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Causality and Falsifiability in Macroeconomics

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To Alice, Amanda and Maya, the direct causes of my happiness.

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Resumo

Desenvolvemos o conceito de resposta ao impulso em um formato causal, definindo ferramentas analíticas adequadas para diferentes análises de políticas e aplicando nossas técnicas em modelos contendo características como variáveis de confusão ou não linearidades através de experimentos de Monte Carlo. Também aplicamos algumas das técnicas apresentadas a problemas práticos em macroeconomia, calculando respostas ao impulso para o PIB, taxa de juros, inflação e taxa de câmbio real a decisões de política monetária do Banco Central do Brasil. O conceito de causalidade em estatística e econometria também é revisto, contrastando nossa abordagem com outras mais tradicionais, destacando diferenças conceituais e mostrando como um importante aspecto das teorias científicas, chamado falseabilidade, pode ser melhorado em economia se uma abordagem causal ao desenvolvimento de teorias for levada em consideração.

Palavras-chave: Análise causal; Modelo Causal Estrutural; Causalidade em séries temporais; Resposta ao impulso; Política monetária; Falseabilidade.

Abstract

We develop the concept of impulse response in a causal fashion, defining analytical tools suitable for different policy analysis and giving applications of our techniques to models containing features like confounding or nonlinearities through Monte Carlo experiments. We also apply some techniques presented to practical macroeconomic problems, computing impulse responses of GDP, interest rate, inflation and real exchange rate to monetary policy decisions of Banco Central do Brasil. The concept of causality in statistics and econometrics is also reviewed, contrasting our approach with traditional ones, presenting conceptual differences and showing how an important feature of scientific theories, called falsifiability, may be enhanced in economics if a causal approach to theory development is carried on.

Keywords: Causal analysis; Structural Causal Model; Time series causality; Impulse response; Monetary policy; Falsifiability.

List of abbreviations and acronyms

BCB	Banco Central do Brasil, the central bank of Brazil.
CEF	Conditional expectation function.
CGI	Causal generalized impulse response function.
CIRF	Counterfactual impulse response function.
COPOM	Comitê de política monetária, the brazilian committee that decides monetary policy in BCB.
DAG	Directed acyclic graph.
DGP	Data generating process.
DSGE	Dynamic stochastic general equilibrium model.
ECDSP	Expected counterfactual difference sequential plan response.
ECGI	Expected causal generalized impulse response function.
EGCIRF	Expected generalized counterfactual impulse response function.
FOMC	Federal open market committee.
GAM	Generalized additive model.
GCIRF	Generalized counterfactual impulse response function.
GDP	Gross domestic product.
IPCA	Índice nacional de preços ao consumidor amplo, the brazilian consumer price index.
IRF	Impulse response function.
LATE	Local average treatment effect.
OLS	Ordinary least squares.
SCM	Structural causal model.
SEM	Structural equations model.
SVAR	Structural vector autoregression.
VAR	Vector autoregression.

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Introduction

"In economics, it is easier to agree on the data than to agree on causality."

— Moshe Vardi

This thesis presents the concept of Impulse Response Function (IRF) in a causal fashion, which may or may not agree with its current use in econometrics literature. This agreement depends on context and structural assumptions underlying models of interest. Concepts presented here are closer to the interpretation of causal effects in microeconometrics literature (see, e.g., Cameron and Trivedi (2005)). Nonetheless, we apply these ideas in a time series context looking for applications in empirical macroeconomics.

In a nutshell, a Counterfactual Impulse Response Function (CIRF), defined in Chapter 2, is some response of a system relative to a baseline, resultant from the *manipulation* of one or more of its inputs. The baseline may be another intervention or no intervention at all. Manipulation is understood as a policy intervention stripped out of any confounding effect (an exogenous intervention). We regard this of interest because various macroeconomic policies are designed exactly in this way, and policymakers are interested in these interventional effects. Our practical example is the monetary policy with inflation targeting regime based on manipulation, by a Central Bank, of the short-term nominal interest rate. This is the approach adopted in Brazil since 1999, and in Chapter 3 we estimate CIRFs for Brazil to infer causal effects of variations on Selic rate over macroeconomic aggregates.

Our approach contrasts with main approaches in macroeconometrics. These are broad and, in practice, vary a lot, but the most popular approaches are New Keynesian Dynamic Stochastic General Equilibrium models (DSGE) such as SAMBA at Central Bank of Brazil (CASTRO et al., 2015) or NAWM and CMR at European Central Bank (SMETS et al., 2010), Structural Vector Autoregressions (SVAR) and Semi-structural models such as Brayton et al. (2014), Kichian, Rumler and Corrigan (2010) or Minella and Souza-Sobrinho (2009). DSGE models does not have a direct causal content, instead being more prone to policy regime analysis than to direct interventions. Semi-structural models may have a causal interpretation close to ours, but demand lots of debatable modeling assumptions. SVARs may have a direct causal interpretation or not, depending on modeling assumptions. These issues are discussed in more detail in Chapters 1 and 2.

We regard our approach as a simple way to infer the effects of causal interventions on economic systems evolving in time[1]. Being simple, it has fewer assumptions than mainstream approaches, serving two main purposes: test economic assumptions and theories and be a benchmark for more complex models whose structural assumptions are beyond direct empirical test.

