# Causation Delays and Causal Neutralization up to Three Steps Ahead: The Money-Output Relationship Revisited

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**Abstract**

In this paper, we develop a parametric test procedure for multiple horizon "Granger" causality and apply the procedure to the well established problem of determining causal patterns in aggregate monthly U.S. money and output. As opposed to most papers in the parametric causality literature, we are interested in whether money *ever* "causes" (can *ever* be used to forecast) output, *when* causation occurs, and *how* (through which causal chains). For brevity, we consider only causal patterns up to horizon $h = 3$. Our tests are based on new recursive parametric characterizations of causality chains which help to distinguish between mere noncausation (the total absence of indirect causal routes) and causal neutralization, in which several causal routes exists that cancel each other out such that noncausation occurs. In many cases the recursive characterizations imply greatly simplified linear compound hypotheses for multi-step ahead causation, and permit Wald tests with the usual asymptotic $\chi^2$-distribution.

A simulation study demonstrates that a sequential test method does not generate the type of size distortions typically reported in the literature, and null rejection frequencies depend entirely on how we define the "null hypothesis" of non-causality (at which horizon, if any).

Using monthly data employed in Stock and Watson (1989), and others, we demonstrate that while Friedman and Kuttner's (1993) result that detrended money growth fails to cause output one month ahead continues into the third quarter of 2003, a significant causal lag may exist through a variety of short-term interest rates: money appears to cause output after at least one month passes, although in some cases using recent data

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conflicting evidence suggests money may never cause output and be truly irrelevant in matters of real decisions.

**1. Introduction** We are interested in testing for linear causal patterns over multiple horizons within aggregate measures of output, money supply and interest rates. In particular, we test for the precise horizon at which the growth of the money supply causes output growth; for the possibility of *causal neutralization*, defined below; and we characterize through which indirect route, involving aggregate prices and interest rates, money causes output when evidence suggests causation occurs. In order to do so, we develop recursive techniques for characterizing typically nonlinear causality chains in terms of linear parametric restrictions.

Following Granger's (1969) and Sims' (1972) seminal studies, Lütkepohl (1993), Sims (1980) and Renault and Szafarz (1991) point out that indirect multi-step ahead causality from $Y$ to $X$ is possible in multivariate systems with auxiliary variables $Z$. Dufour and Renault (1998) set out a broad nonparametric and parametric theory of general horizon causality in Hilbert space.

The existence of causality chains in multivariate time series processes allows for multi-period *causation delays*: periods of noncausation followed by causation, a property conformable with the sluggishness of many macroeconomics events; and *causal neutralization*: multiple causal routes at some time horizon $h > 1$ may exist through $Z$, yet cancel each other out such that noncausation holds. Thus, in what sense $Y$ causes $X$ depends intimately on time horizon and the presence of auxiliary variables $Z$.

A simple, efficient, and asymptotically standard test procedure for multi-step ahead causation which can be employed to characterize causality chains and causal neutralization, however, has yet to be established. Lütkepohl and Müller (1994) and Lütkepohl and Burda (1997), for example, develop a Wald-type test for the highly non-linear parametric VAR conditions established by Dufour and Renault (1998). Due to matrix nonlinearities under the null hypothesis of noncausation, however, the limit distribution of the test statistic need not be standard : their solution is to add an arbitrary degree of noise to the estimated coefficients, severely effecting empirical power.

Using a more intuitive approach, Dufour *et al* (2003) utilize an $h$-step ahead VAR model in which some vector process $W_{t+h}$ is regressed on $(W_t, ..., W_1)$. After accounting for serial correlation in the resulting innovations series, a direct test of linear coefficient restrictions is all that is required to test for noncausation at one specific horizon $h \geq 1$. This procedure provides an elegantly simply method for testing multivariate noncausation at arbitrary time horizons, but entails several notable shortcomings. First, because the method by construction allows for a test of noncausality only at one horizon at a time, it follows that an efficient compound test of multiple horizon noncausality is infeasible. Indeed, second, a new VAR model must be estimated for each test making test result comparisons across horizons particularly difficult, even if a probability bounds scheme is employed. Third, the method usually cannot itself be used

to distinguish between simple noncausation (the total absence of indirect causal routes) and causal neutrality. For example, if we find noncausation from $Y$ to $X$ (with a multivariate auxiliary variable $Z$) for the individual horizons $h = 1...3$, it is impossible to tell whether noncausality was absolute at $h = 3$, or whether multiple causal routes through $Z$ cancelled each other out[1].

Moreover, the recursive causality chain representations presented here make it clear that noncausation over horizons $1...h$ followed by causality at $h + 1$ can occur *if and only if* an indirect causality chain exists. The procedure of Dufour *et al* (2003), however, does not provide a means to ensure such a logical outcome is analyzed[2]. Chao *et al* (2001), by comparison, consider an out-of-sample forecast improvement approach to testing non-causality. While this technique matches Granger's (1969, 1980), cf. Wiener (1956), original operational version of testing for causal patterns in time-series, this method, like Dufour *et al*'s (2003), only tests for non-causality at a particular horizon, and cannot be used in a simple efficient fashion to address causal chains.

In this paper, we develop new recursive parametric representations of causality chains which in many cases allow for clear horizon-to-horizon characterizations of causal delays and neutralization. The recursions developed here in many cases imply greatly simplified sequential linear restrictions for testing non-causality at arbitrary time horizons. When nonlinear restrictions cannot be avoided, however, in many cases Wald tests with standard distribution limits are still available. A relatively straightforward Bonferroni-type bounds can be applied to analyze the sequential test size.

For the sake of brevity, we consider causal patterns only up to horizon $h = 3$. Consult Hill (2005) for an expanded version of the present paper in which general results for arbitrary horizons $h \geq 1$ are developed.

A simulation study demonstrates that a sequential test method does not generate the type of size distortions in benchmark cases typically reported in the literature, and null rejection frequencies depend entirely on how we define the "null hypothesis" of non-causality. We show, evidently for the first time, that sequentially testing for non-causation $h$-steps ahead *only if we reject tests of non-causation at all horizons* essentially tames much of the distortion of classic tests of 1-step ahead non-causality commonly found in Wald tests. Nonetheless, in several cases we find that size distortions still persist, however the distortions

[1] It seems, however, that some information regarding causal neutralization can de adduced from their approach by incorporating methods developed here. Using their approach verbatim, however, does not lead to an understanding of causal neutralization in the case described above.

[2] For example, in their study of monthly GDP ($X$), the federal funds rate ($Y$), and the GDP deflator and non-borrowed reserves ($Z$), horizon specific tests suggest $Y$ fails to cause $X$ for horizons 1 and 2, and causes $X$ at horizon $h = 3$. This is possible only if an indirect causal route $Y \to Z \to X$ exists where causation occurs one-month ahead: see Theorems 2 and 4, below. However, their test procedure reveals that $Y$ fails to cause $Z$ one-month ahead and $Z$ fails to cause $X$ one month ahead, a characteristic that implies *noncausation at all horizons*, which contradicts their conclusion. Their finding, moreover, is robust to the possibility that the multivariate process $X, Y, Z$ is nonstationary: see Tables 4 and 7 of Dufour *et al* (2003). A compound multiple horizon test procedure sensitive to causal chain structure will diminish the likelihood that such contradictory conclusions are made.

favor detecting causation sooner than it actually occurs.

When applied to the now classic monetarist question of whether and how monthly money statistically influences aggregate output, we are able to detect significant causal delays from money growth to growth in output through a variety of interest rates and inflation. In a seminal paper, Stock and Watson (1989) address deterministic and stochastic trend components in monthly money, output, inflation and the Treasury bill rate, and find significant evidence that detrended money growth causes output growth during the period of January 1959 - December 1985. Friedman and Kuttner (1993) then extend the sample period used in Stock and Watson (1989) through 1990 for the same variables and find detrended money growth fails to cause output growth one month ahead. Moreover, using the same sample period as Stock and Watson (1989), Friedman and Kuttner (1993) replace the essentially risk-free Treasury bill rate with the commercial paper rate and again find money likely does not cause output. In particular, the commercial paper-Treasury bill rate spread appears to contain the most predictive power in their preferred system of variables. See, also, Friedman and Kuttner (1992); and see Chao *et al* (2001) and Rothman *et al* (2001) for alternative treatments of 1-step ahead causality in money-output/income[3].

It is important to point out that in none of the above studies is there a rigorous statistical analysis, or even mention, of the possibility of an indirect causal link from money to interest and interest to output, arguably the most obvious transmission mechanism by which fluctuations in the money supply will eventually impact real decisions. Dufour *et al* (2003), however, do discuss indirect causal chains from non-borrowed reserves to GDP through the federal funds rate, however their conclusion is not supported by their own analysis. They point out that non-borrowed reserves causes the federal funds rate one-month ahead, and the rate causes GDP 3-months ahead, and deduce a causal chain exists. However, as discussed above, their study does not reveal *how* the federal funds rate causes GDP 3-months ahead because evidence suggests no causal chains exist from the rate to GDP: the federal funds rates does not cause anything one-step ahead, and therefore it is difficult to reconcile their conclusion of "indirect causality" from non-borrowed reserves to GDP through the interest rate.

In our study, we employ Stock and Watson's (1989) and Friedman and Kuttner's (1993) data samples, and an extended sample through August 2003. We typically find that money matters after at least a one-month lag: contrary to Friedman and Kuttner's (1993) finding that the "*role of money...is trivial*" in the presence of the Treasury bill or commercial paper rates before 1991, money

[3]Causal patterns will clearly be sensitive to functional specification, specification of the causal moment (mean, variance, etc), and in-sample versus out-of-sample methods are employed. Following Stock and Watson (1989), Friedman and Kuttner (1993), and many others, we employ linear VAR models for our analysis of multi-step ahead causation. For studies of nonlinear causation in mean between money and output, see, e.g., Rothman *et al* (2001) who use multivariate smooth transition autoregressive models (STAR). For out-of-sample methods, see Swanson (1998) and Chao *et al* (2001).

growth is apparently *imperative* for forecasting output growth as early as two months ahead, and therefore money growth may contain predictive information for forecasting output growth one-quarter ahead. In models of money supply growth $\Delta m$, output growth $\Delta y$, inflation $\Delta p$, fluctuations in an interest rate $\Delta r$ and a rate spread $rr$, however, we find only one case in which evidence suggests causal neutralization occurs, and the evidence is quite weak once size bounds are accounted for. In every model and period considered in this paper, when a causal delay from money to output is detected it is significantly manifest in a short chain through interest rates, $\Delta m \to \Delta r \to \Delta y$, or in a longer sequence of multiple chains through interest and a rate spread, $\Delta m \to (\Delta r, rr) \to \Delta y$.

It is notable that in only a few models treated in our study do we find significant evidence in favor of causation exactly 3-months (one-quarter) ahead, although evidence strongly suggests causation 1 or 2 months ahead[4]. Thus, although our study in general strengthens the claims that money does not cause output exactly one-quarter ahead[5], we do find evidence causation occurs *about* one quarter ahead, which demonstrates the empirical weakness of focusing entirely on *one-step ahead* causation.

There are, however, notable limitations of our parametric approach. First, we do not deconstruct *all* nonlinear noncausality conditions which *do* have recursive linear presentations: there are many cases we must omit for the sake of brevity, and which can be deduced by imitating the ideas developed here. Indeed, we focus on linear conditions for multi-step ahead noncausation through 3-steps ahead which are particularly useful in our study of money and output, and reasonably expansive enough to be useful in many other empirical applications. Second, there are many cases in which recursive necessary and sufficient *linear* parametric conditions for noncausation do not exist: depending on the horizon, the dimension of $Z$ and the relationships from $Y$ to $Z$ and $Z$ to $X$, there are many contexts in which we are forced to face Wald tests of nonlinear hypotheses that may result in non-standard limit distributions. In these cases, assuming we have exhausted simple recursive linear conditions for noncausation and can no longer (simply) ascertain whether absolute noncausation of causal neutralization may be occurring, a test method like Dufour *et al*'s (2003) or Lütkepohl and Burda's (1997) appears to be all that is left.

This is not a limitation of our study per se, but the limitations of VAR para-

[4] Of the combined 12 models and sample periods studied, we find three cases in which evidence points to causation exactly one-quarter ahead, and two of those occur in Friedman and Kuttner's (1993) sample period and chosen models. Furthermore, all three cases occur in Stock and Watson's (1989) and Friedman and Kuttner's (1993) samples with the latter's chosen augmented model with the Treasury bill and commercial paper rate spread, evidence which arguably contradicts Friedman and Kuttner's (1992) own finding that quarterly money fails to cause real income one-quarter ahead.

[5] For example, Feige and Pearce (1979) perform standard Granger tests, as well as employ Sims (1972) distibuted lag and AR(2) pre-filter method, and find money fails to cause GNP one quarter ahead. It should be noted that Friedman and Kuttner (1992) employ data measured in quarterly increments, and causal patterns at the monthly level for monthy data need not match patterns at the quarterly level for quarterly data. The discrepency lies in the complexities involved with time aggregration of stock and flow variables, an issue which has not been thoroughly analyzed in the metric projection theory literature.

metric recursions in multivariate settings. In general, in VAR systems with low dimension auxiliary processes (e.g. 1-4 components of $Z$), the results contained here will often be comprehensive enough to analyze completely multi-step ahead multivariate causal routes. Indeed, when there is only one auxiliary variable (i.e. $Z$ is scalar-valued), a very simple linear compound necessary and sufficient condition exists for noncausation. In any event, in our empirical study of a popular data set including money and income, in which $Z$ contains either 2 or 3 variables (inflation, interest and the spread between risky and risk-free rates), we never come across a model or sample period in which non-standard Wald tests are implied.

The rest of the paper contains the following topics. In Section 2 we briefly define prediction-based causality, and detail parametric representations of causal chains in Section 3. Section 4 contains details on the test approach, Section 5 discusses test size bounds and size distortions, and Section 6 contains the empirical study. Concluding remarks are left for Section 7. Appendix 1 contains tables; Appendix 2 contains a small simulation study; and Appendix 3 contains all proofs..

Throughout, we employ the following notation conventions. For Hilbert spaces $A$ and $B$, we write $A + B$ to denote the Hilbert space spanned by all components of $A$ and $B$. We write $U_t \perp V_t$ for $m$-vector processes $U_t$ and $V_t$ to denote orthogonality between all scalar components for all $t$, $u_{i,t} \perp v_{j,t}$, $i, j = 1...m$, which in $L_2(\Omega, \mathfrak{F}_t, Q)$ implies $E(u_{i,t} v_{j,t}) = 0$ for every $i, j = 1...k$ and every $t$. For an $m$-vector-process $\{W_t : t \in \mathbb{Z}\}$, let $\overline{sp}(W_s : s \leq t) = \overline{sp}(W_{i,s} : i = 1...m,\ s \leq t)$ denote the closed linear span.

**2. Causality Preliminaries** We define non-causality in the manner of Granger (1969), which was augmented to a multiple horizon parametric framework by Dufour and Renault (1998). Consider some $m$-vector processes $\{W_t\}$ with trivariate representation $W_t = (X_t', Y_t', Z_t')'$, where $X_t$, $Y_t$, and $Z_t$ have dimensions $m_x \geq 1$, $m_y \geq 1$ and $m_z \geq 0$ respectively, and $m = m_x + m_y + m_z \geq 2$. We assume $W_t$ is defined in the Hilbert space $L_2(\Omega, \mathfrak{F}_t, Q)$, where $\mathfrak{F}_t$ denotes an increasing $\sigma$-field of all past and present information at time $t$, $\mathfrak{F}_t = \sigma(W_s : s \leq t)$, and $Q$ denotes a proper probability measure. Denote by $I$ an information universe, and let $I_{XZ} = \overline{sp}(X_s : s \leq t) + \overline{sp}(Z_s : s \leq t)$ for an arbitrary time period $t$.

In principle, none of the following results rely on stationarity assumptions. For example, we may allow time to be bounded in the finite past. For brevity, we consider only an unbounded past.

We say the subvector $Y_t$ "*does not cause*" $X_t$ *at horizon* $h > 0$ in some Hilbert space (denoted $Y \overset{h}{\nrightarrow} X | I_{XZ}$) if inclusion of $\overline{sp}(Y_s : s \leq t)$ does not improve the metric projection of $X_{t+h}$ for all $t$ (i.e. the normed prediction error remains unchanged); $Y_t$ "*does not cause*" $X_t$ *up to horizon* $h > 0$ (denoted $Y \overset{(h)}{\nrightarrow} X | I_{XZ}$) if inclusion of $\overline{sp}(Y_s : s \leq t)$ does not improve the metric projection of $X_{t+k}$, for each $k = 1...h$ and for all $t$; and $Y_t$ "*does not cause*" $X_t$ *at any horizon* $h > 0$ (denoted $Y \overset{(\infty)}{\nrightarrow} X | I_{XZ}$) if inclusion of $\overline{sp}(Y_s : s \leq t)$ does not improve the

metric projection of $X_{t+h}$, for every $h > 0$ and for all $t$. In $L_2(\Omega, \mathfrak{F}_t, Q)$, forecast improvement is measured by a diminishment in the mean-squared-forecast-error.

It is important to point out that the definition of non-causality implies causation $Y \overset{h}{\rightarrow} X$ occurs *if and only if* at least one scalar component of the closed linear span of $Y_{i,s}$, $i = 1...m_y$, $s \leq t$, improves a forecast of at least one scalar component $X_{j,t+h}$, $j = 1...m_x$.

The following result will be useful for subsequent discourse, and follows straightforwardly from Proposition 2.3 of Dufour and Renault (1998).

**Theorem 1** *Consider the VAR process* (2), *define* $Z = (U', V')'$. *i. If* $Y \overset{1}{\nrightarrow} (X, Z)|I_{XZ}$, *or* $(Y, Z) \overset{1}{\nrightarrow} X|I_{XZ}$, *then* $Y \overset{(\infty)}{\nrightarrow} X|I_{XZ}$; *ii. If* $(Y, U) \overset{1}{\nrightarrow} (X, V)|I_{XV}$, *then* $Y \overset{(\infty)}{\nrightarrow} X|I_{XZ}$; *iii. In order for non-causation* $Y \overset{1}{\nrightarrow} X|I_{XZ}$ *to be followed by causation* $Y \overset{h}{\rightarrow} X|I_{XZ}$, *for some* $h > 1$, *it is necessary for* $Y \overset{1}{\rightarrow} Z \overset{1}{\rightarrow} X$.

*Remark* 1: If the auxiliary process $Z$ affords the partition $Z = (U', V')'$ such that $(Y, U) \overset{1}{\nrightarrow} (X, V)|I_{XV}$, then no form of causal chain can exist, and $Y \overset{(\infty)}{\nrightarrow} X|I_{XZ}$: even if $Y \overset{1}{\rightarrow} U$ and/or $V \overset{1}{\rightarrow} X$, causal-chains are broken by $Y \overset{1}{\rightarrow} U \overset{1}{\nrightarrow} X$ or $Y \overset{1}{\nrightarrow} V \overset{1}{\rightarrow} X$ or $Y \overset{1}{\rightarrow} U \overset{1}{\nrightarrow} V \overset{1}{\rightarrow} X$, etc. Similarly, if non-causation $Y \overset{1}{\nrightarrow} X|I_{XZ}$ holds, and $Y \overset{1}{\nrightarrow} Z|I_{XZ}$ or $Z \overset{1}{\nrightarrow} X|I_{XZ}$ holds, then a broken causal chain exists, and non-causation for all horizons exists.

*Remark* 2: Because $Y \overset{1}{\nrightarrow} (X, Z)$ or $(Y, Z) \overset{1}{\nrightarrow} X$ are sufficient for $Y \overset{(\infty)}{\nrightarrow} X$, non-causation $Y \overset{1}{\nrightarrow} X|I_{XZ}$ followed by causation $Y \overset{h}{\rightarrow} X|I_{XZ}$, $h \geq 2$, can only occur if a causal-chain exists, $Y \overset{1}{\rightarrow} Z \overset{1}{\rightarrow} X$. However, except in the univariate $Z$ case (see Theorem 4.*i*, below), a "causal chain", $Y \overset{1}{\rightarrow} Z \overset{1}{\rightarrow} X$, is generally not sufficient for causation $Y \overset{h}{\rightarrow} X|I_{XZ}$, $h \geq 2$, due to the multiplicity of possible causal routes which may cancel out.

Assume $W_t$ has an autoregressive representation

$$W_t = \sum\nolimits_{i=1}^{\infty} \pi_i W_{t-i} + \epsilon_t, \quad \epsilon_t \perp \overline{sp}(W_s : s \leq t), \tag{1}$$

where $\epsilon_t$ denotes an $L_2(\Omega, \mathfrak{F}_t, Q)$ $m$-vector with zero mean, non-singular moment matrix $E[\epsilon_t \epsilon_t']$, and is $L_2(\Omega, \mathfrak{F}_t, Q)$ orthogonal to the span $\overline{sp}(W_s : s \leq t)$. The coefficients $\pi_i$ are real-valued $m \times m$ matrices for each $i$, and the infinite series $\sum_{i=1}^{\infty} \pi_i W_{t-i}$ is assumed to converge in mean-square. Most $L_2(\Omega, \mathfrak{F}_t, Q)$ processes of interest will have a representation (1) either in levels, or after some standard transformation, e.g. first differencing[6].

