nature of the regime changing policies. Based on the results we detrended the first difference of GNP naming it $dgnpdt$ and removed intercepts and trend for inflation and T-bill rate calling the new variables $inflarss$ and $tb3myss$ respectively.

[12]Lag Length: 1, based on Schwarz information criterion. With minimized Dickey-Fuller t-statistic which select the date providing the most evidence against the null hypothesis of a unit root and in favour of the breaking trend alternative hypothesis.

4 An equation

The considerations of section 2 and 3 lead to a functional formulation of the form:

$$dnetincome = F(dgnpdt; ddm2; inflarss; uratea; baa10yma; tb3myss) \quad (4.1)$$

Let us expose a possible explanation of the relation between the variables to get insight of structural relations and links of the elements involved.

Broadly speaking banking income comes; first, from the spread between the interest rates that it receives on investments and those what it pays for resources. Secondly, from the revaluation of on and off-balance sheet positions. Therefore, banks income is affected by credit risk (related to counterpart default) and market risk (affecting on and off-balance sheet positions prices).

Suppose that in normal times interest rates are more correlated with market risk (TBill rates and spreads) as a change in interest rate affects more the price of long-term assets than short-term investments[13].

On the other hand, unemployment rate and GNP are more closely correlated with credit risk; lower GNP and higher unemployment tend to elevate probability and exposure to default (Hughes & Poi, 2015).

Macroeconomic theory suggest that a greater quantity of money lowers credit restrictions (Christiano & Eichenbaum, 1991). Moreover, an expansionary monetary policy tends to boost asset prices[14] and reduce market risk. Recently, Quantitative Easing is an example of this kind of policy, which basically involves central banks buying assets such as government securities (Japan) or agency debt and agency mortgage backed securities (United States). The European Central Bank develop this policy via Repo operations whose collateral are, in a substantial part, bank loans and not government bonds (Joyce, Miles, Scott, & Vayanos, 2012).

Moderate inflation rates tend to lower default rates. Asset prices (and therefore market risk) have been reported disconnected from inflation for periods (high asset price volatility and low inflation) (Bernanke & Gertler, 2000) and the Mundell-Tobin effect has little practical impact for moderates inflation[15] (Temple, 2000).

4.1 Structural break

The next step is to perform a regression of *dnetincome* as dependent variable using ordinary last squares (OLS)[16].

[13] A non-decreasing yield curve is a sufficient condition i.e., positive or null first maturity derivative in the relevant section of the curve.

[14] See (Mishkin, 2001) for various proposed mechanism.

[15] Higher capital purchases due to inflation boost by higher monetary growth rate that depresses real interest rate, increasing capital accumulation and the natural level of output.

[16] C represents constant in tables 2 and 3.

Table 2: Regression. Ordinary last squares. First data point: 1961 (both cases), last data points 1980 and 2017 respectively

Dependent Variable: DNETINCOME								
Sample: 1961 1980 Observations: 20					Sample: 1961 2017 Observations: 57			
Variable	Coefficient	Std. Error	t-Statistic	Prob.	Coefficient	Std. Error	t-Statistic	Prob.
C	0.64348	0.54516	1.180351	0.259	-14.28488	7.028658	-2.032377	0.0474
DGNPDT	0.007653	0.002502	3.058342	0.0092	0.057264	0.010326	5.545379	0
DDM2	0.001286	0.004886	0.263177	0.7965	0.029392	0.012127	2.423736	0.019
INFLARSS	17.78568	7.602974	2.339306	0.0359	148.295	122.5847	1.209734	0.2321
URATEA	-8.252758	11.82566	-0.697869	0.4976	323.3011	119.7064	2.700785	0.0094
TB3MYSS	3.313522	10.61741	0.312084	0.7599	-36.22344	116.6908	-0.310423	0.7575
BAA10YMA	7.474284	18.6316	0.401162	0.6948	-412.0978	242.5506	-1.699018	0.0955
	1980	2017			1980	2017		
R-squared	0.727827	0.506078		Mean dependent var	0.600313	2.631531		
Adjusted R-squared	0.602209	0.446808		S.D. dependent var	0.58071	16.05614		
S.E. of regression	0.366258	11.94206		Durbin-Watson stat	1.444284	1.660305		
Sum squared resid	1.743883	7130.641		Prob(F-statistic)	0.003954	0.000002		

A proportion of near 0.45 of the path of banking income is explained by the regression as the adjusted R^2 for the period 1961-2017 in table 2 indicates. That is consistent with the restrictions of section 1; in particular, that several variables are absent of the analysis such as operational factors of institutions[17] or banks dependencies on some economic sector[18]. The adjusted R^2 of near 0.6 for the period 1961-1980 is a hint that the relation between variables changes relevantly in time, being more stable for the reduced period 1961-1980.

The three tests performed (Chow, Quandt-Andrews and Bai–Perron) give 2007 as a time of a structural break, all tests with a p-value close to 0. Due to the presence of this structural break we include a slope changing dummy variable of *dgnp* from 2007 onwards in the regression of table 3 as a way to show the change of regime. The significant gain of near 0.2 in adjusted R^2 and a p-value of 0 for the slope coefficient of the dummy variable are indications of the nature of the changes in the individual path of the variables and of the changing nature of their relations.

4.2 Heterocedasticity

Due to the long period involved and possible changes in the volatility of the variables we also perform a regression using White heterocedastic consistent standard errors and covariance (WHC). Results are not substantially different of the obtained using OLS as seen in table 3 in terms of adjusted R^2 and F-statistics. The inclusion of the slope changing dummy of *dgnp* makes *dgnpdt* in the OLS specification not significant with a p-value of 0.064. In the WHC formulation the coefficient of *dgnpdt* is significant, with a p-value of 0.0272. This instability reflects the issue that controlling for heterocedastic errors is rel-

[17] For instance management appetite for risk or exposure to fraud.

[18] Some institutions have a greater exposure to the housing loan market whereas others may be more linked to financing information technology.

evant. More generally, the significance-changes of the coefficient of one variable (*dgnpdt*) due to the introduction of another (slope changing dummy of *dgnp*) is an indication of possible multicollinearity.

With the WHC method *ddm*2 and *dgnpdt* have a positive relation with *dnetincome*. When GNP growth is positive then growth of banking net income increases, when money quantity growth expands then growth of banking net income increases. Both are expected results; banking net income take part of economic growth and money quantity growth expansion per unit of time leads to it intermediary to enlarge his income growth.