Moreover, our approach, together with Structural Causal Models (SCMs) (PEARL, 2009) in general, serves as a general framework for identification of causal effects expressed as combinations of conditional distributions or expectations, opening paths for causal analysis based in all sorts of inferential techniques designed for fitting probability distributions, be them classical statistical ones or modern machine learning techniques.

We define CIRFs as (almost) model-free concepts, avoiding references to any additivity or linearity assumption. To do so, in Chapter 2 we carefully define what is a causal intervention on a system and then define CIRFs based on this concept. Furthermore, we devise methods to estimate these CIRFs based on observational data whenever possible, discuss cases where it is impossible and point to testable implications of the qualitative causal models underlying the identification of these causal effects. Also, we made Monte Carlo simulations of systems containing features such as endogeneity or non-linearities and apply our methods

[1] Actually, it works for any time evolving system which can be modeled in a causal fashion.

on these artificial data to show the effectiveness of our approach. Finally, we apply part of the machinery developed in Chapter 2 to real macroeconomic problems, identifying causal impulse responses of GDP, interest rate, inflation and real exchange rate to monetary policy decisions in Chapter 3. The conceptual framework adopted in defining causal interventions is the Structural Causal Model (SCM) approach detailed in Pearl (2009), which is briefly discussed in Chapter 1. We also make conceptual considerations regarding causal reasoning, modeling strategies, regressions and falsifiability with a critical review of the literature including historical facts important for the big picture perspective. Appendix A details models used for inference in Chapter 2, Appendix B gives information on data treatment used in Chapter 3 and Appendix C gives robustness checks and diagnostics for inferences made in Chapter 3.

1 Literature Review

> *"A great book seeks to explain causality, not correlation. It works to point out the circumstances in which it works, and where it doesn't. And in so doing, it is broadly applicable."*
>
> — Clayton M. Christensen

1.1 Introduction

In this chapter we review the literature on identification of causal relations in econometrics and statistics. Besides reviewing recent advances only, we go back to the origins of concepts essential to this endeavor because, in our opinion, and also according to Pearl (2009) and some references therein, something was "missing" in the way. The missing link was exactly a *general language* for causality, which was addressed in some ways in statistics and econometrics, but not in a framework as general as the Structural Causal Model (SCM) presented and axiomatized in Balke and Pearl (1994a), Balke and Pearl (1994b), Galles and Pearl (1997), Galles and Pearl (1998), Halpern (2000) and Pearl (2009). Our causal approach to impulse responses definition and estimation presented in Chapter 2 is based on SCMs.

1.2 A brief history of causality in statistics and econometrics

In the late 19th and the early 20th century, statistics emerged as an independent field mainly through the works of Francis Galton and Karl Pearson. Galton discovered the concept of correlation in the fall of 1888 (STIGLER, 1989), and problems or discussions about the relationship between causal inference and correlation analysis pervades statistical science since then.

For Galton (1889 apud ALDRICH, 1995), correlation measures the extent to which two variables are governed by common causes, a point of view which Aldrich ascribes to the interest of Galton in heredity and physical anthropology. George Udny Yule and Karl Pearson are recognized as the developers of the main interpretation of correlation followed by statisticians for the past century (ALDRICH, 1995), and the distinction between correlation and causation is stressed by Yule in his works (see, e.g., Yule (1900) and Yule (1903)). Aldrich (1995) analyzes Pearson and Yule development of the concept of correlation and its possible spuriousness.

At this time, a new field of research was born. Epstein (2014) dates the beginning of modern econometrics with an analysis of labor market by Henry Ludwell Moore (MOORE, 1911), but the first Nobel Memorial Prize in Economic Sciences in 1969 recognizes Jan Tinbergen and Ragnar Frisch as founders of econometrics. Anyway, since its inception, econometrics dealt with causality related ideas. Moore (1914) is the first attempt of inference on a demand curve using multiple regression (EPSTEIN, 2014). More interestingly, his modeling strategy used summer rain as an exogenous driver of supply of agricultural commodities, and Epstein (2014) says "it seems evident that Moore assumed the exogenous changes in supply would trace out the demand curve".

Another important step towards a science of causality was done by geneticist Sewall Green Wright, who created what became known as path analysis (WRIGHT, 1921; WRIGHT, 1923; WRIGHT et al., 1925). This technique is a special case of structural equation modeling (SEM) and an ancestor of modern graphical models largely used in SCMs. Causality was a so controversial issue that one of Wright's critics denied its independent existence at all, moreover asserting that causality is indeed correlation:

> "Causation" has been popularly used to express the condition of association, when applied to natural phenomena. There is no philosophical basis for giving it a wider meaning than partial or

> absolute association. In no case has it been proved that there is an inherent necessity in the laws of nature. Causation is correlation. (NILES, 1922)

Despite extreme viewpoints, researchers in statistics continued thinking about causation at some level. Fisher (1926) showed that randomization could be used to make causal inference. His highly influential books *The Design of Experiments* (FISHER, 1925) and *Statistical Methods for Research Workers* (FISHER et al., 1926) are considered the foundational works in the literature of experimental design (YATES, 1964; STANLEY, 1966; BOX, 1980), also introducing the important concept of null hypothesis and the convenience rule of 5% level of significance for its rejection. Neyman (1923) also made contributions on randomized experiments (RUBIN, 1990), and later work together with ideas in Rubin (1974) laid down what is now known in statistics literature of causal inference as the Neyman-Rubin potential outcome model (PEARL, 2009).