By Hilbert projection operator linearity and orthogonality, (1), the $h$-step ahead projection of $W_{t+h}$ onto the sub-space $\overline{sp}(W_s : s \leq t)$ satisfies the recursion

$$\hat{W}_{t+h} = \sum\nolimits_{i=1}^{\infty} \pi_i \hat{W}_{t+h-i} = \sum\nolimits_{i=1}^{\infty} \pi_i^{(h)} W_{t+1-i}, \tag{2}$$

[6] In what follows, we explicitly ignore the issue of cointegration and VECM's, however only slight modifications to (1) and the following discourse is required to include this case.

where $\hat{W}_{t+h-i} \equiv W_{t+h-i} \forall i \geq h$, and the coefficient matrix sequence $\{\pi_i^{(h)}\}_{i=1}^{\infty}$ is defined by the recursive relationship

$$\pi_1^{(0)} = I_m, \quad \pi_j^{(1)} = \pi_j, \quad \pi_j^{(h+1)} = \pi_{j+1}^{(h)} + \pi_1^{(h)}\pi_j. \tag{3}$$

See, e.g., Dufour and Renault (1994).

Consider the $(X', Y', Z')'$-conformable partition of the coefficient sequence

$$\pi_j^{(h)} = \begin{bmatrix} \pi_{XX,j}^{(h)} & \pi_{XY,j}^{(h)} & \pi_{XZ,j}^{(h)} \\ \pi_{YX,j}^{(h)} & \pi_{YY,j}^{(h)} & \pi_{YZ,j}^{(h)} \\ \pi_{ZX,j}^{(h)} & \pi_{ZY,j}^{(h)} & \pi_{ZZ,j}^{(h)} \end{bmatrix}. \tag{4}$$

For example, for every $j \geq 1$, $\pi_{XY,j}^{(h)}$ denotes an $m_x \times m_y$ matrix of constant real numbers.

The following theorem, due to Dufour and Renault (1998: Theorem 3.1), provides a nonlinear basis for parametric tests of noncausality $h$-steps ahead.

**Theorem 2 (*VAR*$(\infty)$ Non-Causality at $h \geq 1$)** *Consider any $m$-vector process $W_t = (X_t', Y_t', Z_t')'$ such that assumptions* (2) *and* (3) *hold. Then, $Y \overset{h}{\nrightarrow} X|I_{XZ}$ if and only if $\pi_{XY,j}^{(h)} = 0$, $\forall j = 1, 2, ...$*

**3. Causality Chains and Neutralization** Because $Y \overset{1}{\nrightarrow} X|I_{XZ}$ and $Y \overset{1}{\nrightarrow} Z|I_{XZ}$ will imply non-causation at all horizons, $Y \overset{(\infty)}{\nrightarrow} X|I_{XZ}$ (cf. Theorem 1), we assume causation $Y \overset{1}{\rightarrow} Z|I_{XZ}$ throughout the remainder of the paper, unless otherwise noted.

Notice that non-causality at horizon $h = 1$ is not in general synonymous with non-causality at every horizon $h \geq 1$ due to the presence of auxiliary variates. Indeed, the coefficient recursion (3) renders the $XY^{th}$-block of $\pi_j$ as

$$\pi_{XY,j}^{(h+1)} = \pi_{XY,j+1}^{(h)} + \pi_{XX,1}^{(h)}\pi_{XY,j} + \pi_{XY,1}^{(h)}\pi_{YY,j} + \pi_{XZ,1}^{(h)}\pi_{ZY,j}. \tag{5}$$

It follows that if non-causality up to horizon $h$ is true, $Y \overset{(h)}{\nrightarrow} X|I_{XZ}$, then, cf. Theorem 2, $\pi_{XY,j}^{(k)} = 0$ for each $k = 1...h$, and subsequently, cf. (5) and Theorem 2, $Y \overset{h+1}{\nrightarrow} X|I_{XZ}$ *if and only if*

$$\pi_{XY,j}^{(h+1)} = \pi_{XZ,1}^{(h)}\pi_{ZY,j} = 0, \forall j \geq 1. \tag{6}$$

Thus, non-causality up to horizon $h \geq 1$ and causality at $h + 1$ can only occur if a causality chain exists such that $\pi_{XZ,1}^{(h)}\pi_{ZY,j} \neq 0$, for some $j \geq 1$. Provided $Y \overset{1}{\rightarrow} Z|I_{XZ}$, then some scalar component of $\pi_{ZY,j}$ is non-zero for some $j \geq 1$. However, from (6) clearly $\pi_{XZ,1}^{(h)} = 0$ is not necessary for $Y \overset{h+1}{\nrightarrow} X|I_{XZ}$ due to the nonlinear row-column combinations that may yet imply $\pi_{XZ,1}^{(h)}\pi_{ZY,j} = 0$ with $\pi_{XZ,1}^{(h)} \neq 0$.

Because $\pi^{(h)}_{XZ,1}\pi_{ZY,j} = 0$ is possible by numerous nonlinear row-column combinations if $Z$ has dimension greater than 1, we seek simplifying conditions that involve linear coefficient restrictions. Without loss of generality, assume $X$ and $Y$ are univariate[7]. We can simplify matters if we partition $Z_t = (U_t', V_t')'$ , where $U_t$ and $V_t$ are $m_u$ and $m_v$-vectors respectively, in such a way that provided $Y \xrightarrow{1} Z|I_{XZ}$, then $Y \xrightarrow{1} U|I_{XZ}$ and $Y \overset{1}{\nrightarrow} V|I_{XZ}$. It is possible to have $m_v = 0$ (i.e. $Y \xrightarrow{1} U = Z$), in which case by convention it is understood that all $V$-related coefficients are identically zero (e.g. $\pi_{XV,j} = 0$).

The condition $Y \overset{1}{\nrightarrow} V|I_{XZ}$ implies $\pi_{VY,j} = 0$, $\forall j \geq 1$, and

$$\pi^{(h)}_{XZ,1}\pi_{ZY,j} = \left[\pi^{(h)}_{XU,1}|\pi^{(h)}_{XV,1}\right] \begin{bmatrix} \pi_{UY,j} \\ \hline \pi_{VY,j} \end{bmatrix} = \pi^{(h)}_{XU,1}\pi_{UY,j}. \tag{7}$$

Thus, if $Y \overset{(h)}{\nrightarrow} X|I_{XZ}$ and $Y \xrightarrow{1} U|I_{XZ}$ are true, (6) and (7) imply $Y \overset{h+1}{\nrightarrow} X|I_{XZ}$ *if and only if* $\pi^{(h)}_{XU,1}\pi_{UY,j} = 0$, $\forall j \geq 1$.

We have two cases to consider. Provided $m_u = 1$, then a relatively simple recursion exists for determining non-causation multiple-steps ahead. The multivariate case, $m_u > 1$, proves to be somewhat more challenging. We consider the cases in turn.

### 3.1 Univariate $U$, $\pi^{(h)}_{XU,1} = 0$

Let $m_u = 1$ ($m_v \geq 0$), and assume $Y \overset{(h)}{\nrightarrow} X|I_{XZ}$ and $Y \xrightarrow{1} U|I_{XZ}$ are true. Notice that $Y \xrightarrow{1} U|I_{XZ}$ implies at least one $\pi_{UY,j} \neq 0$, and because we assume $U$ is univariate, we deduce $\pi^{(h)}_{XU,1}\pi_{UY,j} = 0$ for every $\forall j \geq 1$ *if and only if* $\pi^{(h)}_{XU,1} = 0$.

**Lemma 3** *Consider the VAR process* (2), *define* $Z = (U', V')'$, *assume* $Y \xrightarrow{1} U|I_{XZ}$ *and* $Y \overset{1}{\nrightarrow} V|I_{XZ}$, *and let* $m_u = 1$. *Assume* $Y \overset{(h)}{\nrightarrow} X|I_{XZ}$ *for some* $h \geq 2$.

*i.* $Y \overset{(h+1)}{\nrightarrow} X|I_{XZ}$ *if and only if* $\pi^{(h)}_{XU,1} = 0$;

*ii.* $\pi^{(h)}_{XU,1} = \pi_{XU,h} + \sum_{i=1}^{h-1} \pi^{(h-i)}_{XX,1}\pi_{XU,i} + \sum_{i=1}^{h-1} \pi^{(h-i)}_{XV,1}\pi_{VU,i}$;

*iii.* *If* $\pi_{XU,i} = \pi_{VU,i} = 0$, $i = 1...h-1$, *then* $\pi^{(h)}_{XU,1} = \pi_{XU,h}$.

**Theorem 4** *Consider the VAR process* (2), *define* $Z = (U', V')'$. *Assume* $Y \overset{1}{\nrightarrow} X|I_{XZ}$, $Y \overset{1}{\nrightarrow} V|I_{XZ}$ *and* $Y \xrightarrow{1} U|I_{XZ}$, *with* $m_u = 1$.

*i.* *If* $m_z = 1$ (*hence* $U = Z$) *and* $Y \overset{(h)}{\nrightarrow} X|I_{XZ}$ *for any* $h \geq 1$, *then* $Y \overset{(h+1)}{\nrightarrow} X|I_{XZ}$ *if and only if* $\pi_{XZ,h} = 0$;

*For all remaining results, let* $m_z > 1$ *and* $m_u = 1$ :

*ii.* $Y \overset{(2)}{\nrightarrow} X|I_{XZ}$ *if and only if* $\pi_{XU,1} = 0$;

[7] Dufour and Renault (1994, 1998) prove that noncausation from vector process $Y$ to vector process $X$ is equivelant to noncausation from each scalar component $Y_i$ to each scalar component $X_j$. Thus, it suffices to consider the causal structure from $Y$ to $X$ by considering the scalar components individually.

*iii.* *If* $Y \overset{(2)}{\nrightarrow} X|I_{XZ}$ *and* $U \overset{1}{\nrightarrow} V|I_{XYV}$, *then* $Y \overset{(3)}{\nrightarrow} X|I_{XZ}$ *if and only if* $\pi_{XU,2} = 0$;

*iv.* *If* $Y \overset{(2)}{\nrightarrow} X|I_{XZ}$ *and* $V \overset{1}{\nrightarrow} (X,U)|I_{XYU}$, *then* $Y \overset{(3)}{\nrightarrow} X|I_{XZ}$ *if and only if* $\pi_{XU,2} = 0$.

*Remark* 1: From Lemma 3, sequentially if $Y \overset{(h)}{\nrightarrow} X|I_{XZ}$, then the total impact of $U$ on $X$ at $h+1$-steps ahead is accumulated in $\pi^{(h)}_{XU,1} = \pi_{XU,h} + \sum_{i=1}^{h-1} \pi^{(h-i)}_{XX,1}\pi_{XU,i} + \sum_{i=1}^{h-1} \pi^{(h-i)}_{XV,1}\pi_{VU,i}$. For example, $Y$ may cause $X$ at $h+1 = 2$ through $Y \to U \to X$, or at horizons $h+1 = 3$ by multiple routes, $Y \to U \to X \to X$ and $Y \to U \to V \to X$ and $Y \to U \to X \to X$. If the multiple causal routes cancel each other out such that noncausation holds at horizon $h+1$ (i.e. $\pi^{(h)}_{XU,1} = 0$), we say "*causal neutralization*" has occurred. Moreover, notice that causal neutralization can only occur for horizons $h \geq 3$: at horizon $h = 2$, too little time has passed for multi-path complexities to develop, and causation occurs only through $Y \overset{1}{\to} U \overset{1}{\to} X$: thus $\pi_{XU,1} = 0$ is necessary and sufficient for noncausation up to 2-steps ahead.

*Remark* 2: By result $(i)$ of Theorem 4, if the auxiliary process $Z$ is univariate, then there is only one indirect route by which $Y$ can cause $X$, thus it is necessary and sufficient to analyze only $\pi_{XZ,h} = 0$ sequentially for each $h = 1, 2, ...$

*Remark* 3: From result $(iii)$, if the indirect link via $U$ to $V$ does not exist, then for $Y \overset{(3)}{\nrightarrow} X|I_{XZ}$ it suffices merely to check $Y \to U \to X$ by sequentially inspecting $\pi_{XU,i} = 0$, $i = 1, 2, ...$ Likewise, by result $(iv)$, if the indirect chains implied from $V$ to $U$ and $V$ to $X$ do not exist, then again it suffices to check sequentially $\pi_{XU,i} = 0$. Either case represents complete non-causality, $Y \overset{(3)}{\nrightarrow} X|I_{XZ}$.

*Remark* 4: Results $(iii)$ and $(iv)$ extend to the general horizon case. For example, if $Y \overset{1}{\nrightarrow} V|I_{XZ}$, $Y \overset{(2)}{\nrightarrow} X|I_{XZ}$ and $U \overset{1}{\nrightarrow} V|I_{XYV}$, then $Y \overset{(h)}{\nrightarrow} X|I_{XZ}$ *if and only if* $\pi_{XU,i} = 0$, $i = 1...h-1$, for any $h \geq 2$. See Hill (2005).

Notice that the conditions for Theorem 4.*i*,*ii* are necessary and sufficient, but the conditions for Theorem 4.*ii*,*iv* are only sufficient. If the sufficient conditions for $Y \overset{(3)}{\nrightarrow} X|I_{XZ}$ in Theorem 4.*iii*,*iv* fail to exist, we are left with testing the necessary and sufficient condition $\pi^{(2)}_{XU,1} = 0$ directly.

In this case Theorem 4.*ii* and Lemma 3.*ii* are sequentially helpful: if $Y \overset{1}{\nrightarrow} X|I_{XZ}$ and $\pi_{XU,1} = 0$, then we deduce both that $Y \overset{(2)}{\nrightarrow} X|I_{XZ}$, and $\pi^{(2)}_{XU,1}$ reduces to

$$\begin{aligned} \pi^{(2)}_{XU,1} &= \pi_{XU,2} + \pi_{XX,1}\pi_{XU,1} + \pi_{XV,1}\pi_{VU,1} = 0 \\ &= \pi_{XU,2} + \pi_{XV,1}\pi_{VU,1}. \end{aligned} \tag{8}$$

**Corollary 5** *Consider the VAR process* (2), *and define* $Z = (U', V')'$. *Assume* $Y \overset{(2)}{\nrightarrow} X|I_{XZ}$, $Y \overset{1}{\nrightarrow} V|I_{XZ}$ *and* $Y \overset{1}{\to} U|I_{XZ}$, *with* $m_u = 1$. *Assume* $\pi_{XU,2} \neq 0$ *and/or* $\pi_{VU,1} \neq 0$. *Then*

*i.* $Y \overset{(3)}{\nrightarrow} X|I_{XZ}$ *if and only if* $\pi_{XU,2} + \pi_{XV,1}\pi_{VU,1} = 0$;

*ii.* *If* $\pi_{XU,2} \neq 0$ *and* ($\pi_{VU,1} = 0$ *or* $\pi_{XV,1} = 0$), *then* $Y \overset{(3)}{\rightarrow} X|I_{XZ}$;

*iii.* *If* $\pi_{XU,2} = \pi_{XV,1} = 0$ *then* $Y \overset{(3)}{\nrightarrow} X|I_{XZ}$, *and* $Y \overset{(4)}{\nrightarrow} X|I_{XZ}$ *if and only if*

$$\pi_{XU,3} + \pi_{XV,2}\pi_{VU,1} = 0; \tag{9}$$

*iv.* *If* $\pi_{XU,2} = \pi_{VU,1} = 0$ *then* $Y \overset{(3)}{\nrightarrow} X|I_{XZ}$, *and* $Y \overset{(4)}{\nrightarrow} X|I_{XZ}$ *if and only if*

$$\pi_{XU,3} + \pi_{XV,1}\pi_{VU,2} = 0; \tag{10}$$

*Remark* 1: Result (*ii*) rules out *causal neutralization* at horizon $h = 3$ such that causation occurs. Results (*iii*)-(*iv*) rule out immediate causal links from $U$ to $V$ to $X$, and $U$ to $X$, such that non-causation occurs but not neutralization. We consider the possibility of neutralization in the subsequent subsection.

### 3.2 Causal Neutralization at h = 3 and Nonlinear Hypotheses

The above cases (Theorem 4.*ii*-*iv* and Corollary 5.*ii*-*iv*) cover every (non-redundant) possibility for $Y \overset{(2)}{\nrightarrow} X|I_{XZ}$ sequentially to imply $Y \overset{(3)}{\nrightarrow} X|I_{XZ}$, except for cases where $V$ is multivariate and *causal neutralization* occurs at horizon $h = 3$.

The omitted cases are (*v*) $\pi_{VU,1} \neq 0$, $\pi_{XV,1} \neq 0$, with possibly $\pi_{XV,1}\pi_{VU,1} = 0$ when $m_v > 1$; and (*vi*) $\pi_{XU,2} \neq 0$, $\pi_{VU,1} \neq 0$ and $\pi_{XV,1} \neq 0$, where $\pi_{XU,2} + \pi_{XV,1}\pi_{VU,1} = 0$ is possible.

In case (*v*) if $\pi_{XU,2} = \pi_{XV,1}\pi_{VU,1} = 0$, then noncausality $Y \overset{(3)}{\nrightarrow} X|I_{XZ}$ follows, and we proceed (in principle) to inspect conditions for subsequent noncausality at horizon $h > 3$. The fact that $\pi_{XU,2} = 0$, $\pi_{VU,1} \neq 0$, $\pi_{XV,1} \neq 0$, and $\pi_{XV,1}\pi_{VU,1} = 0$ implies possible causal neutralization: for example, $U$ may cause $X$ through multiple routes by different components of $V$ all of which cancel each other out. However, the variable $U$ may cause components of $V$ which do not cause $X$: in this case, neutralization has not taken place.

In the second omitted case, (*vi*), there are connections from $Y$ to $U$ to $X$, and from $Y$ to $U$ to $V$ to $X$, where the immediate dual causal routes from $U$ to $V$ to $X$, and $U$ to $X$, cancel out such that causal neutralization occurs unambiguously.

In either case, the most efficient strategy is to check directly the nonlinear combination $\pi_{XU,2} + \pi_{XV,1}\pi_{VU,1}$, cf. (8). Thus, if *all* of the linear sufficient conditions of Theorem 4 and Corollary 5 fail to hold, the only remaining condition is, indeed, nonlinear, and necessary and sufficient, cf. Corollary 5.*i*: if $Y \overset{(2)}{\nrightarrow} X|I_{XZ}$, $Y \overset{1}{\nrightarrow} V|I_{XZ}$, $Y \overset{1}{\rightarrow} U|I_{XZ}$, with $m_u = 1$, then $Y \overset{(3)}{\rightarrow} X|I_{XZ}$ *if and only if* $\pi_{XU,2} + \pi_{XV,1}\pi_{VU,1} = 0$.

Importantly, the nonlinear compound hypothesis $H_0 : \pi_{XU,2} + \pi_{XV,1}\pi_{VU,1} = 0$ will always lead to a test statistic with standard chi-squared limiting distribution as long as we employ a VAR model of order $p \geq 2$, even when the true order is $p = 1$. Consider, for example, a VAR($p$) model, put $p \geq 2$, define $\pi \equiv$

$(\pi_1, ..., \pi_p)$, an $m \times mp$ matrix, and define $\Pi \equiv vec(\pi)$, an $m^2 p \times 1$ vector that vertically stacks the columns of $\pi$. Denote the nonlinear restriction (8) as

$$r(\Pi) = \pi_{XU,2} + \pi_{XV,1}\pi_{VU,1} = 0, \tag{8'}$$

an $m_x \times m_u$ matrix. Then, for univariate $X$ and $U$, for example,

$$(\partial/\partial\Pi) r(\Pi) = (0, .., \pi_{VU,1}, ..., \pi_{XV,1}, ..., 1, ..., 0)', \tag{8''}$$

a gradient which will lead to a nonsingular Wald statistic covariance matrix, irrespective of the coefficient magnitudes $\pi_{VU,1}$ and $\pi_{XV,1}$. The result similarly holds for multivariate $X$ and $U$. Thus, even if the true VAR order satisfies $p = 1$ such that $\pi_{XU,2} = 0$, we can always perform a test of the given nonlinear restriction, (8). In our empirical study, because we never obtain an optimal VAR order less than $p = 6$, we are therefore easily able to test for noncausation through at least $h = 3$ months ahead.

Unfortunately, if we detect causal neutralization at horizon $h = 3$, it becomes prohibitively difficult to establish causal properties at subsequent horizons $h > 3$. Consult Hill (2005) for details of possible strategies in this case.

### 3.3 Compound Linear Conditions for Multi-Step Ahead Non-Causation

Together, Theorem 4 and Corollary 5 imply compound sufficient, and necessary and sufficient, conditions for non-causation up to horizon $h = 2$ or 3. As usual, assume $Y \overset{1}{\nrightarrow} X|I_{XZ}$, and partition $Z = (U', V')'$.

**Theorem 6** *Consider the VAR process* (2), *define* $Z = (U', V')'$ *such that* $Y \overset{1}{\rightarrow} U|I_{XZ}$, $m_u = 1$. *Then,*

*i.* $Y \overset{(2)}{\nrightarrow} X|I_{XZ}$ *if and only if* $Y \overset{1}{\nrightarrow} (X, V)|I_{XZ}$, $\pi_{XU,1} = 0$;

*ii.* $Y \overset{(3)}{\nrightarrow} X|I_{XZ}$ *if and only if* $Y \overset{1}{\nrightarrow} (X, V)|I_{XZ}$, $\pi_{XU,1} = 0$, $\pi_{XU,2} + \pi_{XV,1}\pi_{VU,1} = 0$.

*Moreover, each of the following are sufficient conditions for complete* $Y \overset{(3)}{\nrightarrow} X|I_{XZ}$ :

*iii.* $Y \overset{1}{\nrightarrow} (X, V)|I_{XZ}$, $U \overset{1}{\nrightarrow} V|I_{XZ}$, $\pi_{XU,i} = 0$, $i = 1...2$;

*iv.* $Y \overset{1}{\nrightarrow} (X, V)|I_{XZ}$, $V \overset{1}{\nrightarrow} (X, U)|I_{XZ}$, $\pi_{XU,i} = 0$, $i = 1...2$.