Table 3: Regression with dummy variable. OLS and WHC

Dependent Variable: DNETINCOME								
Observations: 57								
OLS					WHC			
Variable	Coefficient	Std. Error	t-Statistic	Prob.	Coefficient	Std. Error	t-Statistic	Prob.
C	-13.40643	5.558188	-2.412014	0.0197	-13.40643	9.208048	-1.455947	0.1518
DGNPDT	0.019992	0.010555	1.894061	0.0641	0.019992	0.008782	2.276582	0.0272
DDM2	0.039039	0.009741	4.007633	0.0002	0.039039	0.012271	3.18129	0.0025
INFLARSS	144.9833	96.9015	1.496192	0.141	144.9833	117.1481	1.237606	0.2218
URATEA	185.873	97.78868	1.900762	0.0632	185.873	156.2847	1.189323	0.24
TB3MYSS	-120.1234	93.46263	-1.285256	0.2047	-120.1234	77.44155	-1.55115	0.1273
BAA10YMA	197.5502	220.7753	0.894802	0.3753	197.5502	186.8941	1.057016	0.2957
@YEAR>2006*DGNP	-83.95068	15.07314	-5.569554	0	-83.95068	10.67095	-7.867213	0
	OLS	WHC		OLS	WHC		WHC	
R-squared	0.697548	0.697548	Mean dependent var	2.631531	2.631531	Wald F-statistic	1764.214	
Adjusted R-squared	0.654341	0.654341	S.D. dependent var	16.05614	16.05614	Prob(Wald F-statistic)	0	
S.E. of regression	9.439852	9.439852	Durbin-Watson stat	2.112305	2.112305			
Sum squared resid	4366.429	4366.429	Prob(F-statistic)	0	0			

4.3 Multicollinearity

Data users like monetary authority, financial institutions and researchers need to determine which variable[19] drive the path of net banking income. For a structural analysis of the relationship of each independent variable with the depended one, multicollinearity brings an obstacle. In this case the standard error of individual parameters do not allow to (statistically) determine the form of the relationship.

The joint contribution of the MV is significant as the null value of the F statistic probability, the 2.11 of the Durbin Watson test and the 0.65 of adjusted R^2 indicates in table 3. Furthermore, except for *ddm*2 and *dgnpdt*, no significant (at 5 percent level) variable coefficient are obtained in WHC case. At the same time, the coefficient of the variable *uratea* switch sign in table 2 which can be seen as a further sign of the coefficients instability. Our findings are in accord with Kapinos et al. (Kapinos & Mitnik, 2016)[20] which point collinearity as a mayor issue in constructing top-down stress models and Papadopoulos

[19] In our case; money, GNP, inflation, unemployment, spread or interest rate (and the sign of the relationship).

[20] Related to Comprehensive Capital Analysis and Review (CCAR).

(Papadopoulos, Papadopoulos, & Sager, 2016) who point multicollinearity as a mayor problem to develop satellite models for risk assessment.

Excluding some variables of the regression while retaining near the same explanation power is a usual solution. But when the relation between variables changes in time this may be no more a helpful technique (parameters are time dependent). It is possible to argue that the dropped variables may develop his influence via those that remains in the equation. Even so, eliminating variables that do not have a statistically significant coefficient may lead to a missing information problem that can present itself in several ways. We describe briefly some of them. First, variables may be statistically significant depending on the time frame. $Inflarss$ in table 2 has a statistical significant coefficient depending on the time frame used. We will explore this time changing feature in section 5. Second, variations in the variable left in the equation may be erroneously attributed to changes in this variable and not to modifications in the excluded. In our framework, that can be the case of *uratea* with *dgnpdt* where changes of *uratea* or in the coefficient of *uratea* can be misinterpreted as changes of *dgnpdt*. Third, if the relation is of a feedback loop (mutual influence), or a network of variables with feedback loops, dropping one of the variable involved may involve missing relevant information over second round effects (Committee on the Global Financial System, 2005) and lead to an incomplete understanding of the dynamics of the relations (convergent, oscillatory or divergent).

5 Granger causality

Feedback effects between variables are relevant issues when modelling, in particular for bank stress testing (Basel Committee on Banking Supervision, 2009). To test for possible feedback loops we can use the Granger causality test and see if both variables are Granger causing the other. Furthermore, the relation between variables in such a long period may not be stable due to changes in: the structural framework of economic policies (i.e. inflation before and after 1982), conditions of production[21], proportion of production[22] or crisis as the of 1987 or 2008. We have seen in section 4 that this is statistically the case in the circumstance of the 2007-2008 crisis for equation 4.1. To account for such changes, the analysis is performed using a rolling-window Granger-causality test approach, based on a modified bootstrap estimation with a fixed window size[23] for various sample and lag sizes (Swanson, 1998).

The complete set of causal and feedback relation is exposed in appendix A[24].

[21] Regulatory changes, information technology (velocity of transmission and digital trading).

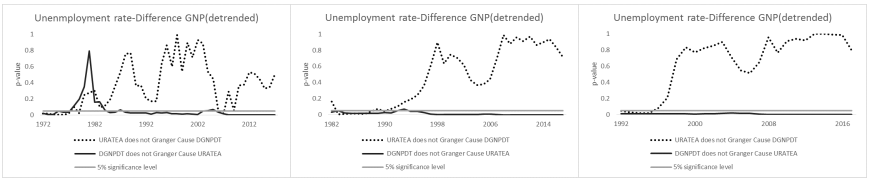
[22] Variation of banking non-interest earnings from 1985 onwards (Hoshi & Kashyap, 1999).

[23] Starting at n=1961, using j data points and continuing with n+1, n+2..., j fixed; see in appendix A a complete explanation. Lag refers to the included in the Granger causality test.

[24] In this section we want to highlight a few relevant relations. Year in graphs is the last year of each sample series. We identify periods with the last two digits of first and last years.

5.1 Granger causality: Macroeconomic variables

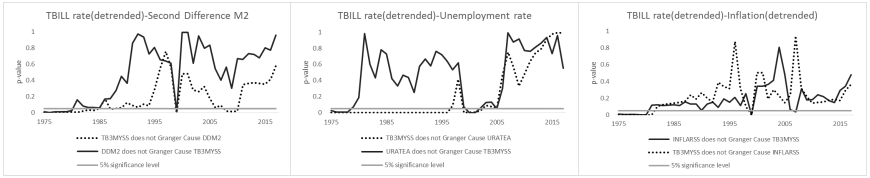
Next we use the example of figure 2 to analyse some features of the MV network.



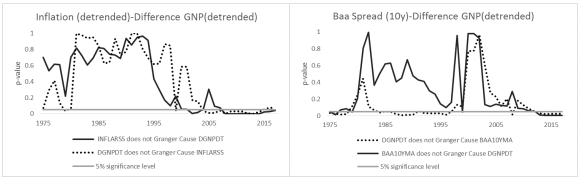
(a) fig 1 (b) fig 2 (c) fig 3

Figure 2: Unemployment Rate and Difference GNP (detrended), Sample series size of 12 (1 lag, 46 series), 22 (2 lags, 36 series), 32 (3 lags, 26 series)

Variability: a feature that the relations between the MV show is that they are not stable in the sense that their causality and feedback relation behave within certain limits. In figure 2 for the relation between *uratea* and *dgnpdt* we see, for different lag, sample size, and series quantity[25], different regimes. For one lag, *dgnpdt* causes almost everywhere *uratea* [26] whereas *uratea* causes *dgnpdt* during the seventies and in 2007. The other two examples show a relative stable causal relation from *dgnpdt* to *uratea* and feedback at the beginning of the series, although the starting points for each series are different, the 1980s for 2 lags and the 1990s for 3 lags.



(a) fig 1 (b) fig 2 (c) fig 3



(d) fig 4 (e) fig 5

Figure 3: Tbill rate (detrended), Unemployment Rate and Difference GNP (detrended), Inflation (detrended) and Baa Spread (10y). Sample series size of 15 (1 lag, 43 series)

[25] Due to the rolling windows approach.

[26] Maintained in the case of different lag and sample size specification. See appendix A tables 4,5,6,7 for the complete estimations.

Indirect causation: in figure 3 is shown that a network of feedback relations can be established between variables[27]. From a connection perspective, influence of one variable can go to another indirectly; those that do not have a direct Granger causal link may be statistically connected via other variables.