Meanwhile, in the econometric literature, complications arose mainly on simultaneous equations problems, when researchers tried to estimate supply and demand equations. Wright's path analysis try to infer the causal structure of a set of variables through the matrix of correlation coefficients, and the author saw that market equilibrium could be analyzed by the same tools he developed for heredity problems. Apparently, Sewall Wright was the first to use what is now known as a reduced form model to analyze market equilibrium, and his work was the culminating point of econometric research in United States at that time (EPSTEIN, 2014), although paradoxically it had no impact on econometrics. Back then, in the late 1920's, researchers in Europe, led by Jan Tinbergen and Ragnar Frisch, also studied and help make clear the somewhat unusual nature of econometric inference (EPSTEIN, 2014).

In his 1943-1944 papers: *The Statistical Implications of a System of Simultaneous Equations* (HAAVELMO, 1943) and *The Probability Approach in Econometrics* (HAAVELMO, 1944), the 1989 Nobel Memorial Prize laureate Trygve Haavelmo made fundamental contributions to the analysis of statistical models in economics and their causal content. As highlighted by Heckman and Pinto (2015), he "laid the foundation for counterfactual policy analysis", distinguishing the concept of "fixing" from conditioning.

The conceptual breakthrough of Haavelmo's approach was a distinction between the impact on a system output carried out by "hypothetical" manipulations of the input, and correlations of output and input in observational data (HECKMAN; PINTO, 2015). The former was regarded as causal, and the latter correlational. A simple example may shed some light on this distinction: imagine a policymaker trying to infer what is the impact of police forces on crime rates (a well known problem in studies of crime). Causal intuition says "more police, less crime", but (correlational) data says "more police, more crimes". The issue is obvious. Areas with higher crime rates induce more police forces on those same areas, exactly because policymakers believe that more police *causes* less crime. So, when looking naively at data, more police forces correlates positively with higher crime rates[1]. The real causal question is: if there were more police forces on otherwise identical situations, would crime rates retreat? Notice the counterfactual nature of the question. Actually, Haavelmo's equation manipulation may be seen as an hypothetical experiment, where the experimenter *exogenously* manipulates police forces in some area independently of *potential crime rates on that area for any level of police forces*. These concepts effectively formalize a distinction between correlation and causation, giving precise meaning to the phrase "correlation does not imply causation".

These developments in statistics and econometrics led to two main lines of research on causality.

[1] It is frequent in the literature to regard the positive correlation in this naive data analysis as spurious. We prefer to think that this correlation is legitimate. Spurious is to infer a causal relation from mere correlation without any reason for that. Indeed, information about police forces in some place may help to predict crime rates in this same place, regardless the causal effects of police forces on crime. This is exactly what a conditional distribution is. Our viewpoint emphasizes the distinction between correlation and causation. As mentioned in Aldrich (1995), correlation analysis may go wrong by different reasons. Spurious regression on non-stationary time series (or even stationary ones) (YULE, 1926; GRANGER; NEWBOLD; ECONOM, 2001) induced by faulty statistical inference is fundamentally different from spurious regression resulting from inadequate control of confounders (common causes). This point is stressed in Aldrich (1995), and we think using the same terminology for two fundamentally different concepts misleading.

According to Pearl (2009), the Structural Equation Modeling (SEM) approach to causal inference, with its origins in (WRIGHT, 1921) and (HAAVELMO, 1943), was adopted by economists and social scientists. The Neyman-Rubin potential outcome approach (NEYMAN, 1923; RUBIN, 1974) was adopted by a group of statisticians, and was later adopted by economists, specially microeconometrists (see, e.g., Cameron and Trivedi (2005)). But Pearl (2009) emphasizes that none of these two approaches has become standard in causal modeling. Moreover, Pearl advocates his own line of research, referred as the Structural Causal Models approach (SCM) (PEARL, 2015), also known as graphical causal models, as an unifying language for causal inference. This is the way we follow here.

Before next section, a few words might be said about the concept of Granger-causality, largely used in time series econometrics (GRANGER, 1969). Granger-causality does not necessarily means causality, especially if there are non-measured confounders in the analysis. Roughly speaking, a variable X_t Granger-causes Y_{t+1} if it is significant to predict Y_{t+1} *given all information available at time* t. Of course, consider all information up to t is impossible, and in practice researchers look for just a bunch of variables, frequently missing confounders that may be common causes of X_t and Y_{t+1} (HSIAO, 1982; CARTWRIGHT, 2007; EICHLER, 2012; MAZIARZ, 2015).

These issues on Granger-causality elicit the following fact: to explain is not the same as to predict. As pointed out by Shmueli et al. (2010), this distinction is recognized in the philosophy of science, but overlooked by the statistical literature. We think this distinction is more transparent in the econometrics literature, but not always taken seriously, especially in time series contexts.