### 3.4 Multivariate $U$ and $Z \rightarrow X$

Let $Y \overset{1}{\nrightarrow} X|I_{XZ}$, $Y \overset{(h)}{\nrightarrow} X|I_{XZ}$, $h \geq 1$, and $Y \overset{1}{\rightarrow} U|I_{XZ}$. In the multivariate case ($m_u > 1$), even if $Y \overset{1}{\rightarrow} U|I_{XZ}$ such that at least one $\pi_{UY,j} \neq 0$, it is no longer implied recursively that $Y \overset{h+1}{\nrightarrow} X$ *if and only if* $\pi^{(h)}_{XU,1} = 0$. In this case, in general $\pi^{(h)}_{XU,1}\pi_{UY,j} = 0$ can clearly occur with $\pi^{(h)}_{XU,1} \neq 0$ due to the nonlinear row-column combinations.

While $\pi^{(h)}_{XU,1} = 0$ is no long necessary for noncausality, it is sufficient. With this in mind, and with small modifications to the statement and proof of Lemma 3, each result contained in Theorems 4 and 6, and Corollary 5, holds sequentially

as *sufficient* conditions for $\pi^{(h)}_{XU,1} = 0$. Therefore, use of the compound restrictions detailed in Theorem 6 as a basis for test hypotheses should be guarded: rejection of any such hypothesis cannot be interpreted as evidence in favor of causation, because causal neutralization may hold.

There are still, however, many ways to inspect simplifying linear sufficient conditions. For example, delineating $U = (U_1, ..., U_{m_u})$, if $U_i \overset{1}{\nrightarrow} X$ for each $i$ except $U_k \overset{1}{\rightarrow} X$, then $Y \overset{(h+1)}{\nrightarrow} X|I_{XZ}$ *if and only if* $\pi_{XU_k,i} = 0$, $i = 1...h$.

As another example, rather than define $Z = (U', V')'$ based on $Y \overset{1}{\rightarrow} Z$, we may inspect the causal route from $Z$ to $X$. Provided $Z \overset{1}{\rightarrow} X|I_{XY}$, partition $Z$ into the sub-vectors $Z = (S', T')'$ where $S \overset{1}{\rightarrow} X|I_{XYT}$, $T \overset{1}{\nrightarrow} X|I_{XYS}$ and $m_t = 0$ is possible. If $m_s = 1$ and $Y \overset{1}{\rightarrow} S|I_{XZ}$, then Theorem 4.$i$ holds with $U$ replaced by $S$. The following result can be proved a the manner similar to Lemma 3 and Theorem 4: see Hill (2005).

**Corollary 7** *Consider the VAR process* (2), *and partition* $Z = (S', T')'$ *such that* $S \overset{1}{\rightarrow} X|I_{XYT}$. *Assume* $Y \overset{1}{\rightarrow} S|I_{XZ}$, $m_s = 1$ *and* $Y \overset{(h)}{\nrightarrow} X$. *Then* $Y \overset{(h+1)}{\nrightarrow} X$ *if and only if* $\pi_{XS,h} = 0$.

### 3.5 Multivariate $U$, $S$

If both processes $U$ and $S$ are multivariate, then causal neutralization becomes an imposing issue, and rejection of noncausality sufficient conditions cannot be equated to causality. In this case, nonlinear parametric conditions for noncausality are likely to pervade. However, as pointed out above, there are still cases in which $U$ and $S$ are multivariate and linear conditions for noncausation can be established, depending on the causal relationships within and between the components of $U$ and $S$.

**4. Tests for Causation at Multiple Time Horizons** We now construct a strategy for testing noncausality up to horizon $h = 3$. Consult Tables 1-2 of Appendix 1 for a consolidated list and detailed orders of the enumerated hypotheses and equivalent tests detailed in this section. Similarly, because every case in our study of Section 6 involves an evidently univariate $U$, we tabulate the following test details in Table 3 for this univariate $U$-case in terms of Models 1 and 2 ($\Delta m, \Delta y, \Delta p, \Delta r$) of Section 6.

**1**: **Initial Tests 0.1-0.2** ($Y \overset{(\infty)}{\nrightarrow} X$), **Test 1.0**$^*$ ($Y \overset{1}{\nrightarrow} X$)

Either condition $Y \overset{1}{\nrightarrow} (X, Z)$ or $(Y, Z) \overset{1}{\nrightarrow} X$ is sufficient for non-causation at all horizons, cf. Theorem 2. Evidence in favor of either hypothesis provides evidence in favor of $Y \overset{(\infty)}{\nrightarrow} X|I_{XZ}$, and we stop the test procedure. If we reject both sufficiency conditions, we test[8]

$$Y \overset{1}{\nrightarrow} X. \qquad \text{(Test 1.0}^*\text{)}$$

[8] We use an asterisk to denote tests that represent conditions which are necessary and sufficient for noncausation $Y \overset{(h)}{\nrightarrow} X$. The condition of Test 1.0$^*$ is by definition necessary and sufficient for $Y \overset{1}{\nrightarrow} X$.

If we find evidence in favor one-step ahead non-causation, we proceed.

**2.A (Univ. Z): Test** $\pi_{XZ,h} = 0$

By Theorem 4, sequential evidence in favor of $\pi_{XZ,h} = 0$, is evidence in favor of non-causation up to horizon $h + 1$. No further steps are required: test the linear compound hypotheses

$$H_0 : Y \overset{1}{\nrightarrow} X, \pi_{XZ,i} = 0, i = 1...h$$

for each $h = 1, 2, ...$ Failure to reject provides evidence in favor of $Y \overset{(h+1)}{\nrightarrow} X$.

**2.B (Mult. Z): Intermediary Tests 1.1-1.2 ($Y \overset{1}{\nrightarrow} V$)**

In order to proceed with multiple horizon tests and multiple possible causal chain structures, we need to know which processes $Y$ causes (the $U's$), and which cause $X$ (the $S's$). We therefore perform *intermediary* one-step ahead causality tests. We test

$$Y \overset{1}{\nrightarrow} Z_i \tag{Test 1.1}$$

for each $i = 1...m_z$, and collect $U = (Z_i)$ for each $Z_i$ such that we reject $Y \overset{1}{\rightarrow} Z_i$. Then, test each

$$Z_i \overset{1}{\nrightarrow} X \tag{Test 1.2}$$

and collect $S = (Z_i)$ for each $Z_i$ such that we reject $Z_i \overset{1}{\rightarrow} X$. We proceed to Step 3.A or 3.B depending on the dimension of $U$[9].

**3.A (Univariate U): Compound Tests** $H_0^h : Y \overset{(h)}{\nrightarrow} X$

**3.A.1 Test 2.0*** **($Y \overset{(2)}{\nrightarrow} X$)**

With the sub-vectorization $Z = (U', V')'$ in hand, and $m_u = 1$, we may immediately test for noncausation through horizon $h = 2$. From Theorem 6, a compound test of

$$H_0 : Y \overset{1}{\nrightarrow} (X, V),\ \pi_{XU,1} = 0 \tag{Test 2.0*}$$

is a necessary and sufficient test of $H_0 : Y \overset{(2)}{\nrightarrow} X$. If we fail to reject, we proceed to Step 3.A.2.

**3.A.2 Compound Tests 2.1-2.2, and Tests 3.1-3.2 ($Y \overset{(3)}{\nrightarrow} X$)**

For tests of $Y \overset{(3)}{\nrightarrow} X$, we first approach sufficient conditions which help establish a *lack of causal neutralization* (and therefore *complete noncausation*) by performing the compound tests of

$$H_0 : Y \overset{1}{\nrightarrow} (X, V),\ U \overset{1}{\nrightarrow} V \tag{Test 2.1}$$

and

$$H_0 : Y \overset{1}{\nrightarrow} (X, V),\ V \overset{1}{\nrightarrow} (X, U). \tag{Test 2.2}$$

[9] If we detect $Y \overset{1}{\nrightarrow} Z_i$ for each $i = 1...m_z$ such that $m_u = 0$, in the present section by convention we assume the test process is stopped. We proceed to test $Y \overset{(h)}{\nrightarrow} X$, $h \geq 2$, only if evidence of a causal chain is present (in particular only if we detect $Y \overset{1}{\rightarrow} Z_i$ for some $i$).

If we fail to reject either Test 2.1 or 2.2, from Theorems 4 and 6 compound tests of

$$H_0 : Y \overset{1}{\nrightarrow} (X,V),\ U \overset{1}{\nrightarrow} V,\ \pi_{XU,1} = \pi_{XU,2} = 0 \qquad \text{(Test 3.1)}$$

or

$$H_0 : Y \overset{1}{\nrightarrow} (X,V),\ V \overset{1}{\nrightarrow} (X,U),\ \pi_{XU,1} = \pi_{XU,2} = 0 \qquad \text{(Test 3.2)}$$

are tests of *complete* $H_0 : Y \overset{(3)}{\nrightarrow} X$. Failure to reject either Test 3.1 or 3.2 suggests noncausation up to horizon $h = 3$ *without causal neutralization* (because links $Y \overset{1}{\to} V$, $U \overset{1}{\to} V$ or $V \overset{1}{\to} (X,U)$ do not exist). Rejection of Test 3.1 or 3.2 after a failure to reject the necessary and sufficient Test 2.0*, and a failure to reject Test 2.1 or 2.2, implies $Y \overset{(2)}{\nrightarrow} X$ and $Y \overset{3}{\to} X$.

For the sake of convention, if we fail to reject Test 2.1, then we simply perform Test 3.1 and stop. If we reject Test 2.1, then we perform Test 2.2 and proceed.

Rejection of both compound Tests 2.1 and 2.2 implies we must inspect the necessary and sufficient for $Y \overset{(3)}{\nrightarrow} X$, and consider causal neutralization: in this case, we proceed to the Step 3.A.3.

#### 3.A.3 Tests 3.3* ($Y \overset{(3)}{\nrightarrow} X$)

If we fail to reject Test 2.0* ($Y \overset{(2)}{\nrightarrow} X$), then $Y \overset{1}{\nrightarrow} (X,V)$, $\pi_{XU,1} = \pi_{XU,2} + \pi_{XV,1}\pi_{VU,1} = 0$ is necessary and sufficient for $Y \overset{(3)}{\nrightarrow} X$. We test

$$\begin{aligned} H_0 \ : \ & Y \overset{1}{\nrightarrow} (X,V) \\ & \pi_{XU,1} = 0,\ \pi_{XU,2} + \pi_{XV,1}\pi_{VU,1} = 0. \end{aligned} \qquad \text{(Test 3.3*)}$$

Rejection implies $Y \overset{(2)}{\nrightarrow} X$ and $Y \overset{(3)}{\to} X$. Failure to reject implies $Y \overset{(3)}{\nrightarrow} X$ with possible *causal neutralization*, hence we proceed.

#### 3.A.4 Tests 3.4-3.6 ($Y \overset{(3)}{\nrightarrow} X$ and causal neutralization)

We have arrived here if we fail to reject each of $Y \overset{1}{\nrightarrow} X$ (Test 1.0*), the necessary and sufficient condition for $Y \overset{(2)}{\nrightarrow} X$ (Test 2.0*), and the necessary and sufficient condition for $Y \overset{(3)}{\nrightarrow} X$ (Test 3.3*). We now test

$$H_0 \ : \ Y \overset{1}{\nrightarrow} (X,V),\ \pi_{XU,1} = \pi_{XU,2} = 0 \qquad \text{(Test 3.4)}$$

$$H_0 \ : \ Y \overset{1}{\nrightarrow} (X,V),\ \pi_{XU,1} = \pi_{XU,2} = \pi_{VU,1} = 0 \qquad \text{(Test 3.5)}$$

$$H_0 \ : \ Y \overset{1}{\nrightarrow} (X,V),\ \pi_{XU,1} = \pi_{XU,2} = \pi_{XV,1} = 0. \qquad \text{(Test 3.6)}$$

If we fail to reject Tests 3.3*, 3.4 and either 3.5 or 3.6, then we have evidence that $\pi_{XU,2} + \pi_{XV,1}\pi_{VU,1} = 0$, $\pi_{XU,2} = 0$ and $\pi_{VU,1} = 0$ and/or $\pi_{XV,1} = 0$. In this case, causal neutralization is ruled out, and we conclude $Y \overset{(3)}{\nrightarrow} X$ *completely*.

If we fail to reject Tests 3.3*, 3.4 and reject both Tests 3.5 and 3.6, then we have evidence $\pi_{XU,2} + \pi_{XV,1}\pi_{VU,1} = 0$ and $\pi_{XU,2} = 0$, implying $\pi_{XV,1}\pi_{VU,1} = 0$, hence $\pi_{VU,1} \neq 0$ and $\pi_{XV,1} \neq 0$ implies causal neutralization is possible. If $m_v = 1$, then causal neutralization has occurred. If we fail to reject Test 3.3*, but reject each of Tests 3.4-3.6, we conclude causal neutralization has occurred at horizon $h = 3$.

Test sequences in the univariate $U$-case are summarized in Table 1, below:

**Table 1: Test Sequences**

| | | | | | | |
|---|---|---|---|---|---|---|
| Test 0.1 | fail to reject → stop | | | | | |
| Test 0.2 | fail to reject → stop | | | | | |
| reject ↓ | | | | | | |
| Test 1.0* | reject → stop | | | | | |
| fail to reject ↓ | | | | | | |
| Test 1.1[a] | $m_u$=0 → stop | | | | | |
| $m_u$=1 ↓ | | | | | | |
| Test 1.2 | | | | | | |
| Test 2.0* | reject → stop | | | | | |
| fail to reject ↓ | | | | | | |
| Test 2.1 | reject → | Test 2.2 | reject → | Test 3.3* | reject → stop | |
| fail to reject ↓ | | fail to reject ↓ | | fail to reject ↓ | | |
| Test 3.1 | | Test 3.2 | | Test 3.4 | | |
| stop | | stop | | Test 3.5 | | |
| | | | | Test 3.6 | | |

Notes: a. If we detect $m_u > 1$ and decide to proceed to Tests 1.2-3.6, then it is understood that the test hypotheses represent sufficient conditions only.

**3.B (Multivariate U)**

In this case, the sequence of hypotheses of Step 3.A can be used to test the sufficient condition $\pi^{(h)}_{XU,1} = 0$ for noncausality. Rejection, however, of either any of Tests 2.0*-3.3* cannot be interpreted as evidence in favor of causality. It would be worthwhile then to pursue the logic of Section 3 in order to deduce further possible linear sufficient conditions, or consider the direct $h$-step ahead test procedure of Dufour *et al* (2003) in the event we exhaust the possibility of analyzing in a simple way causal neutralization and its implications for subsequent nonlinear coefficient zero conditions.

## 5. Size Bounds and Size Distortions

### 5.1 Size Bounds

Consider testing for $Y \overset{(2)}{\nrightarrow} X$. By convention, we proceed to multiple-horizon tests only if we detect a causal chain through $Z$ (i.e. only if we detect $m_u \geq$

1). The necessary and sufficient condition is $Y \overset{1}{\nrightarrow} (X, V)$, $\pi_{XU,1} = 0$, for some mutually exclusive sub-vectorization $Z = (U', V')'$, $Y \overset{1}{\rightarrow} U$, provided $m_u = 1$. We sequentially choose $U$ and $V$ based on preliminary tests of $Y \overset{1}{\nrightarrow} Z_i$ for each $i = 1...m_z$[10]. If $V$ contains elements caused by $Y$, then $Y \overset{1}{\nrightarrow} (X, V)$ cannot hold: in this case, a consistent test method will detect with asymptotic probability of one that $Y \overset{1}{\rightarrow} (X, V)$ even if $Y \overset{(2)}{\nrightarrow} X$ is true.

Of course, if $Y \overset{1}{\nrightarrow} Z_i$ for some $Z_i$, we will incorrectly reject $Y \overset{1}{\nrightarrow} Z_i$ asymptotically with probability, say, $\alpha_z$. For example, let $U = Z_1$. If a consistent test method is used, then asymptotically there is a probability one that we detect $Y \overset{1}{\rightarrow} Z_1$, but suppose that we detect $Y \overset{1}{\rightarrow} Z_i$ for $i = 1, 2$ (i.e. we use $U = [Z_1, Z_2]$). In this case the methodology of Section 4 remains valid as tests of *sufficient* conditions for non-causation $Y \overset{(h)}{\nrightarrow} X$.

However, $Y \overset{(2)}{\nrightarrow} X$ can be true with $\pi_{XU_2,1} \neq 0$ (we only require $\pi_{XU_1,1} = 0$, cf. Lemma 3, given $Y \overset{1}{\rightarrow} Z_i$ for only $Z_1$). In this case, if we test $Y \overset{(2)}{\nrightarrow} X$ by testing $Y \overset{1}{\nrightarrow} (X, V)$, $\pi_{XU,1} = [\pi_{XU_1,1}, \pi_{XU_2,1}] = 0$, and if a consistent test method is used, asymptotically there is a probability one that we reject $Y \overset{(2)}{\nrightarrow} X$, and the probability that we reach this point is the probability we incorrectly reject any $Y \overset{1}{\nrightarrow} Z_i$. Of course, this is a moot subject if we do not pursue the test strategy of Section 4 in the case of a detected $m_u > 1$.

If $m_z \leq 3$, as in our study of money and income, then we have the following result. Consult Hill (2005) for a proofs of Lemmas 8 and 9.

**Lemma 8** *Denote by $\alpha_z$ the common nominal size of each individual test of $Y \overset{1}{\nrightarrow} Z_i$, $i = 1...m_z$. Then* i. *if $m_u = 0$, there is at most a probability of $m_z \times \alpha_z$ that we detect $m_u > 0$. Furthermore,* ii. *if $m_u = 1$ and $m_z \leq 3$, the likelihood that we correctly select $U$ is at least as large as $1 - (m_z - 1)\alpha_z$ provided a consistent test method is employed.*

*Remark* 1: It is recommended that we set the size of tests of $Y \overset{1}{\nrightarrow} Z_i$ very low (e.g. $\alpha_z = .01$): with $\alpha_z = .01$, $m_z = 3$, and $m_u = 1$ there is at least a 98% likelihood that we correctly specify $U$.

With respect to performing sequential tests in order to arrive at tests of $Y \overset{(2)}{\nrightarrow} X$ or $Y \overset{(3)}{\nrightarrow} X$, a Bonferroni-type bounds suffices for analyzing test size.

**Lemma 9** *Let $\alpha_{\#,\#}$ denote the nominally chosen significance level for Test #.#. For each $h = 1, 2, 3$, define the hypothesis $H_0^{(h)} : Y \overset{(h)}{\nrightarrow} X$. Assume $U$ is*

[10] While it is interesting in its own right whether $Y \overset{1}{\nrightarrow} Z$ jointly (which we test for in any case, and is sufficient for non-causality at all horizons), we must *necessarily* perform individual tests of $Y \overset{1}{\nrightarrow} Z_i$ in order precisely to adduce $U = (Z_i)$ based on evidence in favor of $Y \overset{1}{\rightarrow} Z$. The individual tests of $Y \overset{1}{\nrightarrow} Z_i$ are not treated as a sequential substitute for testing the joint hypothesis of $Y \overset{1}{\nrightarrow} Z$.

*correctly selected, and* $m_u = 1$. *Then if a consistent test method employed, as* $n \to \infty$

$$\begin{aligned} P\left(\textit{reject } H_0^{(1)} | H_0^{(1)} \textit{ is true}\right) &\leq \alpha_{1.0} \\ P\left(\textit{reject } H_0^{(2)} | H_0^{(2)} \textit{ is true}\right) &\leq \alpha_{1.0} + \alpha_{2.0} \\ P\left(\textit{reject } H_0^{(3)} | H_0^{(3)} \textit{ is true}\right) &\leq \alpha_{1.0} + \alpha_{2.0} + \alpha_{3.1} + \alpha_{3.2} + \alpha_{3.3}. \end{aligned} \tag{11}$$

*Remark* 1: The test for $Y \overset{1}{\nrightarrow} X$ has a bounded size of $\alpha_{1.0}$ because we only perform Test $1.0^*$ if we reject both sufficient conditions for $Y \overset{(\infty)}{\nrightarrow} X$, cf. Tests 0.1 and 0.2.

*Remark* 2: Assuming $U$ is correctly selected and $m_u = 1$, if we set $\alpha_{1.0} = \alpha_{2.4} = \alpha_{3.1} = \alpha_{3.2} = \alpha_{3.3} = \alpha$, say, then $P(\text{reject } H_0^{(3)} | H_0^{(3)} \text{ is true}) \leq 5 \times \alpha$. If we set each nominal level to .01-.02, then assuming $U$ is correctly selected and $m_u = 1$ the upper bound probability of a Type I error for a test of $H_0^{(3)} : Y \overset{(3)}{\nrightarrow} X$ is 5%-10%.

*Remark* 3: If $m_u \geq 1$ is true and $m_u > 1$ detected, the hypotheses associated with $Y \overset{(h)}{\nrightarrow} X$, $h \geq 2$, cf. Section 4, are still valid as sufficient conditions for non-causality. If we reject any of Tests $2.0^*$-$3.3^*$, we cannot conclude causation, and any conclusion of causation as a matter of practice necessarily increases the probability of a Type I error. In this case, the above bounds are not valid and can be straightforwardly evaluated in manner similar to the line of proof.

Finally, suppose *complete* noncausation $Y \overset{(3)}{\nrightarrow} X$ is true. Then by Theorem 4.*ii*, $\pi_{XU,1} = 0$, by Corollary 5.*i*, $\pi_{XU,2} + \pi_{XV,1}\pi_{VU,1} = 0$, and by completeness it must be the case that $\pi_{XU,2} = 0$, and $\pi_{XV,1} = 0$ and/or $\pi_{VU,1} = 0$. The following result can be proved using logic identical to the line of proof of Lemma 9. We reject *complete* noncausation $Y \overset{(3)}{\nrightarrow} X$ if we reject noncausation $Y \overset{(3)}{\nrightarrow} X$, or accept $Y \overset{(3)}{\nrightarrow} X$ but deduce there is neutralization.