Time lag: from a time perspective, causality and feedback effects does not need to appear at the same moment, therefore a variable can influence other indirectly various lags later. In figure 3 we see that the feedback relation in 1999 between $tb3myss$ and $ddm2$ may influence $uratea$ in 00-01 because $tb3myss$ has a feedback relation with $uratea$ at that time, but $uratea$ influence of 00-01 may not pass to $ddm2$ because $tb3myss$ Granger cause $ddm2$ not until 2008. At the same time it will be more difficult to pass to the other variables the earlier 1970s influence of $ddm2$ over $tb3myss$ because of the time past until the mid 1990s.

Time density: during the earlier 1970s there is evidence of a network of feedback loops between the variables of sub-figure 1, 2, 3 and 5 and $dgnpdt$ causing $inflarss$ in sub-figure 4 (all of figure 3).

Time point connectivity: year 1999 seems to be an important date for interrelations; $baa10y$ causes $dgnpdt$, $dgnpdt$ causes $inflarss$, and $inflarss$ has feedback relation with $tb3myss$, whereas $tb3myss$ causes $uratea$ and has feedback relation with $ddm2$.

Causality direction: $uratea$ causes $tb3myss$ in 1996, with feedback relations of $tb3myss$ with $ddm2$ and $inflarss$ in 1999, $inflarss$ causes $dgnpdt$ in 02-03 and $dgnpdt$ causes $baa10y$ not until 2008.

The last two examples shows that this kind of analysis is useful to see causality direction.

Clustering: the feedback of $inflarss$ and $dgnpdt$ interact with the feedback of $dgnpdt$ and $baa10y$ during 13-14, but it is no link available in these relations that passes this cluster of relations to other variables. Crucial (at least in this concrete variable network formulation) is the link of $inflarss$ and $tb3myss$ in 2007 to pass the influence to $uratea$ and $ddm2$.

These examples show us that persistence is a critical condition for transmission, and that, the longer the separation between periods of Granger causality the less probable that a transmission may occur.

Transmitter: In the network of figure 3 $tb3mss$ occupies a transmitter position, the link of $inflarss$ to $tb3mys$ is one that allows transmissions in the 2000s. In the early 1970s the network of feedback connections is especially dense. One other important fact is that the network structure is time dependent, connections are constantly changing.

[27] For all characteristics, we restrict our attention to the relations of the variables presented in figure 3, for a complete network of relations see tables 5 and 6.

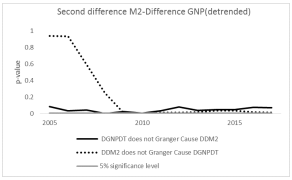
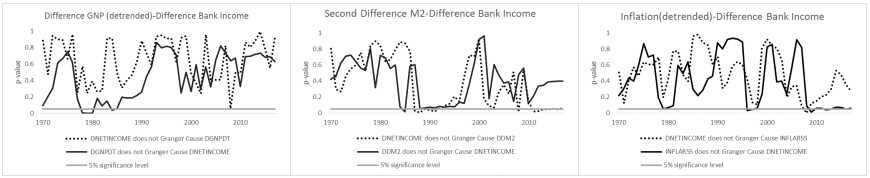


Figure 4: Granger causality between second difference of Money and detrended difference GNP, five lags

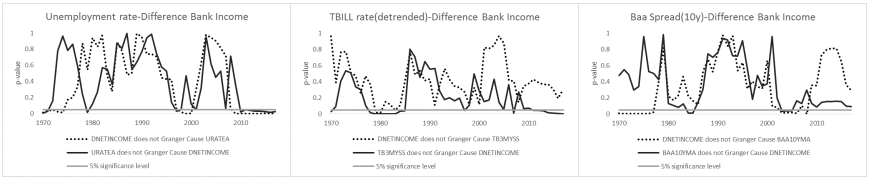
In figure 4 we see that one of the most studied relationships does not has feedback relation until five lags, but as we have already seen the causation may be transmitted via a chain network of other variables. Furthermore, this process of "long memory" may be an indication that influence of one variable to another may take some time to appear as suggested by numerous authors cited by Walsh in the first chapter (Walsh, 2017) for relations between interest rates, output and inflation.

This long memory may be a relevant indirect transmission mechanism. Net banking income may be affected (indirectly) several periods later through another variable even without any direct relationship.

5.2 Granger causality: Banking net income and macroeconomic variables



(a) fig 1 (b) fig 2 (c) fig 3

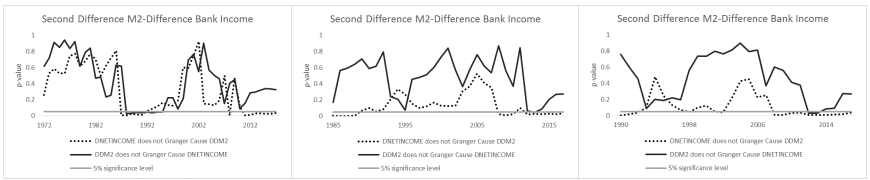


(d) fig 4 (e) fig 5 (f) fig 6

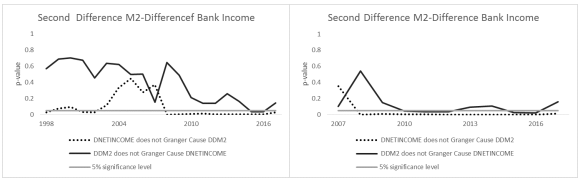
Figure 5: Granger causality one lag, series of 10

We see in figure 5 that the relations between MV and banking net income are also highly volatile. In all of them the range of variation of the $p - value$ cover almost the entire possible spectrum. As seen in subsection 5.1, the longer the separation between periods of Granger causality the less probable that a

transmission may occur. The network structure is time dependent; connections are constantly changing. Numbers of lag are relevant (see tables 5 and 6) and the (possibly changing) parameter value of each relation is also critical[28].



(a) fig 1 (b) fig 2 (c) fig 3



(d) fig 4 (e) fig 5

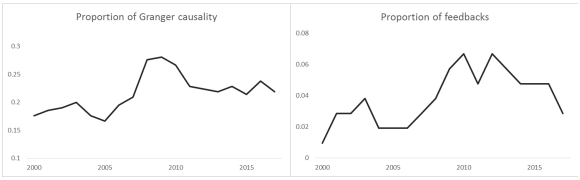
Figure 6: Granger causality between DDM2 and Dnetincome; 1 to 5 lags, series of 12, 25, 30, 38 and 47 observations respectively

Figure 6 shows the most persistent feedback relation. It is only absent in 1 lag, 10 sample series size and 2 lag, 20 and 22 sample series size (see table 6). The importance of this relation is that it can pass on all influence of the other MV and also allows to transfer shocks of the banking sector to the macroeconomic structure. This relation can be explained (given the sign in tables 2 and 3) as that an increasing growth of money quantity tends to boost income of bank in two ways; it contributes to less restrictive conditions in the credit market and provides liquidity to the market which boost asset prices. Both tent to raise banking income by elevating income from credits (more money is available to lend; liquidity effect) and higher asset prices giving the higher liquidity.

5.3 Granger causality: Evidence of increased relations

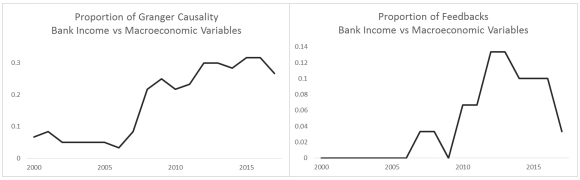
The data shows that an increase in feedback relations can not be rejected at five percent level of significance when the last data points of the series are situated around 2008.