1.3 What is a model? Regression vs. structural equations

Before reviewing the main concepts of Structural Causal Models, we reflect about the concept of model itself. According to Oxford Dictionary, a model is “a simple description of a system, used for explaining how something works or calculating what might happen, etc.”. Notice the distinction among concepts of explaining and predicting. In our context this distinction is crucial. A classical econometric example is the distinction among structural and reduced form models, like Vector Autoregressions and Structural Vector Autoregressions (VARs and SVARs, respectively), for example. VARs may have a good forecast performance (prediction), but its interpretation may be impossible (explanation). Because of this limitation, SVARs were introduced.

Hurwicz (1966) accounts for the original meaning of “structural” in econometrics. He pointed out that a system in its structural form may be not uniquely defined, with different systems consistent with the same set of possible values for its variables. He calls this set of possible solutions for the system its *history*. If more than one configuration of the system generates the same history, than the model is *non-identifiable*, and may only be identified with additional information.

An important property of structural models, stressed by Hurwicz (1966) and in various forms also by Marschak (1950), Mesarovic (1969), Sims (1976), Simon (1977) and others, is the notion that a structural model remains *invariant* to some changes in the system. For example, the other parameters remain the same if one of them is changed, or the relations among variables remain the same if one equation is substituted by another. This issue is examined at length in Pearl (2009), with historical and philosophical discussions.

For us, a model models what it is designed to model. This may sound silly, but is much overlooked. For example, a reduced-form VAR is a model for correlations among a set of variables, and does not make sense to ask for causal conclusions based on it, unless one assumes its equations as representing causal relations, which is not usually the case, or exists an identifiable causal model equivalent to it, like an SVAR.

The demand-supply economic model for price and quantity of some good is another example. The demand and supply curves have a causal meaning, because they represent, for the demand, the quantity

consumers would buy given some price p, and the supply curve is similar. These are counterfactual queries. Also, these curves may be interpreted in a manipulative way, predicting how firms and consumers would react to *exogenous* variations on p. But, the demand-supply model goes further, and asserts that the market price on equilibrium is given by the intersection of both curves. This is a model of market equilibrium, and cannot answer questions about how price reaches this equilibrium, something stressed out as early as in Walras (1896). In this form, the demand-supply model *does not* admit manipulation of prices, because the result of such manipulation cannot be answered by the model. Indeed, given its parameters, the solution for the price is unique and cannot be changed.

Another possibility is to interpret demand-supply causal relations on a different manner. Suppose that quantity demanded q is caused by prices p through a demand equation, and prices respond to demand through another equation[2]. In this form causality goes in a way that admits price manipulation, but it is possible to rearrange equations and have the same model as before. This highlights another aspect of causal relations: they may be *unidirectional*, an issue we discuss later in this section.

These observations about the demand-supply model are important, since a large part of macroeconomic models model demand-supply type relations. So, in a model where monetary policy is enforced by a central bank through a Taylor rule, for example, the interest rate path prescribed by the model is a rate of equilibrium which obeys the Taylor rule and other constraints in the economy, modeled by other equations in the model. This mechanistic equilibrium behavior contrasts with the direct manipulation of the interest rate by central banks with inflation target regimes, like the Banco Central do Brasil (BCB), which targets an overnight interest rate to some value intending to bring inflation rate to its pre-determined target (BOGDANSKI; TOMBINI; WERLANG, 2000). Of course, it may be argued that central bankers follow a Taylor rule, and since this is not perfectly true, traditional economic models put an exogenous random error on the rule. Causal questions about manipulation of interest rates are answered by the system's response to some of these exogenous shocks.

Another important concept is that of a statistical model. Some models are statistical by nature, like VARs mentioned before, but the fact that some random variables are part of the model does not make them *purely statistical* in our understanding. McCullagh (2002) cites accepted theories in statistical literature regarding statistical models as sets of probability distributions over a sample space. That is, a statistical model is a pair $\langle S, \mathcal{P} \rangle$ where S is the sample space and $\mathcal{P}$ is a *set* of probability distributions over S. In a parameterized statistical model there is a parameter set Θ and a function $P : \Theta \mapsto \mathcal{P}$ assigning to a parameter $\theta \in \Theta$ the probability distribution P_θ on S. A parameterization is identifiable if distinct parameter values give rise to distinct distributions (COX; HINKLEY, 1979; MCCULLAGH, 2002; LEHMANN; CASELLA, 2006; BERNARDO; SMITH, 2009; COX, 2017). These authors does not detail what these different parameterizations may represent. They can be simple parameters of the distribution, like mean and variance for the normal model, but they also may be parameters representing mechanisms of variable determination, or be interpreted as causal chains, or even another thing, like a long run equilibrium value. So, we will use the term *pure statistical model* to represent a model of some distribution regardless the mechanism of interaction among its variables. A multivariate normal distribution can inform us about joint and conditional distributions, independently on how the interactions of the variables leads to the multivariate normal behavior. Correlations in this model may arise because of causal links among variables, or have its origin on unobserved common causes. The important thing is that the multivariate normal model alone *cannot* answer questions about these mechanisms.