**Lemma 10** *Let* $H_0^{(3,c)}$ *denote the hypothesis that* $Y \overset{(3)}{\nrightarrow} X$ *occurs completely, and without causal neutralization. Assume* $U$ *is correctly selected, and* $m_u = 1$. *Then if a consistent test method employed, as* $n \to \infty$

$$\begin{aligned} P\left(\textit{reject } H_0^{(3,c)} | H_0^{(3,c)} \textit{ is true}\right) \leq\ & \alpha_{1.0} + \alpha_{2.0} + \alpha_{3.1} + \alpha_{3.2} \\ & + \alpha_{3.3} + \alpha_{3.4} + \min\{\alpha_{3.5}, \alpha_{3.6}\}. \end{aligned} \tag{12}$$

### 5.2 Size Distortions

It is well known that Wald tests based on multivariate time-series models tend to lead to over rejections of true null hypotheses when either the chi-squared or $F$ distribution is used, (e.g. Dufour *et al*, 2003; Dufour and Jouini, 2003; Lütkepohl and Müller, 1994; Lütkepohl and Burda, 1997). However, these studies do not use the type of sequential hypotheses proposed here, nor do they consider the performance of multi-step ahead causality tests when non-causation

truly occurs at all horizons or after discrete delays. Consult Appendix 2 in which we perform a broad simulation study using VAR(6) $m$-vector processes, $m = 5$. We demonstrate that a sequential test, based on pre-testing for non-causation at all horizons, essentially eliminates size distortions with respect to the classic test of 1-step ahead non-causation when causation never occurs; tames size distortions of tests of $Y \overset{1}{\nrightarrow} X$ when causation truly occurs at horizon $h > 1$; and can be argued to improve power with respect to the detection of causation[11].

**6. Money and Output** We now employ the set of variables studied in the widely cited works of Stock and Watson (1989) and Friedman and Kuttner (1993), and others (see also, e.g., Swanson, 1998). For the period Jan. 1959 - Aug. 2003, we use the logarithm of monthly, seasonally adjusted, nominal $M1$ ($m$), the logarithm of unadjusted output measured by the industrial production index ($y$), the logarithm of the wholesale price index ($p$), the 90-day Treasury bill rate ($r_b$), and the 90-day commercial paper rate ($r_p$).

Except for the commercial paper rate, all data are taken from the databases made publicly available by the Federal Reserve Bank of Saint Louis, and seasonal adjustment, where applicable, was performed at the source. The commercial paper rate was taken from the NBER data archive for the period 1959:01-1971:12, and from the Federal Reserve Bank of Saint Louis for the period 1972:01-2003:08. All variables are differenced once based on significant evidence in favor of one positive unit root in each series, giving $\Delta m, \Delta y, \Delta p, \Delta r_b, \Delta r_p$. Moreover, following Friedman and Kuttner (1992, 1993) we also consider the commercial paper-bill rate spread $rr_{pb} = r_p - r_b$ in levels: unit-root tests suggest the two series $r_p$ are cointegrated such that $r_p - r_b$ is $I(0)$, hence differencing is not required.

In order to control for any apparent trend in any sample period considered, we opt to pass all 5 processes $\Delta m, \Delta y, \Delta p, \Delta r_b, \Delta r$ and the rate spread $r_p - r_b$ though linear trend filters. Test results for processes passed through quadratic trend filters are nearly identical to those reported below for linearly detrended processes[12].

[11] In simulations not reported here we also consider a parametric bootstrap technique to better approximate test statistic $p$-values. Gor a given nominal size, the parametric bootstrap in general leads to sharp emprical size improvements, but at a non-neglibile drop in empirical power. See, e.g., Hill (2004) for evidence of the parametric bootstrap in triviate causal systems. Separately, we considered 5-vector systems identical to the processes considered in Appendix 2, below, and find empirical sizes of sequential tests reasonably near nominal levels, once size bounding is acounted for. However, obvious power limitations exist with respect to the detection of causation at $h \geq 2$. Because asymptotic tests do not demonstrate distortions with respect to tests of $Y \overset{1}{\nrightarrow} X$ when non-causation occurs at all horizons and we pre-test for $Y \overset{(\infty)}{\nrightarrow} X$, and generate near perfect power when $Y \overset{1}{\rightarrow} X$, we do not find the parametric bootstrap a convincing alternative to standard test techniques when a sequential test method is employed. Because of space limitations, we leave for future research a study of the performance of the parametric bootstrap for sequential tests of multiple horizon non-causation.

[12] It is interesting to point out that the primary trend arguments of Stock and Watson (1989) are no longer significant from a statistical perspective. Their argument that detrended money growth is the imperative measure of money in the money-income model, due to significant

Following Stock and Watson (1989) and Friedman and Kuttner (1992, 1993), we consider 4-variable models of money growth, income growth, inflation, and fluctuations in interest rates; and 5-variable models with the rate spread included in order to control for the apparent predictive power of the commercial paper rate. In order to allow for direct comparisons with existing studies, we consider sample periods studied in Stock and Watson (1989): 1959-1985; Friedman and Kuttner (1993): 1959-1990; and an extended sample period from 1959 through August of 2003.

**6.1 Money, Output, Inflation, Interest** We first consider the four-variable system of money growth $\Delta m$, output growth $\Delta y$, inflation $\Delta p$, and interest fluctuation $\Delta r$, where $r$ denotes either the Treasury bill or commercial paper rates. The models are respectively Model 1 $(\Delta m, \Delta y, \Delta p, \Delta r_b)$ and Model 2 $(\Delta m, \Delta y, \Delta p, \Delta r_p)$.

For each period, we estimate a VAR model, where the $\Delta y$-representation follows,

$$\begin{aligned} \Delta y_t &= \sum_{i=1}^{p} \pi_{yy,i} \Delta y_{t-i} + \sum_{i=1}^{p} \pi_{ym,i} \Delta m_{t-i} \\ &\quad + \sum_{i=1}^{p} \pi_{yr,i} \Delta r_{t-i} + \sum_{i=1}^{p} \pi_{yp,i} \Delta p_{t-i} + \epsilon_{y,t}. \end{aligned} \tag{13}$$

The order $p$ is selected by minimizing the AIC, subject to reasonably noisy residuals[13]. In general, we follow the model selection methods of Tiao and Box (1981) and Lütkepohl (1991)[14].

evidence that money growth was increasing over time, while important in its time no longer statistically captures the basic traits of the data. In the extended period 1959-2003, we find that money growth now demonstrates a slight, but significant, inverted quadratic trend, undoubtedly a remnant of spurious cycle properties of the 1990's. Similar evidence exists for the wholesale price index, however evidence of either linear or quadratic trend in each differenced interest rate series is insignificant at the 5%-level.

The level rate spread $rr_{pb}$ however, demonstrates a significant positive trend with $r_{b,t} - r_{p,t} < 0$ substantially in the 1980's, nearing 0 in the 1990's. This can be explained by the recessionary periods of the mid-1970's and mid-1980's during which time bankruptcies lead to a trend of decreased bond ratings for firms and therefore a tendency for the commercial paper rate to increase; and the decrease risk associated with the rampant growth period of the 1990's. It could be argued that the trend will not continue ($r_{b,t} - r_{p,t} > 0$ is highly unlikely), and any statistical detection is spurious to the chosen sample.

[13] The SIC never leads to a VAR model with sufficiently noisy residuals. We opt, therefore, only to use the AIC.

[14] For the system including the Treasury bill rate, $\Delta m, \Delta y, \Delta p, \Delta r_b$, and for both truncated periods through 1985 and 1990, a VAR(6) model was found to be optimal: both the AIC was minimized and the standard vector-version of the Ljung-Box test failed to reject the white-noise null at the 10% level in which 12 and 24 residual autocorrelations were used. For the extended period through Aug. 2003, the minimum AIC model occurred with $p = 13$, however evidence of linear dependence exists in the residuals. Models with orders 18 and 24, however, were only slightly sub-optimal relative to the AIC, and we failed to reject the hypothesis of white-noise at the 5% and 8%-levels, respectively. Because a slight improvement with respect to residual noisiness occurs with order $p = 12$, and all subsequent substantive results remain the same, we opt for this latter specification. In any case, for all VAR models of orders less than 14 and for all periods discussed above or below, VAR polynomials are stable.

Simulated size distortions do not exist for tests for noncausation 1-step ahead when non-causation never occurs for moderate sample sizes, or when they do exist they favor correctly detecting causation if it occurs at all (although, potentially earlier than when it occurs: see Appendix 2). Moreover, a parametric bootstrap generates a non-negligible reduction in empirical power[15]. Taking these two issues into consideration as well as space considerations, and in order to improve comparability with Stock and Watson's and Friedman and Kuttner's original results, we perform all tests using a degree-of-freedom corrected Wald statistic, we compute $p$-values based on the $F$ distribution using Lütkepohl's (1991) suggestions for degrees of freedom corrections, and we consider size bounds implied by Section 5.1.

Because we always find significant evidence in favor of $m_u = 1$, we do not pursue further discussion concerning the likelihood of detecting $m_u > 1$.

**6.1.1 Causation Results: Model 1** $(\Delta m, \Delta y, \Delta p, \Delta r_b)$

Consider the model with the Treasury bill rate $\Delta r_b$: results are contained in Table 4.1 in Appendix 1. Tables 4.1-4.4 contain composite test results for all initial tests in the first three rows (i.e. $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ and $\Delta m \overset{1}{\nrightarrow} \Delta y$), all intermediary tests (e.g. $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p)$) and all compound tests of multiple horizon noncausation in the bottom rows (i.e. $\Delta m \overset{(2)}{\nrightarrow} \Delta y$ and $\Delta m \overset{(3)}{\nrightarrow} \Delta y$). Consult Tables 1-3 for test details and sequential orders in terms of $X, Y, Z$ and $\Delta m, \Delta y, \Delta p, \Delta r$. Within each of Tables 4.1-4.4, we remark on the specific test order used based on sequential test results. Our aim is to perform the minimum number of tests required to ascertain plausible causal routes, if any, and whether causal neutralization has occurred. Thus, for each period and each model we do not present results for all tests presented in Tables 1-3.

**Model 1: 1959-1985**
$\Delta m \overset{1}{\rightarrow} \Delta y$ **vs.** $\Delta m \overset{2}{\rightarrow} \Delta y$

For the truncated period[16] 1959:01-1985:12 a la Stock and Watson (1989) we reject initial sufficient conditions for $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ at the 5%-level (Test 0.1: .0000; Test 0.2: .0455)[17], and reject the classic null hypothesis $\Delta m \overset{1}{\nrightarrow} \Delta y$ that money growth does not cause real income growth (Test 1.0*: .0181). For the intermediate period 1959-1990, a la Friedman and Kuttner (1993), we fail to

For VAR systems including the commercial paper rate, $\Delta m, \Delta y, \Delta p, \Delta r_p$, for each sample period we found VAR(6), VAR(6) and VAR(8) models, respectively, to be superior. Both truncated periods rendered sufficiently noisy residuals, however for the extended period the largest Ljung-Box $p$-value was roughly 4%, obtained for a VAR(18) model. The VAR(8) model generated only slightly more noisy residuals, and obtained the lowest AIC: we again side with parsimony, and employ the order $p = 8$.

[15] See footnote 11.

[16] Due to the removal of observations that naturally occurs when lagging for estimation, the resulting sample period is 1959:07-1985. However, for simplicity we refer to the orignal pre-esimation periods.

[17] Parenthetic values denote ennumerated test, cf. Tables 1-3, and $p$-values.

reject the claim that money does not cause real income at any standard level of significance (Test 1.0*: .3580). This confirms the substantive results of those separate papers: simply extending the data sample through 1990 renders money a statistically non-influential factor for forecasting output one month ahead. Moreover, for the intermediate period 1959-1990, we fail to reject a sufficient test of $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ (Test 0.2: .1205) at the 10%-level weakly suggesting non-causation at all horizons.

For the extended period 1959-2003:08 we reject conditions for $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ (Test 0.1: .0155; Test 0.2: .0049), and we again fail to reject the hypothesis that money does not cause real income at any standard level of significance (Test 1.0*: .6794). Indeed, in this case the test $p$-value substantially increases relative to the 1959-1990 period, loosely suggesting money is now "more trivial" for one-month ahead forecasting of output. In this extended period, we reject each sufficient condition for $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ at below the 1%-level.

For the initial period 1959-1985, rejection of $\Delta m \overset{1}{\nrightarrow} \Delta y$ occurs at the 2%-level (Test 1.0*: .0181). In lieu of test size bounds issues, if we opt to fail to reject this test at the 1%-level, say, as a matter of course then subsequent tests suggest a short causal delay. We find $\Delta m \overset{1}{\rightarrow} \Delta r_b$ at a level safely under 1% (Test 1.1.b: .0000) and likely $\Delta r_b \overset{1}{\nrightarrow} \Delta y$ (Test 1.2.b: .1596). Even with this weak evidence in support of a causal link from money to output, if we put $U = \Delta r_b$ we reject the necessary and sufficient condition for $\Delta m \overset{(2)}{\nrightarrow} \Delta y$ (Test 2.0*: .0024), undoubtedly due to the joint presence of coefficient terms for the embedded test of $\Delta m \overset{1}{\nrightarrow} \Delta y$. If we perform both tests of $\Delta m \overset{1}{\nrightarrow} \Delta y$ and $\Delta m \overset{(2)}{\nrightarrow} \Delta y$ sequentially at the 1%-level, we safely reject $\Delta m \overset{(2)}{\nrightarrow} \Delta y$ at the 2%-level. Either way, we have evidence in favor of $\Delta m \overset{1}{\rightarrow} \Delta y$ at the 2%-level, or $\Delta m \overset{1}{\nrightarrow} \Delta y$ and $\Delta m \overset{2}{\rightarrow} \Delta y$ at the 2%-level.

**Model 1: 1959-1990**
$\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ **vs.** $\Delta m \overset{2}{\rightarrow} \Delta y$

Because evidence in favor of $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ is rather weak or strongly rejected (e.g. Test 0.2: .1205), we pursue tests of $\Delta m \overset{(h)}{\nrightarrow} \Delta y$. Evidence supports a broken causal chain in general, $\Delta m \overset{1}{\rightarrow} (\Delta p, \Delta r_p) \overset{1}{\nrightarrow} \Delta y$, specifically via $\Delta m \overset{1}{\rightarrow} \Delta r_p \overset{1}{\nrightarrow} \Delta y$[18]. From Theorem 2, this provides moderate evidence not only that money growth fails to cause output growth one-month ahead, but the far

[18] We find evidence that $\Delta m \overset{1}{\rightarrow} (\Delta p, \Delta r_p)$ only though the Treasury bill rate $\Delta r_p$: we reject $\Delta m \overset{1}{\nrightarrow} (\Delta p, \Delta r_p)$ (Test 1.1: .0000) and reject only $\Delta m \overset{1}{\nrightarrow} \Delta r_p$ safely under the 1%-level (Test 1.1.b: .0000). However, we fail to reject the hypothesis that all of the auxiliary information $Z = (\Delta r_b, \Delta p)$ is non-causal for output growth $\Delta y$ (Test 1.2: .1371): thus, evidence suggests $(\Delta p, \Delta r_b) \overset{1}{\nrightarrow} \Delta m$ (i.e. $Z \overset{1}{\nrightarrow} X$). In particular, we fail to reject the hypothesis that the Treasury bill rate $\Delta r_b$ causes output growth $\Delta y$ at about the 10%-level (Test 1.2.b: .1009).

stronger claim that money growth *never* causes output growth, $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$. Coupled with a sufficient condition test for $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ (Test 0.2: .1205), this strengthens the claim of the "trivial" role of money made by Friedman and Kuttner (1993) for their chosen period.

Note that the levels of hypothesis acceptance above are in the range 10%-15%, hence we could posit hypothesis rejections at these relatively large significance levels. Indeed, we *do reject* an initial sufficient condition for $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ at the 15%-level (Test 0.2: .1205), and specifically the hypothesis that fluctuations in the Treasury bill rate $\Delta r_b$ do not cause output growth $\Delta y$ at roughly the 10%-level (Test 1.2.b: .1009). Thus, we have (admittedly, very) weak evidence of an indirect causal chain $\Delta m \overset{1}{\rightarrow} \Delta r_p \overset{1}{\rightarrow} \Delta y$, and therefore evidence that money growth *eventually* causes output growth.

We define[19] $U = \Delta r_b$ and $V = \Delta p$ (i.e. evidence suggests $\Delta m \overset{1}{\rightarrow} \Delta r_b$ and $\Delta m \overset{1}{\nrightarrow} \Delta p$), such that $m_u = 1$, and proceed with compound tests. We reject the hypothesis of non-causation up to horizon $h = 2$, $\Delta m \overset{(2)}{\nrightarrow} \Delta y$, at the 10%-level using the necessary and sufficient condition (Test 2.0*: .0723). Thus, evidence points to causation from money growth to output growth two-months ahead, $\Delta m \overset{1}{\nrightarrow} \Delta y$ and $\Delta m \overset{2}{\rightarrow} \Delta y$. Performing Test 1.0* at the 1%-level and Test 2.0* at the 8%-levels implies a size bounds of 9%, cf. Lemma 9[20].

In summary, for Friedman and Kuttner's (1993) selected sample period evidence in favor of either noncausality at all horizons or a causation delay is rather ambiguous. We fail to reject $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ at the 15% level based on the intermediary test of $(\Delta p, \Delta r_b) \overset{1}{\nrightarrow} \Delta m$ (i.e. $Z \overset{1}{\nrightarrow} X$), or at the 10%-level using the efficient initial test $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ (Test 0.2: .1205). If the decision is to reject these hypotheses at their respective high levels, we fail to reject $\Delta m \overset{1}{\nrightarrow} \Delta y$ and deduce $\Delta m \overset{2}{\rightarrow} \Delta y$ at the bounded 11%-level using a compact necessary and sufficient condition (Test 2.0*: .0723).

**Model 1: 1959-2003**

[19] It is important to point out that we never find $m_u > 1$ for any time period or any model considered in this paper. Indeed, in separate work not reported here in which we study the same data set over rolling windows of increasing and fixed widths set to the sample lengths of Stock and Watson's (1989) and Friedman and Kuttner's (1993) sample periods, we find $m_u = 2$ in under 3% of all windows for all models. When $m_u = 1$, it is always the case that $U = \Delta r$, depending on which rate is included, with test rejections occuring under the 1%-level. However, we frequently encounter a multivariate $S$, thus all compound hypotheses are constructed according to $U$.

[20] For the sake of fluidity, we will not repeatedly comment on sequential test size issues associated with pre-testing for $U$. There exists overwhelming evidence that $m_u \leq 1$ in all periods for all models; Wald statistics have demonstrated power under standard conditions; and we never perform tests of multiple-horizon non-causation for a case where $m_u > 1$ (simply because we never detect $m_u > 1$). Thus, it is highly unlikely the true $U$ is multivariate, and it is highly likely we have selected the true $U$; and we defer to the size bounds characterized by Lemmas 9 and 10.

$$\Delta m \xrightarrow{2} \Delta y$$

Recall that for the extended period 1959-2003:08, we reject both sufficient conditions for $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ (Test 0.1: .0155; Test 0.2: .0049), and fail to reject the classic hypothesis $\Delta m \overset{1}{\nrightarrow} \Delta y$ (Test 1.0*: .6794). We again find strong evidence that $\Delta m \xrightarrow{1} (\Delta p, \Delta r_b)$ (Test: 1.1: .0023) only though the Treasury bill rate $\Delta r_b$ (Test 1.1.b: .0002). However, we now find significant evidence that fluctuations in the Treasury bill rate causes output growth $\Delta y$ (Test 1.2.b: .0004, compared to .1009 for 1959-1990). This suggests a significant indirect causal chain $\Delta m \to \Delta r_b \to \Delta y$ exists through the Treasury bill (i.e. $U = \Delta r_b$). Similar to the intermediate period 1959-1990, we find borderline evidence of causation two months ahead: we reject the necessary and sufficient condition for $\Delta m \overset{(2)}{\nrightarrow} \Delta y$ (Test 2.0*: .0899). If we perform Test 1.0* at the 1%-level and Test 2.0* at the 9%-level, there is a bound of 10% for a Type I error. Thus, evidence is again not strong but certainly worthy of reporting[21].

### 6.1.2 Causation Results: Commercial Paper Rate

Following Friedman and Kuttner (1993), we substitute the Treasury bill rate for the commercial paper rate, $\Delta r_p$, in the VAR model (17). Test results can be found in Table 4.2. For the truncated period 1959-1985 we again reject non-causality 1-month ahead at the 3%-level (Test 1.0*: .0243)[22].

**Model 2: 1959-1990**
$\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ **vs** $\Delta m \overset{(3)}{\nrightarrow} \Delta y$

For the 1959-1990 period and the extended period through 2003:08, however, evidence strongly supports the claim that money is trivial in one-step ahead prediction of output. We reject both sufficient conditions for $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ (Test 0.1: .0000, Test 0.2: .0424) at the 5%-level, and we fail to reject $\Delta m \overset{1}{\nrightarrow} \Delta y$ (Test 1.0*: .3449).