[28]Not treated in this work, in general the greater this parameter the more influence in a lagged regression.



(a) fig 1 (b) fig 2

Figure 7: Proportion of Granger causality 1 to 5 lags, 18 series of 40 observations, time axis shows last year of series, all variables



(a) fig 1 (b) fig 2

Figure 8: Proportion of Granger causality 1 to 5 lags, 18 series of 40 observations, time axis shows last year of series, only bank net income vs macroeconomic variables

The increments of causality and feedback relations hold when considering banking net income in pairwise relation to MV. A word of caution is pertinent since this relation is not necessary to hold for the existence of transmission effects. The transmission network may operate with only one link (if the other variables are connected), such as the relation of the second difference of M2 and banking net income in figure 6.

This changing relations in the network of variables highlights the fact that the study of emergent properties of the network structure should be relevant. The interrelation of the variables give rise to different network structures[29] for each time point as we can see from the examples of subsections 5.1 and 5.2 and confirm in figures 7 and 8. Each configuration of the variables network may give rise to different output results, different dynamic evolution for individual variables and group of variables.

This changing nature of relations need also be considered for forecasting. The structure of the network determines the value of the dependent variable as the value of the individual independent variable do. A variable that is free from the Lucas critique (and do not change it parameter) can be influenced by another variable that is affected by the critique. Suppose that *dgnpdt*, is

[29]Structure determined by: existence of connections (in the sense that one variable cause other), when connected if there are feedback or one way, time persistence of connections and presence of indirect connections.

free from the Lucas critique[30]. But *uratea* is not and cause *dgnpdt*[31] as seen in subsection 5.1; as a result the system of independent variables is subject to the Lucas critique. This fact may be difficult to grasp if *dgnpdt* remains in a regression with reduced quantity of explanatory variables and *uratea* not[32].

6 Concluding remarks

We have explored the relations between banking net income and MV using US data with standard tools. Employing a linear regression we could not reject the presence of multicollinearity in section 4. This was a strong indication that the variables have relevant relations between them. To have a more exhaustive insight we performed a Granger causality study in section 5. Given the presence of structural breaks we also performed the analysis with a rolling windows approach which lead us to not reject a changing relationship between the variables and to have indications of an augmented causal and feedback relation near the 2008 crisis in subsection 5.3.

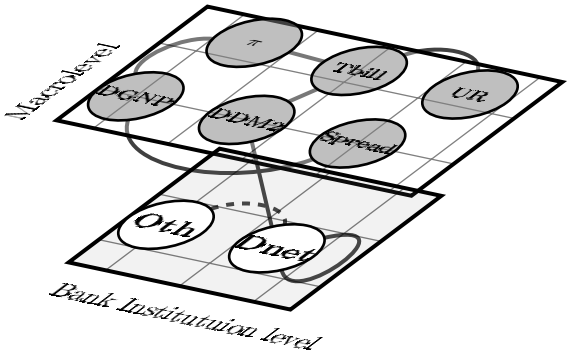


Figure 9: Network of variables

Let us recapitulate what the consequences of increased causality and feedback relations imply. A central argument of this paper is that the augmented interrelation between variables generates a network of (often mutual) influences. When all variables are interrelated, all influence each other (maybe indirectly through other variables). This effect makes difficult to separate linear effects of one variable on the system[33] of other variable movements (because those relations are permanently changing). The longer the memory of a variable, the greater the capacity to exercise as bridge of indirect causation (because it retains and caries on the action of other variables that have influenced it).

[30] One argument to support the idea can be that the trend observed for *dgnp* represent influence of economic policies, once detrended the variable may be free of policy influence.

[31] For concrete periods of causation see A tables 4,5,6,7.

[32] By the argument that *dgnpdt* has a significant parameter and *uratea* not in the WHC regression of subsection 3.

[33] Or on other variable.

We can report that a substantial feedback relation at five percent significance level exist between *ddm*2 and *dnetincome*, and this may be the only channel needed to canalize influence of the MV.

As we see[34] in figure 9 when the system of MV is connected, then one link to bank institution level suffices to expose banks income to all the consequences of dynamic linked MV.

The interconnection between MV and banking income is a well-established standard theory and almost trivial for most economist (Foglia et al., 2011); but some aspects of this interrelation may provide necessary and useful information for the understanding of the connections as seen in subsection 5.2.

First, the time persistence-duration of the connection between net banking income and the MV is relevant to see the possibility of transmissions. Second, the quantity variation of Granger causal connections at each point of time has systemic effects[35]. Third, understanding the emergent properties of the particular network has consequences for its development that can not be captured studying the variables individually or looking at the aggregate consequences of its interactions. Fourth, for forecasting, the trajectories of the relations of the variables are also needed to take in account. If a great proportion of variables are having feedback relations at the same time (or near the same time) the outcome of the dependent variable may be very different (see the increment of Granger causal relations near 2008) of those when these relations are absent. In terms of practical consequences this may imply a necessity for more simulated dynamic paths for the system that take different network structures in account.

An implication of this study is that theories that relies on only a reduced vector of variables may be incomplete, and in crisis situation probably wrong. In section 4 we have set out some relations that relates variables to certain risks. For example, unemployment to credit risk or spreads to market risks. The interactions of variables is a hint that those risks may be also correlated and the variables may influence banking net income not only via one type of risk but via the two mentioned in section 4. And second round effect may be also relevant enough as stated in subsection 4.3 and proved statistically in section 5[36]. The study of interactions of variables is a promising subject and may build bridges between theoretical postures.

[34] Where π represents inflation, Tbill the three months T-bill interest rate, UR the unemployment rate, DGNP Gross National Product, DDM2 the second difference of money quantity, Spread represent the spread between a Treasury and a Corporate Bond, Dnet is banking net income and Oth are other variables that influence banking net income at sector and institution level. Lines represent feedback connections. Figure 9 shows a possible configuration of connections.

[35] Especially the increase of these connections during the last crisis may have systemic consequences.

[36] Lower acceleration of money quantity may lead to lower banking net income, lower banking income to lower acceleration of money quantity and so fort: with the possible transmission effects discussed at length in section 5 and especially in subsection 5.1.

A Granger causality tables

In the following tables periods are indicated with dashes and separated with commas, years are identified by it last two numbers. Table 4 shows Granger causal relations considering entire series for different lags. Lag refers to the included in the Granger causality test. The letter n indicates absence in tables 5 and 6.

Given the presence of a structural break we used a rolling windows approach to see the evolution of Granger causal relations. Taking sub-samples of different size j beginning with date n (1961 in our case for all tests) for each variable par and performing the test for the first j and later beginning at n+1, n+2 and so on maintaining fixed size j until the last data point is included (year 2017 in our case). We use size j= 10, 12, 15, for one lag; 20, 22, 25 for two lags; 30, 32 for three lags, 38 for four lags, 45 and 47 for five lags. Size 40 is used for all lags. For example, when j= 10 then the first sub-sample takes from 1961 to 1970 and the second from 1962 to 1971.