So, we think it is useful distinguish among what is regarded as a *pure* statistical description of data, their data generating process (DGP) and causal inferences regarding these variables. The data generating process is the *mechanism* that generates data. These mechanisms may represent causal relations or another

[2] Pearl (2009, p. 215–217) analyses this model using causal reasoning.

thing. Suppose that the DGP of $\{y, x_1, \ldots, x_k, q, u\}$ is the system of equations (1.1)[3]:

$$\begin{aligned} y &= \beta_0 + \beta_1 x_1 + \cdots + \beta_k x_k + u \\ u &= \gamma q + \epsilon_u \\ x_k &= \delta q + \epsilon_x \end{aligned} \tag{1.1}$$

where $x_1, \ldots, x_{k-1}, \epsilon_x, \epsilon_u$ and q are all $N(0,1)$ independent random variables. These equations are seem as structural, since they are invariant to exogenous manipulation. Indeed, all endogeneity is explicitly modeled, through equations for x_k and u. A pure statistical description of $\{y, x_1, \ldots, x_k, q, u\}$ generated by (1.1) is the distribution $P(v)$ of the vector $v = (y, x_1, \ldots, x_k, q, u)'$, which is multivariate normal $N(\mu, \Sigma)$, with

$$\mu = \begin{bmatrix} \beta_0 \\ 0 \\ \vdots \\ 0 \end{bmatrix} \tag{1.2}$$

and

$$\Sigma = \begin{bmatrix} \sum_{i=1}^{k-1} \beta_i^2 + (\beta_k\delta + \gamma)^2 + \beta_k^2 + 1 & \beta_1 & \ldots & \beta_{k-1} & \beta_k(\delta^2+1) + \gamma\delta & \beta_k\delta + \gamma & \beta_k\gamma\delta + \gamma^2 + 1 \\ \beta_1 & 1 & \ldots & 0 & 0 & 0 & 0 \\ \vdots & \vdots & \ddots & \vdots & \vdots & \vdots & \vdots \\ \beta_{k-1} & 0 & \ldots & 1 & 0 & 0 & 0 \\ \beta_k(\delta^2+1) + \gamma\delta & 0 & \ldots & 0 & \delta^2 + 1 & \delta & \gamma\delta \\ \beta_k\delta + \gamma & 0 & \ldots & 0 & \delta & 1 & \gamma \\ \beta_k\gamma\delta + \gamma^2 + 1 & 0 & \ldots & 0 & \gamma\delta & \gamma & \gamma^2 + 1 \end{bmatrix}. \tag{1.3}$$

In this form, both descriptions are equivalent. To see why, notice that (1.2) and (1.3) follow directly from (1.1). Conversely, given $P(v) = P(y, x_1, \ldots, x_k, q, u)$, which is $N(\mu, \Sigma)$ as above, we have[4]:

$$\begin{bmatrix} u \\ q \end{bmatrix} \sim N\left(\begin{bmatrix} 0 \\ 0 \end{bmatrix}, \begin{bmatrix} \gamma^2 + 1 & \gamma \\ \gamma & 1 \end{bmatrix} \right) \tag{1.4}$$

and this implies $u|q \sim N(\gamma q, 1)$, or $u = \gamma q + \epsilon_u$, with $\epsilon_u \sim N(0,1)$. Also,

$$\begin{bmatrix} x_k \\ q \end{bmatrix} \sim N\left(\begin{bmatrix} 0 \\ 0 \end{bmatrix}, \begin{bmatrix} \delta^2 + 1 & \delta \\ \delta & 1 \end{bmatrix} \right) \tag{1.5}$$

and, analogously, $x_k = \delta q + \epsilon_x$, with $\epsilon_x \sim N(0,1)$. The case for y is a bit more cumbersome. First, notice that

$$\begin{bmatrix} y \\ x_1 \\ \vdots \\ x_{k-1} \\ x_k \\ q \end{bmatrix} \sim N\left(\begin{bmatrix} \beta_0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}, \begin{bmatrix} \sum_{i=1}^{k-1} \beta_i^2 + (\beta_k\delta + \gamma)^2 + \beta_k^2 + 1 & \beta_1 & \ldots & \beta_{k-1} & \beta_k(\delta^2+1) + \gamma\delta & \beta_k\delta + \gamma \\ \beta_1 & 1 & \ldots & 0 & 0 & 0 \\ \vdots & \vdots & \ddots & \vdots & \vdots & \vdots \\ \beta_{k-1} & 0 & \ldots & 1 & 0 & 0 \\ \beta_k(\delta^2+1) + \gamma\delta & 0 & \ldots & 0 & \delta^2 + 1 & \delta \\ \beta_k\delta + \gamma & 0 & \ldots & 0 & \delta & 1 \end{bmatrix} \right). \tag{1.6}$$

Then,

$$y|x_1, \ldots, x_k, q \sim N\left(\overline{\mu}, \overline{\Sigma}\right) \tag{1.7}$$

with[5],

$$\overline{\mu} = \beta_0 + \Sigma_{12}\Sigma_{22}^{-1} v \tag{1.8}$$

[3] This DGP follows closely Wooldridge (2010, p. 66–67).
[4] Formulas for conditional and marginal distributions given a joint normal distribution may be found in Eaton (1983).
[5] Ibid.