For the period 1959-1990 we fail to reject $(\Delta r_p, \Delta p) \overset{1}{\nrightarrow} \Delta y$ (Test 1.2: .1628) implying a broken chain, $Z \overset{1}{\nrightarrow} X$, and, cf. Theorem 2, which suggests that money

[21] Due to space considerations, we do not pursue tests at, nor discussions regarding, subsequent horizons. Considering simulation evidence, cf. Appendix 2, and the present strong evidence in favor of $\Delta m \overset{1}{\nrightarrow} \Delta y$ and moderate evidence in favor of $\Delta m \xrightarrow{2} \Delta y$, it is likely the case that causation is occuring at a horizon near $h = 2$ (e.g. $h \in \{2, 3, 4\}$). Such a conclusion in lieu of test and simulation results can be made in several cases, below, and we will not comment further.

[22] If we pursue a test of $\Delta m \overset{(2)}{\nrightarrow} \Delta y$, the result is identical to Model 1: we find significant evidence in favor of $\Delta m \xrightarrow{1} \Delta r_p$, and reject $\Delta m \overset{(2)}{\nrightarrow} \Delta y$ at the a bounded 4%-level (Test 2.0*: .0173).

growth never causes output growth, $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$. Using the rate on non-risk free commercial loans for the period 1959-1990, evidence suggests the authors were correct in their assessment of the statistically trivial influence monetary policy is likely to have on matters of (forecasting) real income growth.

Intermediary tests suggest $\Delta m \overset{1}{\rightarrow} \Delta r_p$ (Test 1.1.b: .0000) and likely $\Delta r_p \overset{1}{\nrightarrow} \Delta y$ (Test 1.2.b: .1423). This formally demonstrates a likely broken causal chain. Using $U = \Delta r_p$, we fail to reject the necessary and sufficient condition for $\Delta m \overset{(2)}{\nrightarrow} \Delta y$ (Test 2.0*: .1544). Moreover, we fail to reject $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p)$, $\Delta r_p \overset{1}{\nrightarrow} \Delta p$ (Test 2.1: .2737) hence $\pi_{\Delta y \Delta r,i} = 0$, $i = 1...2$ is necessary and sufficient for $\Delta m \overset{(3)}{\nrightarrow} \Delta y$. We fail to reject $\Delta m \overset{(3)}{\nrightarrow} \Delta y$ at the 10%-level (Test 3.1: .1195). Indeed, sequentially setting the nominal test sizes at 1%-2% for Tests 1.0*, 2.0*, 3.1, 3.2 and 3.3* for a size bounds of 5%–10%, cf. Lemma 9, the present outcome for Test 3.1 suggests we should safely fail to reject the claim $\Delta m \overset{(3)}{\nrightarrow} \Delta y$ at the 10%-level.

Using the commercial paper rate, we conclude that fluctuations in the money supply do not cause output growth at least through 3-months, $\Delta m \overset{(3)}{\nrightarrow} \Delta y$, and without the complication of causal neutralization, and possibly through all horizons $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$.

**Model 2: 1959-2003**
$\Delta m \overset{(3)}{\nrightarrow} \Delta y$

For the extended period, 1959-2003:08, by comparison, sharply ambiguous evidence for a causal delay exists. We reject both sufficient conditions for $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ (Test 0.1: .0572 and Test 0.2: .0008), and strongly fail to reject one–month ahead non-causation $\Delta m \overset{1}{\nrightarrow} \Delta y$ (Test 1.0*: .7389). Moreover, there exists evidence at the nominal 1%-level in support of a causal chain through the paper rate[23], $\Delta m \overset{1}{\rightarrow} \Delta r_p \overset{1}{\rightarrow} \Delta y$. It seems that a causal chain exists and causation from money to output eventually occurs.

However, using $U = \Delta r_p$, we overwhelmingly fail to reject the necessary and sufficient condition for $\Delta m \overset{(2)}{\nrightarrow} \Delta y$ (Test 2.0*: .6540). Now, from compound tests we fail to reject $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p)$, $\Delta r_p \overset{1}{\nrightarrow} \Delta p$ (Test 2.1: .3052) hence $\pi_{\Delta y \Delta r,i} = 0$, $i = 1...2$ is again necessary and sufficient for $\Delta m \overset{(3)}{\nrightarrow} \Delta y$. We subsequently fail to reject the test of $\Delta m \overset{(3)}{\nrightarrow} \Delta y$ (Test: 3.1: 2435).

### 6.2 Money, Output, Inflation, Interest, and Interest Spread

Friedman and Kuttner (1992,1993) found the commercial paper rate provides significant predictive information for output growth. In particular, they found

[23] We find $\Delta m \overset{1}{\rightarrow} (\Delta p, \Delta r_p)$ (Test 1.1: .0109); $\Delta m \overset{1}{\rightarrow} \Delta r_p$ (Test 1.1.b: .0018); $(\Delta p, \Delta r_p) \overset{1}{\rightarrow} \Delta y$ (Test 1.2: .0059); and $\Delta r_p \overset{1}{\rightarrow} \Delta y$ (Test 1.2.b: .0357).

that including the rate spread into models of output with either the Treasury or commercial paper rate rendered money growth non-causal (one-month ahead) in their sample running through 1990. In order to control for the same structural dynamics, consider the following VAR representation for output growth with the rate spread included:

$$\begin{aligned}\Delta y_t &= \sum_{i=1}^{p} \pi_{yy,i}\Delta y_{t-i} + \sum_{i=1}^{p} \pi_{ym,i}\Delta m_{t-i} \\ &+ \sum_{i=1}^{p} \pi_{yr,i}\Delta r_{t-i} + \sum_{i=1}^{p} \pi_{yrr,i} rr_{bp,t-i} \\ &+ \sum_{i=1}^{p} \pi_{yp,i}\Delta p_{t-i} + \epsilon_{y,t}.\end{aligned} \tag{14}$$

The models are, respectively, Model 3 $(\Delta m, \Delta y, \Delta p, \Delta r_b, rr_{bp})$ and Model 4 $(\Delta m, \Delta y, \Delta p, \Delta r_p, rr_{bp})$[24].

### 6.2.0 Causation Results: Model 3

Test results can be found in Tables 4.3 and 4.4. Results are qualitatively similar with either the Treasury bill or paper rates. In both cases, we fail to reject the hypothesis $\Delta m \overset{1}{\nrightarrow} \Delta y$ for *every* period at conventional significance levels, where evidence is somewhat stronger when the commercial paper rate is included, and evidence spectacularly suggests one-month ahead noncausation in the intermediate and extended periods: for models with the Treasury bill rate and rate spread, $p$-values are .9865 and .8676, respectively; and for models with the commercial paper rate, $p$-values are .9870 and .8739, respectively.

**Model 3: Treasury Bill Rate and Rate Spread**
**1959-1985**
**$\Delta m \overset{(3)}{\nrightarrow} \Delta y$ and causal neutralization ?**

Consider the sample period 1959-1985, and consider the model with the Treasury bill rate and rate spread: see Table 4.3. We reject the initial sufficient conditions for $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ (Test 0.1: .0000, Test 0.2: .0000) and fail to reject noncausation 1-month ahead $\Delta m \overset{1}{\nrightarrow} \Delta y$ (Test 1.0*: .1890).

Intermediary tests point to complicated plausible routes from money to output. Evidence strongly points to the causal chain links[25] $\Delta m \overset{1}{\rightarrow} \Delta r_b$ and $rr_{bp}$

[24] For VAR systems including both the Treasury bill rate and the rate spread, $\Delta m, \Delta y, \Delta p, \Delta r_b, rr_{pb}$, we found VAR(5), VAR(13) and VAR(13) models to be superior for the respective periods based on minimizing the AIC. However, for all orders considered (up to 30) we reject the hypothesis that the underlying innovations are white-noise at the 5%-level. Similarly, for VAR systems including both the commercial paper rate and the rate spread, $\Delta m, \Delta y, \Delta p, \Delta r_p, rr_{pb}$, we found VAR(7), VAR(13) and VAR(13) models to be superior for the respective periods based on minimizing the AIC, where again no order rendered evidence of white noise at the 5%-level in the vector residual series.

[25] We strongly reject $\Delta m \overset{1}{\nrightarrow} (\Delta p, \Delta r_b, rr_{bp})$ (Test 1.1: .0000) likely due to $\Delta r_b$ (Test 1.1.b: .0000) again at below the 1%-level. However, we reject $(\Delta p, \Delta r_b, rr_{bp}) \overset{1}{\nrightarrow} \Delta y$ (Test 1.2: .0000) due to $rr_{bp}$: we fail to reject $\Delta r_b \overset{1}{\nrightarrow} \Delta y$ (Test 1.2.b: .2646) and reject $rr_{bp} \overset{1}{\nrightarrow} \Delta y$ (Test 1.2.c: .0001).

$\overset{1}{\rightarrow} \Delta y$. Interestingly, once the rate spread is introduced, evidence for a direct link from money to output through fluctuations in the bill rate $\Delta r_b$ substantially weakens, and may not exist at all.

With evidence once again $U = \Delta r_b$, we pursue multi-step ahead tests. We fail to reject the necessary and sufficient condition for $\Delta m \overset{(2)}{\nrightarrow} \Delta y$ (Test 2.0*: .3359). However, sufficient conditions for $\Delta m \overset{(3)}{\nrightarrow} \Delta y$ detailed in Theorem 6 appear to be strongly ruled out[26].

We now test $\Delta m \overset{(3)}{\nrightarrow} \Delta y$ using a sequentially necessary and sufficient condition, $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p, rr_{bp})$ and $\pi_{\Delta y \Delta r,1} = \pi_{\Delta y \Delta r,2} + \pi_{\Delta y(\Delta p,rr),1}\pi_{(\Delta p,rr)\Delta r,1} = 0$, which we fail to reject at any conventional level (Test 3.3*: .2819). Thus, we must pursue further tests to consider the possibility of causal neutralization.

We fail to reject $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p, rr_{bp})$, $\pi_{\Delta y \Delta r,1} = \pi_{\Delta y \Delta r,2} = 0$ (Test 3.4: .3713), we reject $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p, rr_{bp})$, $\pi_{\Delta y \Delta r,1} = \pi_{\Delta y \Delta r,2} = \pi_{(\Delta p,rr)\Delta r,1} = 0$ (Test 3.5: .0929) only at the 10%-level, and reject $m \overset{1}{\nrightarrow} (\Delta y, \Delta p, rr_{bp})$, $\pi_{\Delta y \Delta r,1} = \pi_{\Delta y \Delta r,2} = \pi_{\Delta y(\Delta p,rr),1} = 0$ (Test 3.6: .0004).

This sequentially suggests $\pi_{\Delta y \Delta r,2} = 0$, $\pi_{(\Delta p,rr)\Delta r,1} \tilde{} \ 0$ (?), and $\pi_{\Delta y(\Delta p,rr),1} \neq 0$. In lieu of the failure to reject Test 3.3*, we have evidence $\pi_{\Delta y \Delta r,2} + \pi_{\Delta y(\Delta p,rr),1}\pi_{(\Delta p,rr)\Delta r,1} = 0$, and $\pi_{\Delta y \Delta r,2} = 0$, hence $\pi_{\Delta y(\Delta p,rr),1}\pi_{(\Delta p,rr)\Delta r,1} = 0$. Because evidence supports $\pi_{\Delta y(\Delta p,rr),1}\pi_{(\Delta p,rr)\Delta r,1} = 0$, $\pi_{(\Delta p,rr)\Delta r,1} \tilde{} \ 0$ (?) and $\pi_{\Delta y(\Delta p,rr),1} \neq 0$, this implies causal neutralization is plausible: fluctuations in the commercial paper rate are causal for inflation and the rate spread, and inflation and the rate spread are causal for output, each in such a way that the combined connective route from money to output cancels out at 3-months ahead.

Given size bounds considerations, cf. Lemmas 9 and 10, and the observing that we *do not reject* at a sufficiently low nominal level (e.g. .5%-1%) the claim that $\pi_{(\Delta p,rr)\Delta r,1} = 0$, cf. Test 3.5, arguably we should side with caution and *fail to reject the claim that noncausation* $\Delta m \overset{(3)}{\nrightarrow} \Delta y$ has occurred *completely.*

### Model 3: 1959-1990
### $\Delta m \overset{(2)}{\nrightarrow} \Delta y$ and $\Delta m \overset{3}{\rightarrow} \Delta y$

Consider the middle period 1959-1990. We substantially fail to reject the benchmark hypothesis $\Delta m \overset{1}{\nrightarrow} \Delta y$ (Test 1.0*: .9865). We reject the sufficient conditions for $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ (Test 0.1: .0011, Test 0.2: .0000). Moreover, each intermediary test outcome is essentially identical to the above period 1959-1985

[26] We strongly reject preliminary sufficient conditions $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p, rr_{bp})$, $\Delta r_b \overset{1}{\nrightarrow} (\Delta p, rr_{bp})$ (Test 2.1: .0071) and $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p, rr_{bp})$, $(\Delta p, rr_{bp}) \overset{1}{\nrightarrow} (\Delta r_b, \Delta y)$ (Test 2.2: .0000). Thus, inspection only of $\pi_{\Delta y \Delta r,i} = 0$, $i = 1, 2$ as a sequentially sufficient condition for noncausality $\Delta m \overset{(3)}{\nrightarrow} \Delta y$ is evidently not an option.

with the notable exceptions that we now reject $\Delta m \overset{1}{\nrightarrow} rr_{bp}$ at the 10%-level (Test 1.1.c: .0885) and strongly reject $\Delta r_b \overset{1}{\nrightarrow} \Delta y$ (Test 1.2.b: .0196). Thus, we find significant evidence in favor of $\Delta m \overset{1}{\rightarrow} (\Delta r_b, rr_{bp}) \overset{1}{\rightarrow} \Delta y$. Because the evidence for $\Delta m \overset{1}{\rightarrow} rr_{bp}$ is somewhat weak (Test 1.1.c: .0885) we define $U = \Delta r_b$ for subsequent tests.

Similar to the period 1959-1985, we fail to reject the necessary and sufficient condition for $\Delta m \overset{(2)}{\nrightarrow} \Delta y$ (Test 2.0*: .2814), and strongly reject the compound tests for $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p, rr_{bp})$, $\Delta r_b \overset{1}{\nrightarrow} (\Delta p, rr_{bp})$ (Test 2.1: .0002) and $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p, rr_{bp})$, $(\Delta p, rr_{bp}) \overset{1}{\nrightarrow} (\Delta r_b, \Delta y)$ (Test 2.2: .0000). Thus, again we must consider testing for $\Delta m \overset{(3)}{\nrightarrow} \Delta y$ using the sequentially necessary and sufficient condition.

We now strongly reject the necessary and sufficient condition for $\Delta m \overset{(3)}{\nrightarrow} \Delta y$ (Test 3.3*: .0008). Even using a size bounding strategy, we safely reject the hypothesis $\Delta m \overset{(3)}{\nrightarrow} \Delta y$ at the 5%-level: we fail to reject Tests 1.0* and 2.0* each at the 1%-level, we reject both Tests 2.1 and 2.2 at the 1%-level, and reject Test 3.3* at the 1%-level. Thus, evidence suggests both non-causation from money to income up to 2-months ahead, and causation at exactly 3-months ahead, $\Delta m \overset{(2)}{\nrightarrow} \Delta y$ and $\Delta m \overset{3}{\rightarrow} \Delta y$.

### Model 3: 1959-2003
### $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ vs. $\Delta m \overset{1}{\nrightarrow} \Delta y$ and $\Delta m \overset{2}{\rightarrow} \Delta y$

For the period 1959-2003:08, we immediately fail to reject the sufficient condition for $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ (Test 0.1: .4575) and the test of $\Delta m \overset{1}{\nrightarrow} \Delta y$ (Test 1.0*: .8676). However, it is important to point out that we do *strongly reject* the hypotheses that money fails to cause the Treasury bill rate (Test 1.1.b: .0063) and the Treasury bill rate fails to cause output (Test 1.2.b: .0010), thus disparate evidence suggests a causal chain may exist, $\Delta m \overset{1}{\rightarrow} \Delta r_b \overset{1}{\rightarrow} \Delta y$. Moreover, now evidence unambiguously suggests money is only causal for the bill rate: we only reject $\Delta m \overset{1}{\nrightarrow} \Delta r_b$ (Test 1.1.b: .0063) and fail to reject $\Delta m \overset{1}{\nrightarrow} \Delta p$ and $\Delta m \overset{1}{\nrightarrow} rr_{bp}$ at any conventional level (Test 1.1.a: .6856, Test 1.1.c: .8897).

We now reject the necessary and sufficient for $\Delta m \overset{(2)}{\nrightarrow} \Delta y$ (Test 2.0*: .0544). If we perform Test 1.0* at the 1%-level and Test 2.0* at the 6%-level, there is a bound of 7% for a Type I error. Thus, evidence sharply splits for non-causation at all horizons, $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$, using an efficient test of a sufficient condition, and a causal delay of one month, $\Delta m \overset{1}{\nrightarrow} \Delta y$ and $\Delta m \overset{2}{\rightarrow} \Delta y$, using sequential necessary and sufficient tests.

### Model 4: Paper Rate and Rate Spread

We now focus on models with the paper rate and rate spread: consult Table 4.4. Results are closely similar to those with the Treasury bill rate.

**Model 4: 1959-1985**
$\Delta m \overset{(2)}{\nrightarrow} \Delta y$ **and** $\Delta m \overset{3}{\rightarrow} \Delta y$

For the early period 1959-1985, we reject both sufficient conditions for $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ (Test 0.1: .0000, Test 0.2: .0000). Evidence again suggests $\Delta m \overset{1}{\nrightarrow} \Delta y$ (Test 1.0*: .2146), with a highly significant link $\Delta m \overset{1}{\rightarrow} \Delta r_p \overset{1}{\rightarrow} \Delta y$. As in every other case above or below, we deduce $U = \Delta r$.

We fail to reject the necessary and sufficient condition for $\Delta m \overset{(2)}{\nrightarrow} \Delta y$ (Test 2.0*: .5621), we fail to reject at the 10%-level a test of $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p, rr_{bp})$, $(\Delta p, rr_{bp}) \overset{1}{\nrightarrow} (\Delta r_p, \Delta y)$ (Test 2.1: .0893), and reject Test 2.2 (.0000). However, considering the evidence is rather weak to allow for the use of the subsequent sufficient condition associated with Test 3.1, we consider only the necessary and sufficient nonlinear condition of Test 3.3*. We strongly reject the necessary and sufficient condition for $\Delta m \overset{(3)}{\nrightarrow} \Delta y$ (Test 3.3*: .0014), suggesting a significant causal delay of exactly two months exists, $\Delta m \overset{(2)}{\nrightarrow} \Delta$ and $\Delta m \overset{3}{\rightarrow} \Delta y$. Using Lemma 9, the upper bound size on the test of $\Delta m \overset{(3)}{\nrightarrow} \Delta y$ is determined only by the nominal sizes (i.e. 1%) of Tests 1.0*, 2.0*, 3.1-3.3*: if we reject Test 2.1 at the 10% level, this does not effect the size bounds on the test of $\Delta m \overset{(3)}{\nrightarrow} \Delta y$. We deduce that we reject $\Delta m \overset{(3)}{\nrightarrow} \Delta y$ at a bounded level of 5%.

**Model 4: 1959-1990**
$\Delta m \overset{(2)}{\nrightarrow} \Delta y$ **and** $\Delta m \overset{3}{\rightarrow} \Delta y$

Causal patterns for the intermediate period are identical, with a sharper degree of significance. In particular, we now reject both initial conditions for $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$, we fail to reject the necessary and sufficient condition for $\Delta m \overset{(2)}{\nrightarrow} \Delta y$ (Test 2.0*: .2187), and we reject Tests 2.1-2.2 at below the 1%-level (Test 2.1: .0000, Test 2.2: .0000). We then reject the necessary and sufficient condition for $\Delta m \overset{(3)}{\nrightarrow} \Delta y$ (Test 3.3*: .0000). We conclude $\Delta m \overset{(2)}{\nrightarrow} \Delta$ and $\Delta m \overset{3}{\rightarrow} \Delta y$ at a bounded 5%-level. It is interesting to point out that the strongest evidence for causation exactly one-quarter ahead occurs in Friedman and Kuttner's (1993) chosen sample period and preferred model, arguably contradicting their results for quarterly data in Friedman and Kuttner (1992).

**Model 4: 1959-2003**
$\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ **vs.** $\Delta m \overset{1}{\nrightarrow} \Delta y$ **and** $\Delta m \overset{2}{\rightarrow} \Delta y$

Finally, for the extended period evidence pushes the likely horizon of causation up to $h = 2$, although we again cannot reject the hypothesis that money

never causes output: we fail to reject a sufficient condition for $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ (Test 0.1: .5920), and we fail to reject $\Delta m \overset{1}{\nrightarrow} \Delta y$ (Test 1.0*: .3739).

Intermediary tests are somewhat ambiguous: we fail to reject $\Delta m \overset{1}{\nrightarrow} (\Delta p, \Delta r_p, rr_{bp})$ (Test 1.1: .3447), providing further support that $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$, however we find a significant causal link $\Delta m \overset{1}{\rightarrow} \Delta r_p \overset{1}{\rightarrow} \Delta y$ (Test 1.1.b: .0352, Test 1.2.b: .0000) although not at the 1%-level. Moreover, we immediately reject the necessary and sufficient condition for $\Delta m \overset{(2)}{\nrightarrow} \Delta y$ (Test 2.0*: .0246) at the 3%-level. Thus, evidence ambiguously suggests a causal delay of exactly one month, $\Delta m \overset{1}{\nrightarrow} \Delta y$ and $\Delta m \overset{2}{\rightarrow} \Delta y$ at a bounded 4%-level, or non-causation at all horizons.

**7. Conclusion** We have derived simple *linear* recursive parametric representations of causality chains for multivariate processes and arbitrary horizon causation problems that point to simplified linear compound hypotheses in several useful cases. Moreover, unavoidable nonlinear restrictions result in standard Wald tests with the usual asymptotics.