Table 5 displays unilateral Granger causality relations, whereas table 6 presents Granger feedback causality relations. Table 7 give the proportion of years when causal relation exists (both unilateral and feedback). In all this tables we can distinguish various regime-types. Those who have (almost) no Granger causal relation at all such as *uretea* causing *dgnpdt*, those who have sometimes these relations (more commonly with few lags) such as $inflarss$ and $baa10my$ (both ways), those who's relation is persistent, *dgnpdt* with *dnetincome* (both ways), and those who are (almost) always connected such as $ddm2$ with *dnentincome* (both ways).

Table 4: Granger causality test for 1 to 5 lags, entire series

Pairwise Granger Causality Tests															
Sample: 1961 2017	Lags: 1			Lags: 2			Lags: 3			Lags: 4			Lags: 5		
Null Hypothesis:	Obs	F-Statistic	Prob.	Obs	F-Statistic	Prob.	Obs	F-Statistic	Prob.	Obs	F-Statistic	Prob.	Obs	F-Statistic	Prob.
DDM2 does not Granger Cause BAA10YMA	56	0.42432	0.5176	55	0.22023	0.8031	54	0.20309	0.8937	53	1.16807	0.3379	52	1.38744	0.249
BAA10YMA does not Granger Cause DDM2		1.78036	0.1878		0.51735	0.5993		0.98996	0.4057		0.78823	0.5391		0.54175	0.7435
DGNPDT does not Granger Cause BAA10YMA	57	0.5765	0.451	57	4.53409	0.0153	56	2.13752	0.1075	55	1.93784	0.1201	54	1.99462	0.0987
BAA10YMA does not Granger Cause DGNPDT		10.6277	0.0019		4.77282	0.0125		3.24916	0.0296		2.30556	0.0724		2.07007	0.0878
URATEA does not Granger Cause BAA10YMA	57	2.90394	0.0941	57	1.50944	0.2306	56	0.95993	0.4192	55	0.63304	0.6415	54	0.43643	0.8206
BAA10YMA does not Granger Cause URATEA		10.0992	0.0025		9.22999	0.0004		6.07123	0.0013		4.02444	0.007		3.03129	0.0198
TB3MYSS does not Granger Cause BAA10YMA	57	27.3637	0.000003	57	14.8135	0.000008	56	9.10511	0.00007	55	6.40241	0.0004	54	4.71829	0.0016
BAA10YMA does not Granger Cause TB3MYSS		4.41334	0.0403		0.73427	0.4848		1.04973	0.379		0.6867	0.6048		0.51559	0.763
INFLARSS does not Granger Cause BAA10YMA	57	3.48788	0.0673	57	3.857	0.0274	56	2.47503	0.0725	55	1.8822	0.1296	54	1.4483	0.2267
BAA10YMA does not Granger Cause INFLARSS		0.1535	0.6968		0.86556	0.4268		0.53631	0.6596		0.86421	0.4926		0.61323	0.6903
DNETINCOME does not Granger Cause BAA10YMA	57	0.0697	0.7928	56	0.38895	0.6798	55	0.12387	0.9456	54	0.20862	0.9323	53	0.46186	0.8023
BAA10YMA does not Granger Cause DNETINCOME		3.3439	0.073		3.41821	0.0404		2.14171	0.1072		1.71312	0.1637		1.7554	0.1431
DGNPDT does not Granger Cause DDM2	56	4.55421	0.0375	55	3.04034	0.0567	54	4.51579	0.0073	53	3.23163	0.0207	52	2.65872	0.0359
DDM2 does not Granger Cause DGNPDT		0.30665	0.5821		1.36964	0.2636		3.10935	0.0352		3.23893	0.0205		4.50623	0.0023
URATEA does not Granger Cause DDM2	56	0.3607	0.5507	55	0.35421	0.7035	54	0.507	0.6794	53	0.3854	0.8179	52	0.31947	0.8984
DDM2 does not Granger Cause URATEA		0.53397	0.4682		1.44944	0.2444		1.37121	0.2631		1.00059	0.4175		0.81729	0.5444
TB3MYSS does not Granger Cause DDM2	56	1.62651	0.2077	55	1.86097	0.1661	54	1.49934	0.227	53	1.03551	0.3997	52	0.92977	0.4717
DDM2 does not Granger Cause TB3MYSS		0.17235	0.6797		0.22814	0.7968		0.15822	0.9239		0.0688	0.991		0.07967	0.995
INFLARSS does not Granger Cause DDM2	56	2.00596	0.1625	55	0.41145	0.6649	54	0.33547	0.7997	53	0.22529	0.9228	52	0.29933	0.9104
DDM2 does not Granger Cause INFLARSS		0.43054	0.5146		0.26079	0.7715		0.18935	0.9031		0.39305	0.8125		0.31874	0.8989
DNETINCOME does not Granger Cause DDM2	56	28.2797	0.000002	55	10.7053	0.0001	54	6.82774	0.0007	53	4.81767	0.0026	52	3.85102	0.0059
DDM2 does not Granger Cause DNETINCOME		5.3501	0.0246		3.65989	0.0329		2.75764	0.0526		2.94751	0.0305		1.982	0.1017
URATEA does not Granger Cause DGNPDT	57	1.36946	0.247	57	0.35518	0.7027	56	0.22209	0.8806	55	0.18044	0.9474	54	0.1799	0.9687
DGNPDT does not Granger Cause URATEA		31.0284	0.0000008		13.9072	0.00001		9.32668	0.00006		7.48568	0.0001		5.94895	0.0003
TB3MYSS does not Granger Cause DGNPDT	57	5.30983	0.0251	57	1.84887	0.1676	56	1.26935	0.2952	55	0.92773	0.4562	54	0.86124	0.5148
DGNPDT does not Granger Cause TB3MYSS		11.9424	0.0011		2.85032	0.0669		1.8583	0.149		1.31239	0.2795		1.02662	0.4141
INFLARSS does not Granger Cause DGNPDT	57	3.55939	0.0646	57	1.60368	0.2109	56	1.94125	0.1352	55	1.31623	0.2781	54	1.42942	0.2331
DGNPDT does not Granger Cause INFLARSS		0.96578	0.3301		0.12236	0.8851		0.1934	0.9004		0.10373	0.9806		0.17651	0.97
DNETINCOME does not Granger Cause DGNPDT	57	0.10668	0.7452	56	0.05301	0.9484	55	0.51325	0.6751	54	0.47739	0.7521	53	0.31385	0.9019
DGNPDT does not Granger Cause DNETINCOME		0.85648	0.3588		7.1652	0.0018		4.91957	0.0046		4.98266	0.0021		3.91405	0.0053
TB3MYSS does not Granger Cause URATEA	57	15.935	0.0002	57	8.23699	0.0008	56	5.36938	0.0028	55	4.26703	0.0051	54	3.28111	0.0135
URATEA does not Granger Cause TB3MYSS		1.47142	0.2304		0.19544	0.8231		0.13994	0.9356		0.13072	0.9704		0.10126	0.9913
INFLARSS does not Granger Cause URATEA	57	0.01793	0.894	57	8.23789	0.0008	56	5.09674	0.0038	55	4.4714	0.0039	54	3.31271	0.0128
URATEA does not Granger Cause INFLARSS		0.04619	0.8306		0.69646	0.5029		0.33186	0.8023		0.24116	0.9135		0.53652	0.7474
DNETINCOME does not Granger Cause URATEA	57	14.3353	0.0004	56	4.33433	0.0183	55	3.47421	0.023	54	2.60849	0.048	53	2.44501	0.0494
URATEA does not Granger Cause DNETINCOME		6.53945	0.0134		2.30772	0.1098		1.6452	0.1914		0.71335	0.5872		0.70429	0.6234
INFLARSS does not Granger Cause TB3MYSS	57	8.46456	0.0052	57	1.87539	0.1635	56	1.79674	0.1601	55	2.07325	0.0997	54	1.64752	0.1681
TB3MYSS does not Granger Cause INFLARSS		7.1149	0.0101		3.79779	0.0289		2.2611	0.093		1.48179	0.2232		1.3447	0.264
DNETINCOME does not Granger Cause TB3MYSS	57	1.38179	0.245	56	0.00625	0.9938	55	0.03894	0.9896	54	0.06124	0.9928	53	0.14327	0.981
TB3MYSS does not Granger Cause DNETINCOME		7.75714	0.0074		3.79698	0.029		2.58667	0.0639		1.60893	0.1885		1.27195	0.294
DNETINCOME does not Granger Cause INFLARSS	57	0.02032	0.8872	56	0.23188	0.7939	55	0.1683	0.9173	54	0.42399	0.7905	53	0.3159	0.9006
INFLARSS does not Granger Cause DNETINCOME		1.83873	0.1807		1.17088	0.3183		1.28735	0.2894		1.50576	0.2165		1.24873	0.3039