and

$$\overline{\Sigma} = \Sigma_{11} - \Sigma_{12}\Sigma_{22}^{-1}\Sigma_{21} \tag{1.9}$$

where

$$\Sigma_{11} = \sum_{i=1}^{k-1}\beta_i^2 + (\beta_k\delta + \gamma)^2 + \beta_k^2 + 1, \tag{1.10}$$

$$\Sigma_{12} = \begin{bmatrix}\beta_1 & \dots & \beta_{k-1} & \beta_k(\delta^2+1) + \gamma\delta & \beta_k\delta + \gamma\end{bmatrix}, \tag{1.11}$$

$$\Sigma_{22} = \begin{bmatrix}1 & \dots & 0 & 0 & 0\\ \vdots & \ddots & \vdots & \vdots & \vdots\\ 0 & \dots & 1 & 0 & 0\\ 0 & \dots & 0 & \delta^2+1 & \delta\\ 0 & \dots & 0 & \delta & 1\end{bmatrix} \tag{1.12}$$

and

$$\Sigma_{21} = \Sigma_{12}'. \tag{1.13}$$

So,

$$\overline{\mu} = \beta_0 + \beta_1 x_1 + \cdots + \beta_k x_k + \gamma q \tag{1.14}$$

and

$$\overline{\Sigma} = 1 \tag{1.15}$$

which implies

$$y = \beta_0 + \beta_1 x_1 + \cdots + \beta_k x_k + \gamma q + \epsilon_u \tag{1.16}$$

with $\epsilon_u \sim N(0,1)$. Hence, (1.2) and (1.3) imply (1.1). The situation is different if only $\{y, x_1, \dots, x_k\}$ are observable and we are modeling their joint behavior. From (1.2) and (1.3), the joint distribution of the observables is $N(\mu_{obs}, \Sigma_{obs})$ with:

$$\mu_{obs} = \begin{bmatrix}\beta_0\\ 0\\ \vdots\\ 0\end{bmatrix} \tag{1.17}$$

and

$$\Sigma_{obs} = \begin{bmatrix}\sum_{i=1}^{k-1}\beta_i^2 + (\beta_k\delta+\gamma)^2 + \beta_k^2 + 1 & \beta_1 & \dots & \beta_{k-1} & \beta_k(\delta^2+1)+\gamma\delta\\ \beta_1 & 1 & \dots & 0 & 0\\ \vdots & \vdots & \ddots & \vdots & \vdots\\ \beta_{k-1} & 0 & \dots & 1 & 0\\ \beta_k(\delta^2+1)+\gamma\delta & 0 & \dots & 0 & \delta^2+1\end{bmatrix}. \tag{1.18}$$

Now, it is *not* possible to infer process (1.1) from (1.17) and (1.18) without further information. Indeed, if we infer Σ_{obs} from data and try to calculate all parameters in (1.18), we will have $k+1$ independent equations to infer $k+2$ parameters. So, the model (1.1) is not identifiable. A model like (1.17) and (1.18), where we have a statistical distribution for observable data is what we meant as a pure statistical model.

The distinction among pure statistical models and DGPs with causal interpretation (or not) have more consequences on commonly used concepts. One of these concepts is that of identification. The identification problem may have different meanings depending on the context. Most commonly in statistics, a model parameter is identifiable if different parameter values induce different probability distributions (LEHMANN; CASELLA,

2006), or, in other words, these parameter values are observationally distinguishable. Casella and Berger (2002) said "identifiability is a property of the model, not of an estimator or estimation procedure". This is fine, but raises the question: what is a model? As seen before, the main concept of model is a bit confuse, and we reserved the term "pure statistical model" for the modeling of the distribution of data, ignoring generating mechanisms. Estimation of this sort of models is just a matter of fitting.

To make things clear, consider an example. In econometrics, the identification problem is almost the same as in statistics, but frequently have to do with some causal inference problem. In Wooldridge (2010), the author refers to the identification problem as the impossibility of *consistent estimation* of parameters in a model with endogenous variables. This seems to contradict Casella and Berger (2002) cited above, which claim that identifiability is a property of the model, not the estimation procedure. Wooldridge (2010, p. 66–67) gives a single equation example:

$$y = \beta_0 + \beta_1 x_1 + \cdots + \beta_k x_k + u, \tag{1.19}$$

where $E(u) = 0$ and $Cov(x_j, u) = 0$, $j = 1, \ldots, k-1$. If $Cov(x_k, u) \neq 0$, then OLS regression of (1.19) leads to inconsistent estimation of all β_j. But, what we get running OLS on data generated by (1.19)? Following Wooldridge (2010), suppose that u contains an omitted variable q uncorrelated with all explanatory variables but x_k, that is, suppose $u = \gamma q + \epsilon_u$ with $\epsilon_u \sim N(0, 1)$ and $Cov(x_k, q) \neq 0$. Such conditions are exactly those generated by model (1.1). The author is interested in the following *conditional expectation*:

$$E(y|x_1, \ldots, x_k, q) = \beta_0 + \beta_1 x_1 + \cdots + \beta_k x_k + \gamma q. \tag{1.20}$$

Running OLS on available data generated by (1.19) we get

$$E(y|x_1, \ldots, x_k) = \delta_0 + \delta_1 x_1 + \cdots + \delta_k x_k \tag{1.21}$$

which is fine as an estimate of the conditional expectation $E(y|x_1, \ldots, x_k)$ but not as an estimate of the *structural equation* (1.19). The issue is that (1.20) have a causal interpretation, while (1.21) does not, and this is the reason why the author is interested on the former but not the latter. Regressions are designed to estimate approximations of conditional expectation functions (CEF) like (1.20) or (1.21) and, in our viewpoint, parameters δ_j in (1.21) are not biased if estimated by OLS. Indeed, if interest is on (1.21), OLS estimates are the best linear unbiased estimators of δ_j for all j, what is well known. But, in econometrics practice it is common to use this bias terminology, something we think is misleading.