A simulation study demonstrates that sequential Wald tests of non-causality with increasingly complex parameter hypotheses do not uniformly result in over-rejection of the "null" hypothesis. If non-causality occurs at every horizon, there does not exist a distortion toward over-rejection of the classic benchmark test of 1-step ahead non-causation; if causality occurs at horizon $h > 1$, there do exist size distortions, however hypothesis rejections favor the correct detection causality, albeit at an incorrect horizon $k < h$. An important factor which improves small sample size is performing pre-tests of non-causation at all horizons, and proceeding to test subsequent hypotheses only if we reject each sufficient condition.

We implement our test procedure on money-output data sets that have been the subject of numerous studies. For sample periods ending in 1990 or 2003, we find significant evidence that detrended money growth causes output growth after at least a one month delay involving a causal chain through either fluctuations in an interest rate, or a complex chain through both fluctuations in an interest rate and a rate spread. For the sample ending in 2003, moreover, we find significance that fluctuations in the money supply will never statistically alter the growth of output ($\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$).

In only two cases involving Stock and Watson's (1989) chosen sample period in models with the Treasury bill or commercial paper rate do we find evidence for causation exactly one-month ahead. Moreover, the only significant evidence in favor of non-causality by way of causal neutralization occurs with Stock and Watson's (1989) sample period 1959-1985, and a model with the Treasury bill and rate spread. When non-causation through 3-months ahead is apparent, evidence overwhelmingly suggests noncausation by way of neutralization is not occurring, with complete noncausality.

# Appendix 1: Tables

**Table 2**

Hypotheses and Equivalent Tests (X,Y,Z)[a]

Initial Tests

| Test # | *Hypothesis* | *Equivalent Test* | Nec./Suff. |
|---|---|---|---|
| 0.1[b] | $Y \overset{(\infty)}{\nrightarrow} X$ | $Y \overset{1}{\nrightarrow} (X, Z)$ | Suff. |
| 0.2 | $Y \overset{(\infty)}{\nrightarrow} X$ | $(Y, Z) \overset{1}{\nrightarrow} X$ | Suff. |
| 1.0* | $Y \overset{1}{\nrightarrow} X$ | $Y \overset{1}{\nrightarrow} X$ | Nec./Suff. |

Intermediary Tests

| Test # | *Hypothesis* | *Equivalent Test* | Nec./Suff. |
|---|---|---|---|
| 1.1 | $Y \overset{1}{\nrightarrow} Z$ | - | - |
| 1.2 | $Z \overset{1}{\nrightarrow} X$ | - | - |

Compound Tests

| Test # | *Hypothesis* | *Equivalent Test* | Nec./Suff. |
|---|---|---|---|
| 2.0*[c] | $Y \overset{(2)}{\nrightarrow} X$ | $Y \overset{1}{\nrightarrow} (X, V)$, $\pi_{XU,1}= 0$ | Nec./Suff. |
| 2.1 | $Y \overset{1}{\nrightarrow} (X, V)$,<br>$U \overset{1}{\nrightarrow} V$ | -<br>- | -<br>- |
| 2.2 | $Y \overset{1}{\nrightarrow} (X, V)$,<br>$V \overset{1}{\nrightarrow} (U, X)$ | -<br>- | -<br>- |
| 3.1 | $Y \overset{(3)}{\nrightarrow} X$<br>(*complete*)[d] | $Y \overset{1}{\nrightarrow} (X, V)$, $U \overset{1}{\nrightarrow} V$,<br>$\pi_{XU,1}= \pi_{XU,2} = 0$ | Suff. |
| 3.2 | $Y \overset{(3)}{\nrightarrow} X$<br>(*complete*) | $Y \overset{1}{\nrightarrow} (X, V)$, $V \overset{1}{\nrightarrow} (X, U)$,<br>$\pi_{XU,1}= \pi_{XU,2} = 0$ | Suff. |
| 3.3* | $Y \overset{(3)}{\nrightarrow} X$ | $Y \overset{1}{\nrightarrow} (X, V)$, $\pi_{XU,1}= 0$<br>$\pi_{XU,2} + \pi_{XV,1}\ \pi_{VU,1}= 0$ | Nec./Suff. |
| 3.4 | - | $Y \overset{1}{\nrightarrow} (X, V)$,<br>$\pi_{XU,1}= \pi_{XU,2} = 0$ | - |
| 3.5 | (*no neutral.*) | $Y \overset{1}{\nrightarrow} (X, V)$, $\pi_{XU,1}= 0$,<br>$\pi_{XU,2} = \pi_{VU,1} = 0$ | - |
| 3.6 | (*no neutral.*) | $Y \overset{1}{\nrightarrow} (X, V)$, $\pi_{XU,1}= 0$,<br>$\pi_{XU,2} = \pi_{XV,1} = 0$ | - |

Notes: a. The present table presents test hypotheses in the case of univariate $U$.

b. See Table 1 for exact test sequence orders.

c. $V$ denotes those elements of $Z$ not caused by $Y$: $Y \overset{1}{\nrightarrow} V$; $U$ denotes those elements of $Z$ caused by $Y$: $Y \overset{1}{\rightarrow} U$, $m_u = 1$.

d. Complete non-causation $Y \overset{(3)}{\nrightarrow} X$ occurs when multiple causal routes are ruled out.

**Table 3**
Hypotheses and Equivalent Tests $(\Delta m, \Delta y, \Delta p, \Delta r)^a$

Initial Tests

| Test # | *Hypothesis* | *Equivalent Test* | Nec./Suff. |
|---|---|---|---|
| 0.1 | $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ | $\Delta m \overset{(\infty)}{\nrightarrow} (\Delta y, \Delta p, \Delta r)$ | Suff. |
| 0.2 | $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ | $(\Delta m, \Delta p, \Delta r) \overset{(\infty)}{\nrightarrow} \Delta y$ | Suff. |
| 1.0* | $\Delta m \overset{1}{\nrightarrow} \Delta y$ | $\Delta m \overset{1}{\nrightarrow} \Delta y$ | Nec./Suff. |

Intermediary Tests

| Test # | *Hypothesis* | *Equivalent Test* | Nec./Suff. |
|---|---|---|---|
| 1.1 | $\Delta m \overset{1}{\nrightarrow} \Delta p, \Delta r$ | - | - |
| 1.2 | $\Delta p, \Delta r \overset{1}{\nrightarrow} \Delta y$ | - | - |

Compound Tests

| Test # | *Hypothesis* | *Equivalent Test* | Nec./Suff. |
|---|---|---|---|
| 2.0*[b] | $\Delta m \overset{(2)}{\nrightarrow} \Delta y$ | $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p)$, $\pi_{\Delta y \Delta r,1} = 0$ | Nec./Suff. |
| 2.1 | $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p)$,<br>$\Delta r \overset{1}{\nrightarrow} \Delta p$ | -<br>- | -<br>- |
| 2.2 | $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p)$,<br>$\Delta p \overset{1}{\nrightarrow} (\Delta r, \Delta y)$ | -<br>- | -<br>- |
| 3.1 | $\Delta m \overset{(3)}{\nrightarrow} \Delta y$<br>(*complete*) | $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p)$, $\Delta r \overset{1}{\nrightarrow} \Delta p$,<br>$\pi_{\Delta y \Delta r,1} = \pi_{\Delta y \Delta r,2} = 0$ | Suff. |
| 3.2 | $\Delta m \overset{(3)}{\nrightarrow} \Delta y$<br>(*complete*) | $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p)$, $\Delta p \overset{1}{\nrightarrow} (\Delta y, \Delta r)$<br>$\pi_{\Delta y \Delta r,1} = \pi_{\Delta y \Delta r,2} = 0$ | Suff. |
| 3.3* | $\Delta m \overset{(3)}{\nrightarrow} \Delta y$ | $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p)$, $\pi_{\Delta y \Delta r,1} = 0$<br>$\pi_{\Delta y \Delta r,2} + \pi_{\Delta y \Delta p,1} \pi_{\Delta p \Delta r,1} = 0$ | Nec./Suff. |
| 3.4 | - | $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p)$,<br>$\pi_{\Delta y \Delta r,1} = \pi_{\Delta y \Delta r,2} = 0$ | - |
| 3.5 | (*no neutral.*) | $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p)$, $\pi_{\Delta y \Delta r,1} = 0$,<br>$\pi_{\Delta y \Delta r,2} = \pi_{\Delta p \Delta r,1} = 0$ | - |
| 3.6 | (*no neutral.*) | $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p)$, $\pi_{\Delta y \Delta r,1} = 0$<br>$\pi_{\Delta y \Delta r,2} + \pi_{\Delta y \Delta p,1} = 0$ | - |

Notes: a. The present table enumerates tests for models with $\Delta m, \Delta y, \Delta p, \Delta r$, where Y = $\Delta m$, X = $\Delta y$, and Z = $(\Delta p, \Delta r)$: we omit the rate spread $rr$ for the sake of brevity.

b. In every model and time period considered in this paper, $U = \Delta r$, and $V = \Delta p$ (and $rr$ in Models 3 and 4).

**Table 4.1**
Model 1: $\Delta m, \Delta y, \Delta p, \Delta r_b$

Initial Tests

| Test # | *Hypothesis* | *Equivalent Test* | 1959-1985[a] | 1959-1990[b] | 1959-2003[c] |
|---|---|---|---|---|---|
| 0.1 | $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ | $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p, \Delta r_b)$ | .0000[d] | .0000 | .0155 |
| 0.2 | $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ | $(\Delta m, \Delta p, \Delta r_b) \overset{1}{\nrightarrow} \Delta y$ | .0455 | .1205 | .0049 |
| 1.0* | $\Delta m \overset{1}{\nrightarrow} \Delta y$ | $\Delta m \overset{1}{\nrightarrow} \Delta y$ | .0181 | .3580 | .6794 |

Intermediary Tests

| Test # | *Hypothesis* | *Equivalent Test* | 1959-1985 | 1959-1990 | 1959-2003 |
|---|---|---|---|---|---|
| 1.1 | $\Delta m \overset{1}{\nrightarrow} (\Delta p, \Delta r_b)$ | - | .0000 | .0000 | .0023 |
| 1.1.a | $\Delta m \overset{1}{\nrightarrow} \Delta p$ | - | .8617 | .2789 | .4376 |
| 1.1.b | $\Delta m \overset{1}{\nrightarrow} \Delta r_b$ | - | .0000 | .0000 | .0002 |
| 1.2 | $(\Delta p, \Delta r_b) \overset{1}{\nrightarrow} \Delta y$ | - | .3066 | .1371 | .0021 |
| 1.2.a | $\Delta p \overset{1}{\nrightarrow} \Delta y$ | - | .5973 | .4763 | .7089 |
| 1.2.b | $\Delta r_b \overset{1}{\nrightarrow} \Delta y$ | - | .1596 | .1009 | .0004 |

Compound Tests

| Test # | *Hypothesis* | *Equivalent Test* | 1959-1985 | 1959-1990 | 1959-2003 |
|---|---|---|---|---|---|
| 2.0* | $\Delta m \overset{(2)}{\nrightarrow} \Delta y$ | $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p)$, $\pi_{\Delta y \Delta r,1} = 0$ | .0024[e] | .0723[f] | .0899[g] |

Notes: a. Jan. 1959 - Dec. 1985. The test order is Test 01, .02, 1.0*, 1.1, 1.2, 2.0*.
b. Jan. 1959 - Dec. 1990. The test order is Test .01, .02; or .01, .02, 1.0*, 1.1, 1.2, 2.0*.
c. Jan. 1959 - Aug. 2003. The test order is Test .01, .02, 1.0*, 1.1, 1.2, 2.0*.
d. p-values $< .00005$ are denoted .0000.
e. Conclusion: reject each $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ at 5%-level, and reject $\Delta m \overset{1}{\nrightarrow} \Delta y$ at 2%-level.
f. Conclusion: accept $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ at 10%-level; or reject $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ at 15%, accept $\Delta m \overset{1}{\nrightarrow} \Delta y$, and reject $\Delta m \overset{(2)}{\nrightarrow} \Delta y$ at a bounded 9%-level.
g. Conclusion: reject each $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ at 5%-level, accept $\Delta m \overset{1}{\nrightarrow} \Delta y$, and reject $\Delta m \overset{(2)}{\nrightarrow} \Delta y$ at bounded 10%-level.

**Table 4.2**
Model 2: $\Delta m, \Delta y, \Delta p, \Delta r_p$

Initial Tests

| Test # | *Hypothesis* | *Equivalent Test* | 1959-1985[a] | 1959-1990[b] | 1959-2003[c] |
|---|---|---|---|---|---|
| 0.1 | $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ | $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p, \Delta r)$ | .0000 | .0000 | .0572 |
| 0.2 | $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ | $(\Delta m, \Delta p, \Delta r) \overset{1}{\nrightarrow} \Delta y$ | .0083 | .0424 | .0008 |
| 1.0* | $\Delta m \overset{1}{\nrightarrow} \Delta y$ | $\Delta m \overset{1}{\nrightarrow} \Delta y$ | .0243 | .3449 | .7389 |

Intermediary Tests

| Test # | *Hypothesis* | *Equivalent Test* | 1959-1985 | 1959-1990 | 1959-2003 |
|---|---|---|---|---|---|
| 1.1 | $\Delta m \overset{1}{\nrightarrow} (\Delta p, \Delta r_p)$ | - | .0000 | .0000 | .0109 |
| 1.1.a | $\Delta m \overset{1}{\nrightarrow} \Delta p$ | - | .7726 | .2863 | .5400 |
| 1.1.b | $\Delta m \overset{1}{\nrightarrow} \Delta r_p$ | - | .0000 | .0000 | .0018 |
| 1.2 | $(\Delta p, \Delta r_p) \overset{1}{\nrightarrow} \Delta y$ | - | .2322 | .1628 | .0059 |
| 1.2.a | $\Delta p \overset{1}{\nrightarrow} \Delta y$ | - | .4907 | .4072 | .3713 |
| 1.2.b | $\Delta r_p \overset{1}{\nrightarrow} \Delta y$ | - | .1468 | .1423 | .0357 |

Compound Tests

| Test # | *Hypothesis* | *Equivalent Test* | 1959-1985 | 1959-1990 | 1959-2003 |
|---|---|---|---|---|---|
| 2.0* | $\Delta m \overset{(2)}{\nrightarrow} \Delta y$ | $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p)$, $\pi_{\Delta y \Delta r,1} = 0$ | .0173[d] | .1544 | .6540 |
| 2.1 | $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p)$, $\Delta r_p \overset{1}{\nrightarrow} \Delta p$ | - | - | .2737 | .3052 |
| 3.1 | $\Delta m \overset{(3)}{\nrightarrow} \Delta y$ (*compete*) | $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p)$, $\Delta r_p \overset{1}{\nrightarrow} \Delta p$, $\pi_{\Delta y \Delta r,1} = \pi_{\Delta y \Delta r,2} = 0$ | - | .1195[e] | .2435[f] |

Notes: a. The test order is Test .01, .02, 1.0*, 1.1, 1.2, 2.0*.
b. The test order is Test .01, .02, 1.0*, 1.1, 1.2, 2.0*, 2.1, 3.1.
c. The test order is Test .01, .02, 1.0*, 1.1, 1.2, 2.0*, 2.1, 3.1.
d. Conclusion: reject each $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ at 5%-level, and reject $\Delta m \overset{1}{\nrightarrow} \Delta y$ at 3%-level.
e. Conclusion: reject each $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ at 5%-level, and accept complete $\Delta m \overset{(3)}{\nrightarrow} \Delta y$.
f. Conclusion: reject each $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ at 6%-level, and accept complete $\Delta m \overset{(3)}{\nrightarrow} \Delta y$.

**Table 4.3**

Model 3: $\Delta m, \Delta y, \Delta p, \Delta r_b, rr_{bp}$

Initial Tests

| Test # | *Hypothesis* | *Equivalent Test* | -1985[a] | -1990[b] | -2003[c] |
|---|---|---|---|---|---|
| 0.1 | $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ | $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p, \Delta r_b, rr_{bp})$ | .0000 | .0011 | .4575 |
| 0.2 | $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ | $(\Delta m, \Delta p, \Delta r_b, rr_{bp}) \overset{1}{\nrightarrow} \Delta y$ | .0000 | .0000 | .0000 |
| 1.0* | $\Delta m \overset{1}{\nrightarrow} \Delta y$ | $\Delta m \overset{1}{\nrightarrow} \Delta y$ | .1890 | .9865 | .8676 |
| | | Intermediary Tests | | | |
| Test # | *Hypothesis* | *Equivalent Test* | -1985 | -1990 | -2003 |
| 1.1 | $\Delta m \overset{1}{\nrightarrow} (\Delta p, \Delta r_b, rr_{bp})$ | - | .0000 | .0000 | .2262 |
| 1.1.a | $\Delta m \overset{1}{\nrightarrow} \Delta p$ | - | .8390 | .5707 | .6856 |
| 1.1.b | $\Delta m \overset{1}{\nrightarrow} \Delta r_b$ | - | .0000 | .0000 | .0063 |
| 1.1.c | $\Delta m \overset{1}{\nrightarrow} rr_{bp}$ | | .1594 | .0885 | .8897 |
| 1.2 | $(\Delta p, \Delta r_b, rr_{bp}) \overset{1}{\nrightarrow} \Delta y$ | - | .0000 | .0000 | .0000 |
| 1.2.a | $\Delta p \overset{1}{\nrightarrow} \Delta y$ | - | .7929 | .9150 | .9084 |
| 1.2.b | $\Delta r_b \overset{1}{\nrightarrow} \Delta y$ | - | .2646 | .0196 | .0010 |
| 1.2.c | $rr_{bp} \overset{1}{\nrightarrow} \Delta y$ | | .0001 | .0002 | .0000 |
| | | Compound Tests | | | |
| Test # | *Hypothesis* | *Equivalent Test* | -1985 | -1990 | -2003 |
| 2.0* | $\Delta m \overset{(2)}{\nrightarrow} \Delta y$ | $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p, rr_{bp})$, $\pi_{\Delta y \Delta r,1} = 0$ | .3359 | .2814 | .0544[f] |
| 2.1 | $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p, rr_{bp})$, $\Delta r_b \overset{1}{\nrightarrow} (\Delta p, rr_{bp})$ | - | .0071 | .0002 | .0003 |
| 2.2 | $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p, rr_{bp})$, $(\Delta p, rr_{bp}) \overset{1}{\nrightarrow} (\Delta r_b, \Delta y)$ | - | .0000 | .0000 | .0000 |
| 3.3* | $\Delta m \overset{(3)}{\nrightarrow} \Delta y$ | $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p, rr_{bp})$, $\pi_{\Delta y \Delta r,1} = 0$, $\pi_{\Delta y \Delta r,2} + \pi_{\Delta y(\Delta p, rr),1} \pi_{(\Delta p, rr)\Delta r,1} = 0$ | .2819 | .0008[e] | - |
| 3.4 | - | $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p, rr_{bp})$, $\pi_{\Delta y \Delta r,1} = \pi_{\Delta y \Delta r,2} = 0$ | .3713 | - | - |
| 3.5 | (*no neutral.*) | $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p, rr_{bp})$, $\pi_{\Delta y \Delta r,1} = 0$, $\pi_{\Delta y \Delta r,2} = \pi_{(\Delta p, rr)\Delta r,1} = 0$ | .0929 | - | - |
| 3.6 | (*no neutral.*) | $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p, rr_{bp})$, $\pi_{\Delta y \Delta r,1} = 0$, $\pi_{\Delta y \Delta r,2} = \pi_{\Delta y(\Delta p, rr),1} = 0$ | .0004[d] | - | - |

Notes: a. The test order is Test .01, .02, 1.0*, 1.1, 1.2, 2.0*, 2.1, 2.2, 3.3*, 3.4-3.6.

b. The test order is Test .01, .02, 1.0*, 1.1, 1.2, 2.0*, 2.1, 2.2, 3.3*.

c. The test order is Test .01; or Test .02, 1.0*, 1.1, 1.2, 2.0*.

d. Reject each $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ at 5%, and accept $\Delta m \overset{(3)}{\nrightarrow} \Delta y$, possible neutralization through $r$ and $rr$.

e. Reject each $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ at 5%, accept $\Delta m \overset{(2)}{\nrightarrow} \Delta y$, and reject $\Delta m \overset{(3)}{\nrightarrow} \Delta y$ at bounded 5%-level.

f. Accept $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$; or accept $\Delta m \overset{1}{\nrightarrow} \Delta y$, and reject $\Delta m \overset{(2)}{\nrightarrow} \Delta y$ at bounded 7%-level.