Table 5: Years of causality relation, of series with endpoint at the year of column

Pairwise Granger Causality Tests	lag 1	lag 1	lag 1	lag 1	lag 2	lag 2	lag 2	lag 2	lag 3	lag 3	lag 3	lag 4	lag 4	lag 5	lag 5	lag 5
Quantity of series	48	46	43	18	38	36	33	18	28	26	18	20	18	18	13	11
Sapmle size for each serie	10	12	15	40	20	22	25	40	30	32	40	38	40	40	45	47
Null Hypothesis:																
DDM2 does Granger Cause BAA10YMA	74–76	74–77	75–78	n	n	n	87	n	n	n	n	n	n	n	n	n
BAA10YMA does Granger Cause DDM2	96–00,10	97–00,06,09–10	98,00–01,07,09–10	09–10	80,82–86,10	82–86,09–10	09–10	10	10	n	n	n	n	n	n	n
DGNPDT does Granger Cause BAA10YMA	72–73,77–80,83,86–90,06–07	72–73,77–78,85–90,08	77–78,84,86–90,92–97,08,12	08	81–02,08	82–04,08	85–08	00–02,04–09	90–10	92–10	00–10	98–11	00–11	00–11	05–11	07–11
BAA10YMA does Granger Cause DGNPDT	75–76,91–92,10–13,15–16	76,94,12–17	79,99	03–07,10–17	n	n	n	n	n	n	n	n	n	n	n	n
URATEA does Granger Cause BAA10YMA	72,79	72–73,81,03	02–05	07–08	06–07	n	n	n	n	n	n	n	n	n	n	n
BAA10YMA does Granger Cause URATEA	71,83–86,02,09–17	73–78,84–86,08–17	75,79,99,10–17	00,04,11–17	80–82,84–85,11–17	82–86,11–17	85–86,09–17	00,08–17	10–17	09–17	09–17	09–17	09–17	14–15,17	09,15,17	09–12,17
TB3MYSS does Granger Cause BAA10YMA	70–75,81–87,89	73–75,81–93,03	75,81–96,02–06	00	80–01,03–17	82–03,05–17	85–06,08–17	00–17	90–11,13–17	92–13,15–17	00–17	98–17	00–17	00–17	05–17	07–17
BAA10YMA does Granger Cause TB3MYSS	04,13–15	06,16	17	n	n	n	n	n	n	n	n	n	n	n	n	n
INFLARSS does Granger Cause BAA10YMA	74–79,89–93	74–80,90–93	75–80,93,95–96,99	n	80–81,83,00–02,12–13	83–85,02–04,12–14	85,87,05–07	00–04	n	n	n	n	n	02–04	05–06	n
BAA10YMA does Granger Cause INFLARSS	82,09	82	82	n	91–04	92–94,08	95	06–08	07–08	n	08	06–08	06–08	07–08	n	n
DNETINCOME does Granger Cause BAA10YMA	70–77,06,08	72–77,03,06,08	75–77,06–08	08	80–81,08	83,86–87,08	86–87,08	08	n	n	n	08	08	08	08	08
BAA10YMA does Granger Cause DNETINCOME	84–85	84	85	12–13	84–85	n	n	13,15–16	n	n	n	n	n	n	n	n
DGNPDT does Granger Cause DDM2	71–74	72–74	77	10–16	02–08,10	02–04,06–10	01–04,06–10	01–10	06–10	04,06–10	03–04,06–10,13–17	08,10–11,13–14	08,10–11,13–14	04,06,08	06–08	07–08
DDM2 does Granger Cause DGNPDT	00	02	n	02	n	n	n	n	03	n	n	n	n	11–12,16–17	12,14,16–17	12,16–17
URATEA does Granger Cause DDM2	n	n	n	n	85,87–00,04–07	85–90,06–07	85–91	n	90	n	n	n	n	n	n	n
DDM2 does Granger Cause URATEA	86,09	09	09	09	81,11–17	11–17	11–17	16	12–17	14–17	n	n	n	n	n	n
TB3MYSS does Granger Cause DDM2	73,81,87–88,08–09	73,81–82,88,08–10	81–84,87,08–10	02–03,05–10	81–82,84,86–90,05–07,09,11	82–91,06–13	85–91,94,07–11,15–16	08–11	90–91,94,06–07,09–11	94,06,09–11	06–07,09–10	09–10	09–10	n	09	09
DDM2 does Granger Cause TB3MYSS	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n
INFLARSS does Granger Cause DDM2	08–17	10–17	09–17	n	80–91,10–17	82–91,10–17	85–91,12–17	n	90–91	n	n	n	n	n	n	n
DDM2 does Granger Cause INFLARSS	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n
DNETINCOME does Granger Cause DDM2	87–91,08,11–14	87,08,11–17	87,08,11–17	00–01,09,14–17	82,85–88,08–10,12,16	82–88,08–10,12–17	85–88,08–10,12,14–17	08–11,15–17	90–92,98,02,08–11,14–17	92,98,08–11,14–17	01–02,08–11,16–17	98,01–02,08–14,17	01–02,08–14,17	08–15,17	08–09,12–14,17	08–09,13–14,17
DDM2 does Granger Cause DNETINCOME	85	92–95	93–95	n	n	n	n	n	n	n	n	n	n	n	n	n
URATEA does Granger Cause DGNPDT	76	79	78–79,82–84,99	n	81	83	n	n	n	n	n	n	n	n	n	n
DGNPDT does Granger Cause URATEA	70–71,84–86,89–91,93–94,97–00,02–03,08–17	85–86,88–03,06,08–17	88–98,00–08,10–17	00–17	82,88–92,94–17	82,89–91,94–17	95–17	00–17	90–91,96–17	96–17	00–17	98–03,06–17	00–03,05–17	00,06–17	05–17	07–17
TB3MYSS does Granger Cause DGNPDT	95,04,09–13,16	n	n	n	80,13–17	15–17	11–15	n	n	n	n	n	n	n	n	n
DGNPDT does Granger Cause TB3MYSS	81–92,00,02	81–83,85–94,97,00–03,17	81–98,00–07	00–07	81–03,05–08	82–08	85–08	00–08	90–08	92–03,05–08	00–03,06–08	00–03,08	00–03	00–08	05–08	07–08
INFLARSS does Granger Cause DGNPDT	71–72,98–99,01–02,14	72–73,98–02,14	02–03,15–17	14	80,84–95,99–00,14	96,02,09–10,14–16	09–16	n	12–17	12,14–15,17	n	n	n	n	n	n
DGNPDT does Granger Cause INFLARSS	74,05	74,04–06	79,99,04–07	n	08	08	08	n	n	n	n	n	n	n	n	n
DNETINCOME does Granger Cause DGNPDT	n	08	08	08	n	n	n	n	08	08–09	03–06,09	08–09	08–09	08–09	08–09	08–09
DGNPDT does Granger Cause DNETINCOME	78–80,84	78–80,82,84–85	78–80,82,84–85,99	n	80–85,12–14	82–85,12–16	85,90–91,12–17	12–17	07,12–17	07,12–17	12–17	98–00,14–17	00,03–04,14–17	00–05,14–17	05,14–17	14–17
TB3MYSS does Granger Cause URATEA	70,74–76,79–91	74–75,79–93,99	79–96,99,02	00–17	81–01,04–05,08,10–17	82–03,10–17	85–06,10–17	00–17	90–09,14–17	92–11,16–17	00–17	98–13,15	00–15	00–09,11	05–17	07–17
URATEA does Granger Cause TB3MYSS	94	96	05	n	06	07	n	n	n	n	n	n	n	n	n	n
INFLARSS does Granger Cause URATEA	90–91,09–14	92–93,10–14	95–96,12–14	n	80–83,00–01,03–17	82–83,02–03,05–17	85–92,04–06,08–17	00–14,16–17	90–17	92–17	00–12,14,16–17	98–08,14–17	00–08,16–17	00–06,16–17	05–11	07–13
URATEA does Granger Cause INFLARSS	n	n	n	n	91–96	93–95	96	n	n	n	n	n	n	n	n	n
DNETINCOME does Granger Cause URATEA	72–74,99–00,09	74,99–02,09,12	75–76,02–05,08–09	09	07–17	85,08–17	85,09–17	09–17	09–17	09–17	09–17	09–17	09–12,15–17	09–17	09–17	09–17
URATEA does Granger Cause DNETINCOME	79,97	n	n	n	84	n	n	n	n	n	n	n	n	n	n	n
INFLARSS does Granger Cause TB3MYSS	71–72	72,07	07	13–17	80,01–03	03–05	06–07	n	n	13	n	n	n	n	n	n
TB3MYSS does Granger Cause INFLARSS	81,96–97	81	81	n	83–94	83–84,86–94	85,88–94	05–14,16–17	90–93	92–93	08	n	n	n	n	n
DNETINCOME does Granger Cause TB3MYSS	n	00	n	n	n	n	n	n	n	n	n	n	n	n	n	n
TB3MYSS does Granger Cause DNETINCOME	70,78,81–82,84–85,97,07,11–17	78–81,84–85,99,07,09–17	77–82,85,99,02,07,09–17	07,09–17	80–83,07,09–11,14–17	82–83,10–11,16–17	07,09–10,14,16–17	07,09,11	07	07	05–07	n	n	00–01	n	n
DNETINCOME does Granger Cause INFLARSS	08	n	n	n	08	08	08	n	n	n	n	n	n	n	n	n
INFLARSS does not Granger Cause DNETINCOME	79,96–97,09,12,16	00,09–14,17	02–03,09–17	n	80–81,10–17	10,12–17	12–17	n	17	n	n	n	n	n	n	n