In the excellent *Mostly Harmless Econometrics*, Angrist and Pischke (2008, p. 52) say: "A regression is causal when the CEF it approximates is causal". Although it may seen innocuous, this sentence contains the core of our discussion about identification in econometrics. Indeed, equation (1.19) is NOT in the form

$$y = E(y|x_1, \ldots, x_k) + \epsilon, \tag{1.22}$$

where $E(\epsilon|x_1, \ldots, x_k) = 0$. Equation (1.22) is called in Angrist and Pischke (2008, p. 32) the CEF decomposition property, which is valid for ANY random variable with well defined expectations and can be correctly estimated in the linear case by OLS regression of (1.21).

Consider the conditional expectation $E(y|x_1, \ldots, x_k)$ for variables in model (1.1). Through the same steps as before, it can be shown that

$$E(y|x_1, \ldots, x_k) = \beta_0 + \beta_1 x_1 + \cdots + \left(\beta_k + \frac{\gamma\delta}{\delta^2 + 1}\right) x_k. \tag{1.23}$$

If we run the regression

$$y = \beta'_0 + \beta'_1 x_1 + \cdots + \beta'_k x_k + u' \tag{1.24}$$

Wooldridge (2010) says that our estimates will be biased. Well, they are biased as estimators of the parameters of the *structural* equation for y in (1.1) (or (1.19)), as claimed by the author[6]. But, OLS estimators for $\beta'_0, \ldots, \beta'_k$

[6] In our specific example, only β_k would be biased.

are estimators of the *conditional expectation* (1.23). Indeed, following Wooldridge (2010, p. 66–67), consider the "linear projection" of q over $x_1, \ldots, x_k$. Since q is independent of all x_j but x_k, we may write:

$$q = \frac{cov(q, x_k)}{var(x_k)} x_k + \epsilon_q = \frac{\delta}{\delta^2 + 1} x_k + \epsilon_q \tag{1.25}$$

with $\epsilon_q \sim N\left(0, \frac{\delta^2+\gamma^2+1}{\delta^2+1}\right)$. We may use Wooldridge (2010, p. 67) "biased" formula:

$$y = (\beta_0 + \gamma\delta_0) + (\beta_1 + \gamma\delta_1)x_1 + \cdots + (\beta_k + \gamma\delta_k)x_k + \epsilon_u + \gamma\epsilon_q, \tag{1.26}$$

where $\delta_0, \ldots, \delta_k$ are the constant and coefficients of $x_1, \ldots, x_k$ in the linear projection (1.25). After substituting all values, we arrive at (1.23) without bias, as expected by the Gauss-Markov theorem.

So, in our opinion, there is a language inconsistency in using the term bias in econometrics. And this stems from the treatment of regression as a technique for direct estimation of parameters in structural models, something that passes through the ambiguity among CEFs with and without causal content. Wooldridge (2010, p. 13) uses the term "structural conditional expectation" to differentiate them, but this is too much restrictive. As can be seen in Pearl (2009), the front-door criterion results in a distribution that is not simply a conditional expectation[7]. Also, the sequential plan response, explored in Section 2.4, is not simply a conditional expectation and also lots of causal effects identifiable through do-calculus rules (PEARL, 2009) are not expressible as a simple conditional expectations. But they are causal queries of interest, and show that conditional expectations with causal content are just one piece in the universe of causal queries potentially identifiable in a given model.

Then, OLS regressions are tools for linear conditional expectations estimation and must be treated accordingly. If the functional form is adequate is something that may be analyzed from data. But, the causal interpretation of CEFs is something outside the scope of regression analysis, as can be seen in the example, where equation (1.21) is a legitimate CEF but does not have causal content. Causal interpretation of CEFs and identification of causal effects belongs to the domain of causal analysis, and this is one of the reasons for Pearl (2009) to insist in distinct mathematical notations, through the "do" operator and counterfactuals. Of course that our example extracted from Wooldridge (2010) is in any way a critique directed to the author or his book. Indeed, we think Wooldridge (2010) is one of the best available econometrics textbooks, and this is one reason to cite it. His language is overall standard in econometrics literature, and the book is remarkably rigorous and clear. The author is very careful on distinguishing structural equations from conditional expectations and so on. Our point is that the language regarding causal thinking on econometrics and statistics in general is somewhat confusing, even in an excellent book, sometimes clouding concepts that are conceptually simple if you look at them through the lens of causal reasoning, with its sharp distinction between purely statistical (or correlational) concepts and causal queries.

In the example presented, the identifiability problem is a consequence of the unobservability of q and u, a practical limitation that could be overcome by instrumental variables (IV). Someone may argue that the instrumental variables approach shows that this is an estimation problem and the bias terminology is adequate. We do not share this view. The IV approach is indeed a causal effects identification problem, and its closed form as an estimator of regression parameters is a mere algebraic convenience and a consequence of the linearity assumed in most econometric approaches. Indeed, IVs does NOT solve omitted variable bias in the general non-parametric case, always needing some stronger assumptions.