**Table 4.4**

Model 4: $\Delta m, \Delta y, \Delta p, \Delta r_p, rr_{bp}$

| Initial Tests | | | | | |
|---|---|---|---|---|---|
| Test # | *Hypothesis* | *Equivalent Test* | -1985[a] | -1990[b] | -2003[c] |
| 0.1 | $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ | $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p, \Delta r_p, rr_{bp})$ | .0000 | .0042 | .5920 |
| 0.2 | $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ | $(\Delta m, \Delta p, \Delta r_p, rr_{bp}) \overset{1}{\nrightarrow} \Delta y$ | .0000 | .0000 | .0000 |
| 1.0* | $\Delta m \overset{1}{\nrightarrow} \Delta y$ | $\Delta m \overset{1}{\nrightarrow} \Delta y$ | .2146 | .9870 | .8739 |
| Intermediary Tests | | | | | |
| Test # | *Hypothesis* | *Equivalent Test* | -1985 | -1990 | -2003 |
| 1.1 | $\Delta m \overset{1}{\nrightarrow} (\Delta p, \Delta r_p, rr_{bp})$ | - | .0000 | .0002 | .3447 |
| 1.1.a | $\Delta m \overset{1}{\nrightarrow} \Delta p$ | - | .8768 | .5779 | .7887 |
| 1.1.b | $\Delta m \overset{1}{\nrightarrow} \Delta r_p$ | - | .0000 | .0001 | .0352 |
| 1.1.c | $\Delta m \overset{1}{\nrightarrow} rr_{bp}$ | | .4191 | .1416 | .9158 |
| 1.2 | $(\Delta p, \Delta r_p, rr_{bp}) \overset{1}{\nrightarrow} \Delta y$ | - | .0000 | .0000 | .0000 |
| 1.2.a | $\Delta p \overset{1}{\nrightarrow} \Delta y$ | - | .7710 | .9394 | .9252 |
| 1.2.b | $\Delta r_p \overset{1}{\nrightarrow} \Delta y$ | - | .2030 | .0027 | .0000 |
| 1.2.c | $rr_{bp} \overset{1}{\nrightarrow} \Delta y$ | | .0000 | .0000 | .0000 |
| Compound Tests | | | | | |
| Test # | *Hypothesis* | *Equivalent Test* | -1985 | -1990 | -2003 |
| 2.0* | $\Delta m \overset{(2)}{\nrightarrow} \Delta y$ | $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p, rr_{bp})$, $\pi_{\Delta y \Delta r,1} = 0$ | .5621 | .2187 | .0246[f] |
| 2.1 | $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p, rr_{bp})$, $\Delta r_b \overset{1}{\nrightarrow} (\Delta p, rr_{bp})$ | - | .0893 | .0000 | .0000 |
| 2.2 | $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p, rr_{bp})$, $(\Delta p, rr_{bp}) \overset{1}{\nrightarrow} (\Delta r_p, \Delta y)$ | - | .0000 | .0000 | .0000 |
| 3.3* | $\Delta m \overset{(3)}{\nrightarrow} \Delta y$ | $\Delta m \overset{1}{\nrightarrow} (\Delta y, \Delta p, rr_{bp})$, $\pi_{\Delta y \Delta r,1} = 0$, $\pi_{\Delta y \Delta r,2} + \pi_{\Delta y(\Delta p,rr),1}\pi_{(\Delta p,rr)\Delta r,1} = 0$ | .0014[d] | .0000[e] | - |

Notes: a. The test order is Test .01, .02, 1.0*, 1.1, 1.2, 2.0*, 2.1, 2.2, 3.3*.

b. The test order is Test .01, .02, 1.0*, 1.1, 1.2, 2.0*, 2.1, 2.2, 3.3*.

c. The test order is Test .01, .02; or Test 1.0*, 1.1, 1.2, 2.0*.

d. Reject each $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ at 5%, accept $\Delta m \overset{(2)}{\nrightarrow} \Delta y$, and reject $\Delta m \overset{(3)}{\nrightarrow} \Delta y$ at bounded 5%-level.

e. Reject each $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$ at 5%, accept $\Delta m \overset{(2)}{\nrightarrow} \Delta y$, and reject $\Delta m \overset{(3)}{\nrightarrow} \Delta y$ at bounded 5%-level.

f. Accept $\Delta m \overset{(\infty)}{\nrightarrow} \Delta y$; or accept $\Delta m \overset{1}{\nrightarrow} \Delta y$, and reject $\Delta m \overset{(2)}{\nrightarrow} \Delta y$ at bounded 4%-level.

# Appendix 2: Simulation Study

**Set Up** We generated VAR(6) 5-vector processes under the null of non-causation at all horizons, and under alternatives of causation at horizons $h = 1...6$ (non-causation up to horizons $h = 0...5$ where $h = 0$ denotes non-causation at all horizons). Sample sizes are restricted to $n \in \{200, 400, 600, 800, 1000\}$ and 1000 repetitions are performed for each test.

For the simulated process $W_t = \sum_{i=1}^{6} \pi_i W_{t-i} + \epsilon_t$, $\epsilon_t$ denotes an *iid* 5-vector with mutually independent components drawn from a standard normal distribution. The sub-vectorization is $W_t = (X_t, Y_t, Z_t')'$, with $Z_t$ a 3-vector. The matrix coefficients $\pi_i$ are generated as *iid* random variables from the uniform cube $[-.5, .5]^5$: we use $\pi$ only if the resulting characteristic polynomial $I_m - \pi_1 z - ... - \pi_6 z^6$ has all roots outside the unit circle.

During the simulation process, we impose the following restrictions, depending upon the hypothesis to be tested. Benchmark hypotheses are

$$\begin{aligned}
&H_0^{\infty} : \pi_{XY,i} = \pi_{XZ,i} = 0,\ i = 1...6;\\
&H_1^{1} :\ \pi_{XY,i} \neq 0,\ i = 1...6;\\
&H_1^{2} : \pi_{XY,i} = 0,\ i = 1...6.
\end{aligned}$$

Under hypothesis $H_0^{\infty}$, we deduce $Y \overset{1}{\nrightarrow} (X, Z)$, therefore $Y \overset{(\infty)}{\nrightarrow} X$, cf. Theorems 1-2. Under $H_1^1$, causation $Y \overset{1}{\rightarrow} X$, and under $H_1^2$, non-causation $Y \overset{1}{\nrightarrow} X$, and causation $Y \overset{2}{\rightarrow} X$ are true.

Let $Z = (U', V')'$, $U = Z_1$, $V = (Z_2, Z_3)$ (i.e. $m_u = 1$ and $m_v = 2$) and define

$$\begin{aligned}
&H_1^3 : \pi_{XY,i} = \pi_{VY,i} = 0,\ i = 1...6,\ \pi_{XU,1} = 0;\\
&H_1^{4,c} : \pi_{XY,i} = \pi_{VY,i} = \pi_{VU,i} = 0,\ i = 1...6,\ \pi_{XU,1} = \pi_{XU,2} = 0;\\
&H_1^{4,cn} : \pi_{XY,i} = \pi_{VY,i} = 0,\ i = 1...6,\ \pi_{XU,1} = 0,\ \pi_{XU,2} = -\pi_{XV,1}\pi_{VU,1};\\
&H_1^5 : \pi_{XY,i} = \pi_{VY,i} = \pi_{VU,i} = 0,\ i = 1...6,\\
&\qquad \pi_{XU,1} = \pi_{XU,2} = \pi_{XU,3} = 0;\\
&H_1^6 : \pi_{XY,i} = \pi_{VY,i} = \pi_{VU,i} = 0,\ i = 1...6,\\
&\qquad \pi_{XU,1} = \pi_{XU,2} = \pi_{XU,3} = \pi_{XU,4} = 0.
\end{aligned}$$

For $H_1^3$, we have $Y \overset{(2)}{\nrightarrow} X$ and $Y \overset{3}{\rightarrow} X$, cf. Theorem 6. Under $H_1^{4,c}$, $Y \overset{(3)}{\nrightarrow} X$ and $Y \overset{4}{\rightarrow} X$, cf. Theorem 6, where non-causation through 3-steps ahead occurs *completely*; and under $H_1^{4,cn}$ $Y \overset{(3)}{\nrightarrow} X$ and $Y \overset{4}{\rightarrow} X$, where non-causation through 3-steps ahead occurs due to *neutralization*. Finally, under hypotheses $H_1^h$, $h = 5$ or 6, non-causation through $h - 1$ occurs completely, with causation at $h$ occurs (e.g. $H_1^5$: $Y \overset{(4)}{\nrightarrow} X, Y \overset{5}{\rightarrow} X$): see Remark 4 of Theorem 4.

For each simulated series $W_t$, a minimum AIC method is employed for VAR order $p$ selection, where considered orders are restricted to the set $\{1, ..., 16\}$.

The initial tests of non-causation at all horizons is performed at the 5%-level; all other tests are performed at the 1%-level.

**Results** We perform all tests of $Y \overset{(h)}{\nrightarrow} X$ according the sequential order detailed in Section 4, and summarized in Table 1. Table 5, below, contains all simulation results. Processes range from non-causality at all horizons ($H_0^\infty$) to causality at horizon $h = 6$ ($H_1^6$).

Columns contain empirical rejection frequencies based on $p$-values derived from the $F$ distribution, with degrees of freedom adjustments discussed in Lütkepohl (1991)[27]. Tests at horizon $h = 0$ represent tests of noncausation at all horizons (Tests 0.1 and 0.2).

For sequential tests of each $Y \overset{(h)}{\nrightarrow} X$, $h = 1...3$, we perform Tests 0.1 ($Y \overset{(\infty)}{\nrightarrow} X$) and 0.2 ($Y \overset{(\infty)}{\nrightarrow} X$) and proceed only if both are rejected at the 5%-level. We then perform Test 1.0* ($Y \overset{1}{\nrightarrow} X$) and proceed only if we fail to reject at the 1%-level. Sequential tests of of the classic hypothesis $Y \overset{1}{\nrightarrow} X$ are contained the columns labeled "$h = 1$" We then perform intermediary Tests 1.1 and 1.2 (not reported here), and we report the average of detected $m_u$ over all simulations. If we reject $Y \overset{(k)}{\nrightarrow} X$, this is scored as a rejection of all subsequent $Y \overset{(h)}{\nrightarrow} X$, $h > k$. If $m_u > 1$ is detected, we still perform all tests and use a standard rejection region rule as a criterion for test rejection[28].

As a separate experiment, we perform tests of the classic hypothesis $Y \overset{1}{\nrightarrow} X$ irrespective of ("isolated" from) the outcome of tests of $Y \overset{(\infty)}{\nrightarrow} X$: results for these 1-step ahead non-causation tests are presented in columns labeled "$h = 1_{iso}$".

When $Y \overset{(\infty)}{\nrightarrow} X$ is true, tests at $h = 0$ perform well: we always reject $(Y, Z) \overset{1}{\nrightarrow} X$ (which is false)[29], and reject $Y \overset{1}{\nrightarrow} (X, Z)$ (which is true) at roughly a 5%-frequency for $n > 400$. Under $H_0^\infty$, there exists a trend of over-rejecting isolated tests of $Y \overset{1}{\nrightarrow} X$, however, while sequential tests of $Y \overset{1}{\nrightarrow} X$ generate smaller rejection frequencies at or below the nominal level. The average detected dimension of $U$ is reasonably under 1, and close to 0 for $n > 400$ (the true dimension is 0).

The tests perform essentially perfectly when $Y \overset{1}{\rightarrow} X$ is true, as expected, with the average of detected $m_u$ at (or near) the true 3. However, when causation occurs at horizons $h > 1$, size distortions are apparent. Under $H_1^2$, for

[27] Because simulation evidence, cf. Hill (2004b), suggests $p$-values derived from a parametric bootstrap both tame empirical size distortions and hinder empirical power, and due to space considerations, we use only standard $p$-values in the present study.

[28] Because Tests 2.0*-3.3* can be used only to deduce non-causation in the $m_u > 1$ case, the fact that we tabulate a rejection of their respective hypotheses $Y \overset{(h)}{\nrightarrow} X$ when we "reject" the test implies an enlarged Type I error probability. This should be kept in mind as we discuss the simulation outcome.

[29] Test results for $(Y, Z) \overset{1}{\nrightarrow} X$ are not shown: for all hypotheses $(Y, Z) \overset{1}{\nrightarrow} X$ is false, and for all sample sizes empirical power is 1.00.

example, $Y \overset{1}{\nrightarrow} X$ and $Y \overset{2}{\rightarrow} X$ are true and the rejection rate of $Y \overset{1}{\nrightarrow} X$ (which is true) is not far from the nominal 1%-level for large $n$, but occurs at a rate 6-times the nominal level when $n = 400$. Moreover, the power of tests of $Y \overset{(2)}{\nrightarrow} X$ is rather sluggish (always below 80%), but compensated for in tests of $Y \overset{(3)}{\nrightarrow} X$.

This pattern of over-rejecting at horizons sooner than when true causation occurs continues for every other simulated case. For example, when $Y \overset{(2)}{\nrightarrow} X$ and $Y \overset{3}{\rightarrow} X$ are true, we reject $Y \overset{1}{\nrightarrow} X$ at a rate 5-times the nominal level when $n = 400$ (and when $n = 1000$ the rejection rate 1.1% is the roughly the nominal level), and reject $Y \overset{(2)}{\nrightarrow} X$ at roughly 31-times (!) the nominal level (dropping to a rate about 9-times the nominal level, or 8.7% for $n = 1000$).

The size distortions for tests at $h = 2$ or 3 however, drop precipitously as the true horizon of causation increases. For example, when $Y \overset{(5)}{\nrightarrow} X$ and $Y \overset{6}{\rightarrow} X$ are true, we incorrectly reject $Y \overset{1}{\nrightarrow} X$, $Y \overset{(2)}{\nrightarrow} X$ or $Y \overset{(3)}{\nrightarrow} X$ at rates 1.4%, 7.7% and 14.6%, respectively, when $n = 400$; and 1.1%, 2.6% and 6.5%, respectively, when $n = 1000$. For either sample size, the rejection frequency of the classic test of $Y \overset{1}{\nrightarrow} X$ is reasonably near the nominal level 1%. This suggests that a rejection of the hypothesis $Y \overset{1}{\nrightarrow} X$ in practice should be held to indicate causation truly occurs 1-step ahead, or in the very near future (e.g. 1-to-2-steps ahead). The greater the time distance between the actual horizon of causation and the test horizon, the smaller the likelihood of a false rejection of the classic test, and indeed the likelihood is quite close to the chosen level of significance, in particular for the classic test of 1-step ahead non-causation.

Finally, when causal neutralization is true ($H_1^{4,cn}$), the empirical rejection frequency is substantially larger than if non-causation is complete: even when $n = 1000$, we reject $Y \overset{(3)}{\nrightarrow} X$ at a rate 25-times the nominal level of 1%. Even taking into consideration sequential size bounding, this rejection rate is large.

Of course, causal neutralization is occurring at $h = 3$ with causation at $h = 4$, thus the false detection of $Y \overset{3}{\rightarrow} X$ is nonetheless a correct detection of causation "*at some horizon*". Moreover, if the time horizon of causation increases to $Y \overset{(5)}{\nrightarrow} X$ and $Y \overset{(6)}{\rightarrow} X$, then tests of $Y \overset{(h)}{\nrightarrow} X$ work well once size augmentation due to sequential testing is accounted for. Indeed, because we allow for tests of $Y \overset{(h)}{\nrightarrow} X$ even when $m_u > 1$ is detected, using techniques similar to Lemma 10 the upper bound of test sizes for tests of $Y \overset{(2)}{\nrightarrow} X$ and $Y \overset{(3)}{\nrightarrow} X$ can be shown to be, respectively, 4% and 7% when the nominal levels of all Tests 1.0*-3.3* are all set to 1%. When $n = 1000$, the actual rejection rates are, respectively, 3.3% and 6.5%.

**Table 5**

Empirical Size and Power: 5-vector VAR(6)

$H_0^{\infty}: Y \overset{(\infty)}{\nrightarrow} X$

| n | h=0[a] | h=1$_{iso}$[b] | h=1[c] | h=2[d] | h=3[e] | $\overline{m_u}$[f] |
|---|---|---|---|---|---|---|
| 200 | .270[g] | .300 | .014 | .072 | .214 | .431 |
| 400 | .084 | .070 | .022 | .069 | .098 | .246 |
| 600 | .054 | .048 | .013 | .042 | .051 | .162 |
| 800 | .051 | .049 | .011 | .035 | .047 | .145 |
| 1000 | .046 | .048 | .006 | .028 | .031 | .129 |

$H_1^1: Y \overset{1}{\rightarrow} X$

| n | h=0 | h=1$_{iso}$ | h=1 | h=2 | h=3 | $\overline{m_u}$ |
|---|---|---|---|---|---|---|
| 200 | 1.00 | .878 | .878 | .888 | .888 | 2.90 |
| 400 | 1.00 | 1.00 | 1.00 | 1.00 | 1.00 | 3.00 |
| 600 | 1.00 | 1.00 | 1.00 | 1.00 | 1.00 | 3.00 |
| 800 | 1.00 | 1.00 | 1.00 | 1.00 | 1.00 | 3.00 |
| 1000 | 1.00 | 1.00 | 1.00 | 1.00 | 1.00 | 3.00 |

$H_1^2: Y \overset{1}{\nrightarrow} X, Y \overset{2}{\rightarrow} X$

| n | h=0 | h=1$_{iso}$ | h=1 | h=2 | h=3 | $\overline{m_u}$ |
|---|---|---|---|---|---|---|
| 200 | .911 | .133 | .133 | .422 | .733 | 1.55 |
| 400 | .990 | .061 | .061 | .394 | .909 | 1.22 |
| 600 | 1.00 | .039 | .039 | .586 | .950 | 1.17 |
| 800 | 1.00 | .031 | .031 | .671 | .970 | 1.08 |
| 1000 | 1.00 | .015 | .015 | .743 | .988 | 1.04 |

$H_1^3: Y \overset{(2)}{\nrightarrow} X, Y \overset{3}{\rightarrow} X$

| n | h=0 | h=1$_{iso}$ | h=1 | h=2 | h=3 | $\overline{m_u}$ |
|---|---|---|---|---|---|---|
| 200 | .949 | .121 | .119 | .408 | .781 | 1.42 |
| 400 | 1.00 | .052 | .052 | .311 | .837 | 1.16 |
| 600 | 1.00 | .039 | .037 | .148 | .880 | 1.09 |
| 800 | 1.00 | .023 | .020 | .101 | .923 | 1.08 |
| 1000 | 1.00 | .012 | .011 | .087 | .989 | 1.08 |

$H_1^{4,c}: Y \overset{(3)}{\nrightarrow} X, Y \overset{4}{\rightarrow} X$ (*complete*)

| n | h=0 | h=1$_{iso}$ | h=1 | h=2 | h=3 | $\overline{m_u}$ |
|---|---|---|---|---|---|---|
| 200 | .939 | .091 | .091 | .321 | .502 | 1.41 |
| 400 | .976 | .034 | .034 | .053 | .182 | 1.11 |
| 600 | 1.00 | .019 | .019 | .054 | .166 | 1.09 |
| 800 | 1.00 | .015 | .015 | .048 | .138 | 1.10 |
| 1000 | 1.00 | .013 | .013 | .036 | .124 | 1.08 |

$H_1^{4,cn}: Y \overset{(3)}{\nrightarrow} X$ (*neutral*)

| n | h=0 | h=1$_{iso}$ | h=1 | h=2 | h=3 | $\overline{m_u}$ |
|---|---|---|---|---|---|---|
| 200 | .912 | .061 | .061 | .250 | .616 | 1.23 |
| 400 | .975 | .023 | .023 | .419 | .674 | 1.19 |
| 600 | .987 | .021 | .021 | .121 | .493 | 1.13 |
| 800 | 1.00 | .011 | .011 | .093 | .387 | 1.11 |
| 1000 | 1.00 | .012 | .012 | .064 | .255 | 1.11 |

$H_1^5: Y \overset{(4)}{\nrightarrow} X, Y \overset{5}{\rightarrow} X$ (*complete*)

| n | h=0 | h=1$_{iso}$ | h=1 | h=2 | h=3 | $\overline{m_u}$ |
|---|---|---|---|---|---|---|
| 200 | .922 | .033 | .033 | .130 | .326 | 1.11 |
| 400 | .952 | .013 | .013 | .082 | .174 | 1.08 |
| 600 | 1.00 | .011 | .011 | .042 | .140 | 1.06 |
| 800 | 1.00 | .009 | .009 | .028 | .116 | 1.06 |
| 1000 | 1.00 | .011 | .011 | .031 | .099 | 1.04 |

$H_1^6: Y \overset{(5)}{\nrightarrow} X, Y \overset{6}{\rightarrow} X$ (*complete*)

| n | h=0 | h=1$_{iso}$ | h=1 | h=2 | h=3 | $\overline{m_u}$ |
|---|---|---|---|---|---|---|
| 200 | .941 | .031 | .031 | .111 | .282 | 1.15 |
| 400 | .968 | .014 | .014 | .077 | .146 | 1.09 |
| 600 | 1.00 | .009 | .009 | .031 | .120 | 1.08 |
| 800 | 1.00 | .010 | .010 | .032 | .091 | 1.04 |
| 1000 | 1.00 | .011 | .011 | .026 | .065 | 1.05 |

Notes: a. Test 0.1 ($Y \overset{(\infty)}{\nrightarrow} X$) tests the hypothesis $Y \overset{1}{\nrightarrow} (X, Z)$.

b. Isolated Test 1.0* ($Y \overset{1}{\nrightarrow} X$): performed irrespective of outcome of Tests 0.1 and 0.2 ($Y \overset{(\infty)}{\nrightarrow} X$).

c. Sequential Test 1.0* ($Y \overset{1}{\nrightarrow} X$): performed only if Tests 0.1 and 0.2 ($Y \overset{(\infty)}{\nrightarrow} X$) are rejected.

d. Test 2.0*, $Y \overset{(2)}{\nrightarrow} X$.

e. The test of $Y \overset{(3)}{\nrightarrow} X$ sequentially involves Tests 2.1-3.3*, depending upon the sequential outcome, cf. Table 1. We reject $Y \overset{(3)}{\nrightarrow} X$ if we fail to reject Test 2.1 and reject 3.1, or reject 2.1 and fail to reject 2.2 and reject 3.2, or reject 2.1 and reject 2.2 and reject 3.3*.

f. $\overline{m_u}$ denotes the average of all $m_u$, the determined dimension of $U$.

g. Values denote rejection frequencies.