Table 6: Years of feedback relation, series with endpoint at the years of column

Pairwise Granger Causality Tests	lag 1	lag 1	lag 1	lag 1	lag 2	lag 2	lag 2	lag 2	lag 3	lag 3	lag 3	lag 4	lag 4	lag 5	lag 5	lag 5
Mutual Influence																
Feedback																
Quantity of series	48	46	43	18	38	36	33	18	28	26	18	20	18	18	13	11
Sapmle size for each serie	10	12	15	40	20	22	25	40	30	32	40	38	40	40	45	47
DDM2-BAA10YMA	n	n	n	n	81	n	85-86	n	n	n	n	n	n	n	n	n
DGNPDT-BAA10YMA	08–09,17	93,09–11	75–76,13–17	09	80,17	n	n	03	n	n	n	n	n	n	n	n
URATEA-BAA10YMA	03–07	04–07	76–78,06–09	01-03,09–10	09–10	09–10	n	n	n	n	n	n	n	n	n	n
TB3MYSS-BAA10YMA	76–80,03,05–12	72,76–80,04-05,07–15	76–80,99,07–16	01–17	n	n	n	n	n	n	n	n	n	n	n	n
INFLARSS-BAA10YMA	80–81	81	81	n	n	n	n	n	n	n	n	n	n	n	n	n
DNETINCOME-BAA10YMA	03–05	04–05	n	n	n	84–85	85	n	n	n	n	n	n	n	n	n
DGNPDT-DDM2	n	n	n	n	n	n	n	n	n	n	n	n	n	09–10	09–11,13,15	09-11,13-15
URATEA-DDM2	10	10	10	n	09–10	09–10	09–10	n	n	n	n	n	n	n	n	n
TB3MYSS-DDM2	74–80	74–80	75–80,99	n	10	n	n	n	n	n	n	n	n	n	n	n
INFLARSS-DDM2	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n
DNETINCOME-DDM2	n	88–91	88–92	10–13	n	n	13	12–14	12–13	12–13	12–15	15–16	15–16	16	10–11,15–16	10–12,15–16
URATEA-DGNPDT	72–75,05–07	72–77,07	75–77,09	n	83–87	84–88,90	85–94	n	92–95	92–95	n	n	n	n	n	n
TB3MYSS-DGNPDT	93–94,05–08,14–15	95,06–16	08–17	08–17	n	n	n	n	n	n	n	n	n	n	n	n
INFLARSS-DGNPDT	73,08–13	08–13	08–14	n	09–13	11–13	n	n	n	n	n	n	n	n	n	n
DNETINCOME-DGNPDT	n	n	n	n	n	n	n	n	90–91	n	07–08	n	n	n	n	n
TB3MYSS-URATEA	71–73,77–78,95–01	72–73,76–78,97-01	75–78,00–01	n	80,04–05	06,08	09	n	n	n	n	n	n	n	n	n
INFLARSS-URATEA	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n
DNETINCOME-URATEA	70–71,98,10–17	72,10–11,13–17	10–17	10–17	n	84	n	n	n	n	n	n	n	n	n	n
TB3MYSS-INFLARSS	73–80	73–80	75–80,99	00-12	n	n	n	n	n	n	n	n	n	n	n	n
DNETINCOME-TB3MYSS	79–80,83	82–83	83–84	n	n	n	n	n	n	n	n	n	n	n	n	n
DNETINCOME-INFLARSS	n	08	08	n	n	n	n	n	n	n	n	n	n	n	n	n

Table 7: Proportion of causality respect to quantity of series for each lag and sample size combination