# Appendix 3: Formal Proofs

**Proof of Lemma 3.** Consider $(i)$. Assume $Y \overset{(h)}{\nrightarrow} X|I_{XZ}$ for some $h \geq 2$. Recall the partition $Z = (U', V')'$. From the argument following (6), we know $Y \overset{(h+1)}{\nrightarrow} X|I_{XZ}$ *if and only if* $\pi^{(h)}_{XZ,1}\pi_{ZY,i} = 0$, $\forall i \geq 1$, and $Y \overset{1}{\nrightarrow} V|I_{XZ}$ implies $Y \overset{(h+1)}{\nrightarrow} X|I_{XZ}$ *if and only if* $\pi^{(h)}_{XU,1}\pi_{UY,i} = 0$, $\forall i \geq 1$. Moreover, because $U$ is univariate and $Y \overset{1}{\rightarrow} U|I_{XZ}$, we deduce $\pi^{(h)}_{XU,1}\pi_{UY,i} = 0$ occurs for every $i \geq 1$ *if and only if* $\pi^{(h)}_{XU,1} = 0$. This is true irrespective of the dimensions of $X$ and $Y$.

Next, consider $(ii)$, and recall we assume $Y \overset{(h)}{\nrightarrow} X|I_{XZ}$. From part $(i)$, $Y \overset{(h+1)}{\nrightarrow} X|I_{XZ}$ *if and only if* $\pi^{(h)}_{XU,1} = 0$, and from (3), we deduce

$$\pi^{(h)}_{XU,1} = \pi^{(h-1)}_{XU,2} + \pi^{(h-1)}_{XX,1}\pi_{XU,1} + \pi^{(h-1)}_{XY,1}\pi_{YU,1} + \pi^{(h-1)}_{XU,1}\pi_{UU,1} + \pi^{(h-1)}_{XV,1}\pi_{VU,1}. \quad (15)$$

For $h = 2$, $Y \overset{(2)}{\nrightarrow} X|I_{XZ}$ implies $\pi^{(1)}_{XU,1} = 0$ from part $(i)$, above, hence

$$\begin{aligned} \pi^{(2)}_{XU,1} &= \pi_{XU,2} + \pi_{XX,1}\pi_{XU,1} + \pi_{XY,1}\pi_{YU,1} + \pi_{XU,1}\pi_{UU,1} + \pi_{XV,1}\pi_{VU,1} \\ &= \pi_{XU,2} + \pi_{XX,1} \times 0 + 0 \times \pi_{YU,1} + 0 \times \pi_{UU,1} + +\pi_{XV,1}\pi_{VU,1} \\ &= \pi_{XU,2} + \pi_{XV,1}\pi_{VU,1}. \end{aligned} \quad (16)$$

For $h = 3$, $Y \overset{(3)}{\nrightarrow} X|I_{XZ}$ implies $\pi^{(2)}_{XU,1} = \pi^{(1)}_{XU,1} = 0$ from part $(i)$, above, hence

$$\begin{aligned} \pi^{(3)}_{XU,1} &= \pi^{(2)}_{XU,2} + \pi^{(2)}_{XX,1}\pi_{XU,1} + \pi^{(2)}_{XY,1}\pi_{YU,1} + \pi^{(2)}_{XU,1}\pi_{UU,1} + \pi^{(2)}_{XV,1}\pi_{VU,1} \\ &= \pi^{(2)}_{XU,2} + \pi^{(2)}_{XX,1} \times 0 + 0 \times \pi_{YU,1} + 0 \times \pi_{UU,1} + \pi^{(2)}_{XV,1}\pi_{VU,1} \\ &= \pi^{(2)}_{XU,2} + \pi^{(2)}_{XV,1}\pi_{VU,1} \end{aligned} \quad (17)$$

where

$$\begin{aligned} \pi^{(2)}_{XU,2} &= \pi_{XU,3} + \pi_{XX,1}\pi_{XU,2} + \pi_{XY,1}\pi_{YU,2} + \pi_{XU,1}\pi_{UU,2} + \pi_{XV,1}\pi_{VU,2} \\ &= \pi_{XU,3} + \pi_{XX,1}\pi_{XU,2} + 0 \times \pi_{YU,2} + 0 \times \pi_{UU,2} + \pi_{XV,1}\pi_{VU,2} \\ &= \pi_{XU,3} + \pi_{XX,1}\pi_{XU,2} + \pi_{XV,1}\pi_{VU,2}. \end{aligned} \quad (18)$$

Thus,

$$\begin{aligned} \pi^{(3)}_{XU,1} &= \pi^{(2)}_{XU,2} + \pi^{(2)}_{XV,1}\pi_{VU,1} \\ &= \pi_{XU,3} + \pi_{XX,1}\pi_{XU,2} + \pi_{XV,1}\pi_{VU,2} + \pi^{(2)}_{XV,1}\pi_{VU,1}. \end{aligned} \quad (19)$$

Recursively we deduce

$$\pi^{(h)}_{XU,1} = \pi_{XU,h} + \sum\nolimits_{i=1}^{h-1} \pi^{(h-i)}_{XX,1}\pi_{XU,i} + \sum\nolimits_{i=1}^{h-1} \pi^{(h-i)}_{XV,1}\pi_{VU,i}, \quad (20)$$

where we include the term $\pi_{XX,1}^{(h-1)}\pi_{XU,1} = 0$.

Finally, consider $(iii)$. From part $(i)$, $Y \overset{(h)}{\nrightarrow} X|I_{XZ}$ *if and only if* $\pi_{XU,1}^{(h)}$, and from part $(ii)$, we have the identity

$$\pi_{XU,1}^{(h)} = \pi_{XU,h} + \sum\nolimits_{i=1}^{h-1} \pi_{XX,1}^{(h-i)}\pi_{XU,i} + \sum\nolimits_{i=1}^{h-1} \pi_{XV,1}^{(h-i)}\pi_{VU,i}. \tag{21}$$

Now, let $\pi_{XU,i} = \pi_{VU,i} = 0$, $i = 1...h-1$. Then

$$\pi_{XU,1}^{(h)} = \pi_{XU,h} + \sum\nolimits_{i=1}^{h-1} \pi_{XX,1}^{(h-i)} \times 0 + \sum\nolimits_{i=1}^{h-1} \pi_{XV,1}^{(h-i)} \times 0 = \pi_{XU,h}. \tag{22}$$

This completes the proof. ■

**Proof of Theorem 4.** For $(i)$, assume $m_z = m_u = 1$ (i.e. $U = Z$, $Y \overset{1}{\rightarrow} Z|I_{XZ}$), such that $m_v = 0$ by convention.

Consider $h = 2$ and assume $Y \overset{1}{\nrightarrow} X|I_{XZ}$. By Lemma 3.$i$, we have $Y \overset{2}{\nrightarrow} X|I_{XZ}$ *if and only if* $\pi_{XZ,1}^{(1)} = \pi_{XZ,1} = 0$. This proves the result for $h = 2$..

Now, let $Y \overset{(h)}{\nrightarrow} X|I_{XZ}$ for any $h \geq 2$. From Lemma 3.$ii$, it follows that $\pi_{XZ,1}^{(k)}$ has the representation $\pi_{XZ,1}^{(2)} = \pi_{XZ,2}$, and for $2 < k \leq h$

$$\pi_{XZ,1}^{(k)} = \pi_{XZ,k} + \sum\nolimits_{i=1}^{k-1} \left(\pi_{XX,1}^{(k-i)}\pi_{XZ,i}\right). \tag{23}$$

We will prove the result, $Y \overset{(h+1)}{\nrightarrow} X|I_{XZ}$ *if and only if* $\pi_{XZ,h} = 0$, by induction. Assume $Y \overset{(h)}{\nrightarrow} X|I_{XZ}$ *if and only if* $\pi_{XZ,h-1} = 0$ for some $h \geq 2$. Then, notice that $Y \overset{(h)}{\nrightarrow} X|I_{XZ}$ implies $Y \overset{(k)}{\nrightarrow} X|I_{XZ}$ for each $k = 1...h$, thus, by the induction assumption we deduce $Y \overset{(h)}{\nrightarrow} X|I_{XZ}$ *if and only if* $\pi_{XZ,k} = 0$, $k = 1...h-1$. Consequently, by the induction assumption the equality in (23) reduces to

$$\pi_{XZ,1}^{(h)} = \pi_{XZ,h} + \sum\nolimits_{i=1}^{h-1} \left(\pi_{XX,1}^{(h-i)}\pi_{XZ,i}\right) = \pi_{XZ,h}. \tag{24}$$

By Lemma 3, $Y \overset{(h+1)}{\nrightarrow} X|I_{XZ}$ *if and only if* $\pi_{XZ,1}^{(h)} = 0$, and $\pi_{XZ,1}^{(h)} = \pi_{XZ,h}$, therefore $Y \overset{(h+1)}{\nrightarrow} X|I_{XZ}$ *if and only if* $\pi_{XZ,h} = 0$. Because $h \geq 2$ is arbitrary, this proves claim $(i)$ by induction.

Consider $(ii)$, and assume $m_z > 1$ and $1 < m_u \leq m_z$ for the remainder of the proof. From (6) and (7), we know $Y \overset{(2)}{\nrightarrow} X|I_{XZ}$ *if and only if* $\pi_{XU,1}\pi_{UY,j} = 0$, $j \geq 1$. By the assumption $Y \rightarrow U$, we have $\pi_{UY,j} \neq 0$ for some $j$, hence $\pi_{XU,1}\pi_{UY,j} = 0$ *if and only if* $\pi_{XU,1} = 0$.

For $(iii)$, let $Y \overset{(3)}{\nrightarrow} X|I_{XZ}$ and $U \overset{1}{\nrightarrow} V|I_{XZ}$. By Lemma 3, we deduce $Y \overset{(3)}{\nrightarrow} X|I_{XZ}$ *if and only if*

$$\pi_{XU,1}^{(2)} = \pi_{XU,2} + \pi_{XX,1}\pi_{XU,1} + \pi_{XV,1}\pi_{VU,1} = 0. \tag{25}$$

If $U \overset{1}{\nrightarrow} V|I_{XZ}$, then $\pi_{VU,i} = 0$, $i \geq 1$, thus$Y \overset{(3)}{\nrightarrow} X|I_{XZ}$ *if and only if*

$$\pi^{(2)}_{XU,1} = \pi_{XU,2} + \pi_{XX,1}\pi_{XU,1} = 0. \tag{26}$$

Now, we know $Y \overset{(2)}{\nrightarrow} X|I_{XZ}$ *if and only if* $\pi_{XU,1} = 0$, cf. part $(ii)$, hence $Y \overset{(3)}{\nrightarrow} X|I_{XZ}$ *if and only if*

$$\begin{aligned} 0 &= \pi^{(2)}_{XU,1} = \pi_{XU,2} + \sum\nolimits_{i=1}^{1} \pi^{(h-i)}_{XX,1}\pi_{XU,i} \\ &= \pi_{XU,2} + \sum\nolimits_{i=1}^{1} \pi^{(h-i)}_{XX,1} \times 0 = \pi_{XU,2}. \end{aligned} \tag{27}$$

Next, consider $(iv)$ and let $Y \overset{(2)}{\nrightarrow} X|I_{XZ}$ and $V \overset{1}{\nrightarrow} (U,X)|I_{XYU}$. By Lemma 3, we know $Y \overset{(3)}{\nrightarrow} X|I_{XZ}$ *if and only if*

$$\pi^{(2)}_{XU,1} = \pi_{XU,2} + \pi_{XX,1}\pi_{XU,1} + \pi_{XV,1}\pi_{VU,1} = 0, \tag{28}$$

and by Theorem 2, $V \overset{1}{\nrightarrow} (U,X)|I_{XYU}$ implies $\pi_{UV,i} = \pi_{XV,i} = 0$, for each $i \geq 1$. Because $Y \overset{(2)}{\nrightarrow} X|I_{XZ}$, by part $(i)$, $\pi_{XU,1} = 0$, and $Y \overset{(3)}{\nrightarrow} X|I_{XZ}$ *if and only if*

$$\begin{aligned} 0 &= \pi^{(2)}_{XU,1} = \pi_{XU,2} + \pi_{XX,1}\pi_{XU,1} + \pi_{XV,1}\pi_{VU,1} \\ &= \pi_{XU,2} + \pi_{XX,1} \times 0 + 0 \times \pi_{VU,1} = \pi_{XU,2}. \end{aligned} \tag{29}$$

Finally, for $(v)$, assume $\pi_{XU,i} = \pi_{VU,j} = 0$, $i = 1...2$, $j = 1$. Because $\pi_{XU,1} = 0$, from part $(ii)$ we immediately deduce$Y \overset{(2)}{\nrightarrow} X|I_{XZ}$. From Lemma 3, therefore, $Y \overset{(3)}{\nrightarrow} X|I_{XZ}$ *if and only if*

$$\pi^{(2)}_{XU,1} = \pi_{XU,2} + \pi_{XX,1}\pi_{XU,1} + \pi_{XV,1}\pi_{VU,1} = 0, \tag{30}$$

where $\pi_{XU,i} = \pi_{VU,j} = 0$, $i = 1...2$, $j = 1,$, hence

$$\begin{aligned} \pi^{(2)}_{XU,1} &= \pi_{XU,2} + \pi_{XX,1}\pi_{XU,1} + \pi_{XV,1}\pi_{VU,1} \\ &= 0 + \pi_{XX,1} \times 0 + \pi_{XV,1} \times 0 = 0. \end{aligned} \tag{31}$$

Therefore, $Y \overset{(3)}{\nrightarrow} X|I_{XZ}$ follows. ■

**Proof of Corollary 5.** The only claims not immediately implied by Lemma 3 and (8) are $(iii)$ and $(iv)$.

For $(iii)$, we assume $Y \overset{(2)}{\nrightarrow} X|I_{XZ}$ and $\pi_{XU,2} = \pi_{XV,1} = 0$. From (8) this implies $\pi^{(2)}_{XU,1} = 0$, hence $Y \overset{(3)}{\nrightarrow} X|I_{XZ}$, cf. Lemma 3.$i$. Using Lemma 3.$ii$, the assumption $\pi_{XU,2} = \pi_{XV,1} = 0$, and $\pi_{XU,1} = 0$ due to $Y \overset{(2)}{\nrightarrow} X|I_{XZ}$, cf. Lemma

3.$i$, we then deduce

$$\begin{aligned}\pi^{(3)}_{XU,1} &= \pi_{XU,3} + \sum\nolimits_{i=1}^{2} \pi^{(3-i)}_{XX,1}\pi_{XU,i} + \sum\nolimits_{i=1}^{2} \pi^{(3-i)}_{XV,1}\pi_{VU,i} \qquad (32)\\ &= \pi_{XU,3} + \pi^{(2)}_{XX,1}\pi_{XU,1} + \pi_{XX,1}\pi_{XU,2} + \pi^{(2)}_{XV,1}\pi_{VU,1} + +\pi_{XV,1}\pi_{VU,2}\\ &= \pi_{XU,3} + \pi^{(2)}_{XX,1} \times 0 + \pi_{XX,1} \times 0 + \pi^{(2)}_{XV,1}\pi_{VU,1} + +0 \times \pi_{VU,2}\\ &= \pi_{XU,3} + \pi^{(2)}_{XV,1}\pi_{VU,1}.\end{aligned}$$

Using (5) and $Y \overset{1}{\nrightarrow} X|I_{XZ}$ *if and only if* $\pi_{XY,i} = 0$, $i \geq 1$, we have

$$\begin{aligned}\pi^{(2)}_{XV,1} &= \pi_{XV,2} + \pi_{XX,1}\pi_{XV,1} + \pi_{XY,1}\pi_{YV,1} + \pi_{XU,1}\pi_{UV,1} + \pi_{XV,1}\pi_{VV,1} \qquad (33)\\ &= \pi_{XV,2} + \pi_{XX,1} \times 0 + 0 \times \pi_{YV,1} + 0 \times \pi_{UV,1} + 0 \times \pi_{VV,1}\\ &= \pi_{XV,2},\end{aligned}$$

hence $\pi^{(3)}_{XU,1} = \pi_{XU,3} + \pi_{XV,2}\pi_{VU,1}$. From Lemma 3.$i$ we conclude $Y \overset{(4)}{\nrightarrow} X|I_{XZ}$ *if and only if* $\pi_{XU,3} + \pi_{XV,2}\pi_{VU,1} = 0$.

For $(iv)$, we assume $Y \overset{(2)}{\nrightarrow} X|I_{XZ}$ and $\pi_{XU,2} = \pi_{VU,1} = 0$, hence $\pi^{(3)}_{XU,1}$ reduces to

$$\begin{aligned}\pi^{(3)}_{XU,1} &= \pi_{XU,3} + \pi^{(2)}_{XX,1}\pi_{XU,1} + \pi_{XX,1}\pi_{XU,2} + \pi^{(2)}_{XV,1}\pi_{VU,1} + \pi_{XV,1}\pi_{VU,2} \qquad (34)\\ &= \pi_{XU,3} + \pi^{(2)}_{XX,1} \times 0 + \pi_{XX,1} \times 0 + \pi^{(2)}_{XV,1} \times 0 + \pi_{XV,1}\pi_{VU,2}\\ &= \pi_{XU,3} + \pi_{XV,1}\pi_{VU,2}.\end{aligned}$$

The claim then follows from Lemma 3.$i$. ■

# References


[1] Chao, J., Corradi, V. and N. Swanson, 2001, An Out of Sample Test for Granger Causality, Macroeconomic Dynamics 5, 598-620.

[2] Dufour, J. and E. Renault, 1994, Short Run and Long Run Causality in Time Series: Theory, Technical Report, Centre de Recherche et Développement en Économique, Université de Montréal, and Institut d'Economie Industrielle, Université des Sciences Sociales de Toulouse.

[3] Dufour, J. and E. Renault, 1998, Short Run and Long Run Causality in Time Series: Theory, Econometrica 66, 1099-1125.

[4] Dufour, J. and T. Jouini, 2003, Finite Sample Simulation-Based Tests in VAR Models with Applications to Order Selection and Causality Tests, Discussion Paper, Centre Interuniversitaire de Recherche un Analyse des Organisations (CIRANO) and Centre Interuniversaire de Recherche en Économie Quantitative (CIREQ), Université de Montréal.

[5] Dufour, J., D. Pelletier and E. Renault, 2003, Short Run and Long Run Causality in Time Series: Inference, Journal of Econometrics (forthcoming).

[6] Feige, E.L. and D.K. Pearce, 1979, The Causal Relationship Between Money and Income: Some Caveats for Time Series, Review of Economics and Statistics 61, 521-533.

[7] Friedman, B., and K. Kuttner, 1992, Money, Income, Prices, and Interest Rates, American Economics Review 82, 472-492.

[8] Friedman, B., and K. Kuttner, 1993, Another Look at the Evidence on Money-Income Relationship, Journal of Economics 57, 189-203.

[9] Granger, C.W.J., 1969, Investigating Causal Relations by econometric Models and Cross-Spectral Methods, Econometrica 37, 424-438.

[10] Granger, C.W.J., 1980, Testing for Causality: A Personal Viewpoint, Journal of Economic Dyanmics and Control 2, 329-352.

[11] Granger, C.W.J., 1988, Some Recent Developments in a Concept of Causality, Journal of Econometrics 39, 199-211.

[12] Hill, Jonathan B., 2004, Efficient Tests of Long-Run Causation in Trivariate VAR Processes with a Rolling Window Study of the Money-Income Relationship, Dept. of Economics Working Paper #04-13, Florida International University; available at http://econwpa.wustl.edu:80/eps/mac/papers/0407/0407013.pdf.

[13] Hill, Jonathan B., 2005, Causation Delays and Causal Neutralization for General Horizons: The Money-Output Relationship Revisited, Dept. of Economics Working Paper #04-03, Florida International University; available at http://econwpa.wustl.edu:80/eps/em/papers/0402/0402002.pdf.

[14] Hsiao, C., 1979, Auoregressive Modeling of Canadian Money and Income Data, Journal of American Statistical Association 74, 553-560.

[15] Lütkepohl, H., 1991, Introduction to Multiple Time Series Analysis (Springer-Verlag: Berlin).

[16] Lütkepohl, H., 1993, Testing for Causation Between Two Variables in Higher Dimensional VAR Models, in Studies in Applied Econometrics, H. Schneewiess and K. Zimmerman (eds.) (Springer-Verlag: Heidelberg)

[17] Lütkepohl, H., and M. Müller, 1994, Testing for Multi-Step Causality in Time Series, mimeo, Institut für Statistik und Ökonometrie, Humbolt-Universität zu Berlin.

[18] Lütkepohl, H., and M. Burda, 1997, Modified Wald tests under Nonregular Conditions, Journal of Econometrics 78, 315-332.

[19] Renault, E. and A. Szafarz, 1991, True Versus Spurious Instantaneous Causality, Technical Report 9103, Centre d'Economie Mathématique, Université Libre de Bruxelles.

[20] Rothman, P., D. van Dijk and P.H. Franses, 2001, A multivariate STAR analysis of the relationship between money and output, Macroeconomic Dynamics 5, 506-532.

[21] Sims, C., 1972, Money, Income and Causality, American Economics Review 62, 540-552.

[22] Sims, C., 1980, Macroeconomics and Reality, Econometrica 48, 1-48.

[23] Stock, J.H., and M.W. Watson, 1989, Interpreting the Evidence on Money-Income Causality, Journal of Econometrics 40, 161-181.

[24] Swanson, N., 1998, Money and Output Viewed Through a Rolling Window, Journal of Monetary Economics 4, 455-473.

[25] Tiao, G.C. and G.E.P. Box (1981), Modeling Multiple Time Series with Applications, Journal of the American Statistical Association 76, 802-816.

[26] Wiener, N., 1956, The Theory of Prediction, in The Theory of Prediction, ed. E. F. Beckenback (McGraw-Hill: New York).