Proportion of Granger causality	lag 1	lag 1	lag 1	lag 1	lag 2	lag 2	lag 2	lag 2	lag 3	lag 3	lag 3	lag 4	lag 4	lag 5	lag 5	lag 5
Quantity of series	48	46	43	18	38	36	33	18	28	26	18	20	18	18	13	11
Sample size for each serie	10	12	15	40	20	22	25	40	30	32	40	38	40	40	45	47
DDM2 does Granger Cause BAA10YMA	1/16	2/23	4/43	0	1/38	0	1/11	0	0	0	0	0	0	0	0	0
BAA10YMA does Granger Cause DDM2	1/8	7/46	6/43	1/9	4/19	7/36	4/33	1/18	1/28	0	0	0	0	0	0	0
DGNPDT does Granger Cause BAA10YMA	17/48	15/46	23/43	1/9	25/38	2/3	8/11	5/9	3/4	19/26	11/18	7/10	2/3	2/3	7/13	5/11
BAA10YMA does Granger Cause DGNPDT	13/48	6/23	9/43	7/9	1/19	0	0	1/18	0	0	0	0	0	0	0	0
URATEA does Granger Cause BAA10YMA	7/48	4/23	11/43	7/18	2/19	1/18	0	0	0	0	0	0	0	0	0	0
BAA10YMA does Granger Cause URATEA	5/12	21/46	18/43	7/9	7/19	7/18	1/3	11/18	2/7	9/26	1/2	9/20	1/2	1/6	3/13	5/11
TB3MYSS does Granger Cause BAA10YMA	7/12	17/23	38/43	1	37/38	35/36	32/33	1	27/28	25/26	1	1	1	1	1	1
BAA10YMA does Granger Cause TB3MYSS	3/8	19/46	17/43	17/18	0	0	0	0	0	0	0	0	0	0	0	0
INFLARSS does Granger Cause BAA10YMA	13/48	6/23	11/43	0	4/19	1/4	5/33	5/18	0	0	0	0	0	1/6	2/13	0
BAA10YMA does Granger Cause INFLARSS	1/12	1/23	2/43	0	2/19	1/9	1/33	1/6	1/14	0	1/18	3/20	1/6	1/9	0	0
DNETINCOME does Granger Cause BAA10YMA	13/48	11/46	6/43	1/18	3/38	5/36	4/33	1/18	0	0	0	1/20	1/18	1/18	1/13	1/11
BAA10YMA does Granger Cause DNETINCOME	5/48	3/46	1/43	1/9	1/19	1/18	1/33	1/6	0	0	0	0	0	0	0	0
DGNPDT does Granger Cause DDM2	1/12	3/46	1/43	7/18	4/19	2/9	3/11	5/9	5/28	3/13	2/3	1/4	5/18	5/18	8/13	8/11
DDM2 does Granger Cause DGNPDT	1/48	1/46	0	1/18	0	0	0	0	1/28	0	0	0	0	1/3	9/13	9/11
URATEA does Granger Cause DDM2	1/48	1/46	1/43	0	11/38	5/18	3/11	0	1/28	0	0	0	0	0	0	0
DDM2 does Granger Cause URATEA	1/16	1/23	2/43	1/18	5/19	1/4	3/11	1/18	3/14	2/13	0	0	0	0	0	0
TB3MYSS does Granger Cause DDM2	13/48	7/23	15/43	4/9	7/19	1/2	5/11	2/9	2/7	5/26	2/9	1/10	1/9	0	1/13	1/11
DDM2 does Granger Cause TB3MYSS	7/48	7/46	7/43	0	1/38	0	0	0	0	0	0	0	0	0	0	0
INFLARSS does Granger Cause DDM2	5/24	4/23	9/43	0	10/19	1/2	13/33	0	1/14	0	0	0	0	0	0	0
DDM2 does Granger Cause INFLARSS	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
DNETINCOME does Granger Cause DDM2	5/24	13/46	14/43	11/18	5/19	4/9	13/33	5/9	15/28	6/13	2/3	13/20	2/3	5/9	10/13	10/11
DDM2 does Granger Cause DNETINCOME	1/48	4/23	8/43	2/9	0	0	1/33	1/6	1/14	1/13	2/9	1/10	1/9	1/18	4/13	5/11
URATEA does Granger Cause DGNPDT	1/6	4/23	10/43	0	3/19	7/36	10/33	0	1/7	2/13	0	0	0	0	0	0
DGNPDT does Granger Cause URATEA	11/16	18/23	32/43	1	35/38	11/12	1	1	1	1	1	9/10	17/18	13/18	1	1
TB3MYSS does Granger Cause DGNPDT	1/3	6/23	10/43	5/9	3/19	1/12	5/33	0	0	0	0	0	0	0	0	0
DGNPDT does Granger Cause TB3MYSS	11/24	31/46	36/43	1	27/38	3/4	8/11	1/2	19/28	8/13	7/18	1/4	2/9	1/2	4/13	2/11
INFLARSS does Granger Cause DGNPDT	7/24	7/23	12/43	1/18	11/38	5/18	8/33	0	3/14	2/13	0	0	0	0	0	0
DGNPDT does Granger Cause INFLARSS	3/16	5/23	13/43	0	3/19	1/9	1/33	0	0	0	0	0	0	0	0	0
DNETINCOME does Granger Cause DGNPDT	0	1/46	1/43	1/18	0	0	0	0	3/28	1/13	7/18	1/10	1/9	1/9	2/13	2/11
DGNPDT does Granger Cause DNETINCOME	1/12	3/23	7/43	0	9/38	1/4	3/11	1/3	9/28	7/26	4/9	7/20	7/18	5/9	5/13	4/11
TB3MYSS does Granger Cause URATEA	29/48	27/46	26/43	1	33/38	8/9	31/33	1	6/7	11/13	1	17/20	8/9	11/18	1	1
URATEA does Granger Cause TB3MYSS	13/48	11/46	7/43	0	2/19	1/12	1/33	0	0	0	0	0	0	0	0	0
INFLARSS does Granger Cause URATEA	1/6	7/46	5/43	0	21/38	17/36	7/11	17/18	1	1	8/9	3/4	11/18	1/2	7/13	7/11
URATEA does Granger Cause INFLARSS	0	0	0	0	3/19	1/12	1/33	0	0	0	0	0	0	0	0	0
DNETINCOME does Granger Cause URATEA	17/48	15/46	16/43	1/2	11/38	1/3	10/33	1/2	9/28	9/26	1/2	9/20	7/18	1/2	9/13	9/11
URATEA does Granger Cause DNETINCOME	13/48	4/23	8/43	4/9	1/38	1/36	0	0	0	0	0	0	0	0	0	0
INFLARSS does Granger Cause TB3MYSS	5/24	5/23	8/43	1	2/19	1/12	2/33	0	0	1/26	0	0	0	0	0	0
TB3MYSS does Granger Cause INFLARSS	11/48	9/46	8/43	13/18	6/19	11/36	8/33	2/3	1/7	1/13	1/18	0	0	0	0	0
DNETINCOME does Granger Cause TB3MYSS	1/16	3/46	2/43	0	0	0	0	0	0	0	0	0	0	0	0	0
TB3MYSS does Granger Cause DNETINCOME	3/8	19/46	21/43	5/9	6/19	1/6	7/33	1/6	1/28	1/26	1/6	0	0	1/9	0	0
DNETINCOME does Granger Cause INFLARSS	1/48	1/46	1/43	0	1/38	1/36	1/33	0	0	0	0	0	0	0	0	0
INFLARSS does Granger Cause DNETINCOME	1/8	9/46	12/43	0	5/19	7/36	2/11	0	1/28	0	0	0	0	0	0	0

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