To see the linear case, consider the causal graph in Figure 1[8]. Notice that Z is an instrumental variable for X, because it affects X and all of its effects on Y are due to its interaction with X. In econometric parlance, some unmeasured variables that determine Y are correlated with X, and this is represented in the graph by the dashed arc connecting X and Y. Also, there is a variable Z uncorrelated with these unobserved factors

[7] Although it can be estimated with the help of conditional expectations.

[8] Causal diagrams are part of the machinery of SCMs. For details, see Pearl (2009).

and correlated with X. Notice that paths from Z to unobserved confounders are blocked by colliders in X and Y[9]. Moreover, conditioning on X does NOT make Y and Z independent, because the path $Z \rightarrow X \leftrightarrow Y$ is unblocked by the collider in X, that is, it is not possible to test the validity of this instrument. Anyway, by the rules of do-calculus, the causal effect of X on Y in Figure 1 is non-identifiable.[10]

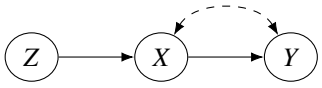

Figure 1 – Directed acyclic graph (DAG) showing an instrumental variable Z.

But, using Wright's path coefficients approach (WRIGHT, 1921; WRIGHT, 1923; WRIGHT et al., 1925), valid for linear models, we may index the arrows in Figure 1 with path coefficients representing linear causal effects (PEARL; GLYMOUR; JEWELL, 2016). These are the coefficients of the linear structural equations relating parent and child variables. For example, suppose that X, Y and Z are related by:

$$\begin{aligned} y &= \beta x + \epsilon_y \\ x &= \alpha z + \epsilon_x \\ z &= \epsilon_z \end{aligned} \tag{1.27}$$

where ϵ_z is independent and ϵ_x is correlated with ϵ_y. This system may be represented by the graph of Figure 2.

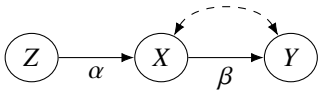

Figure 2 – DAG showing an instrumental variable Z in the linear model given by (1.27).

Now it is easy to see how to use the linearity assumption and IV to identify β. From path coefficients rules (PEARL; GLYMOUR; JEWELL, 2016), the marginal causal effect of Z on Y, that is, the causal effect of a unitary change in Z on the expected value of Y, is given by the product $\gamma = \alpha\beta$, which can be estimated by a simple linear regression of Y on Z. Moreover, α can also be estimated regressing X on Z. So, $\beta = \frac{\gamma}{\alpha}$, which is the familiar two stages least squares IV estimator. Another approach that assumes a sufficient condition for IV to work, different from linearity, is known as LATE (Local Average Treatment Effect), presented in Angrist and Imbens (1995).

All in all, the main lessons to be learned are: statistical models represent probability distributions, but they are not always able to represent the data generating process behind it. As noted by Hurwicz (1966), different data generating processes may give rise to the same probability distribution, and this is the main reason why structural models are distinguished from reduced form models. The distinction is important because some models may address questions not answered by statistical models alone, such as causal queries (PEARL, 2009). Furthermore, OLS regression is NOT a technique for direct estimation of structural models. It is designed for conditional expectation (or distribution) estimation, with reasoning resting on the CEF decomposition property (1.22), which is valid for any random variable with well defined expectations. By construction, residuals in OLS regressions are orthogonal to regressors, mimicking the correspondent property of residuals in CEF decomposition. Structural models like (1.19) does NOT have the properties of CEF decomposition, so there is no reason to expect that OLS regression is a well suited technique for their estimation. Results will be obviously "biased", since OLS is estimating another thing, namely, the CEF, as illustrated before. Of course you may redefine regression changing its estimators to accommodate different

[9] Colliders are variables with two or more arrows pointing into it. Their role in causal analysis relates to the very important notion of d-separation (PEARL, 2009).

[10] If a causal effect in a directed acyclic graph (DAG) is not identifiable by do-calculus rules, than it is not identifiable at all.

residuals properties. But if your structural model is causal, we think is much clearer to inspect it with causal models designed for this task.

All above is somewhat well known, but we think it is overall cloudy when there is not a language to distinguish causal reasoning from statistical one, and also a rigorous definition of causal effects and causal models. These are the reasons to rely on Structural Causal Models, reviewed in next section.

1.4 Structural Causal Models

The Structural Causal Models (SCM) approach detailed in Pearl (2009) is under development since the 1980's, having been born from Bayesian networks (LAURITZEN, 1982; WERMUTH; LAURITZEN, 1982; KIIVERI; SPEED; CARLIN, 1984; PEARL, 1985), and draws its inspiration mainly from Haavelmo's concept of causal intervention (PEARL, 2015). This approach rests on a philosophic viewpoint close to a Laplacian quasi-deterministic concept of causality (PEARL, 2009), where variables in a system relates to each other through *deterministic* equations with stochastic inputs.

SCMs are the workhorse of this thesis and are designed to address causal questions, especially those related with manipulation of variables. We will briefly describe them here and also make some conceptual considerations relative to notions in statistics and econometrics which are somewhat controversial.

Formally, SCMs are defined as:

Definition 1 (Pearl (2009))**.** *A structural causal model is a triple $M = \langle U, V, F \rangle$ where:*

1. *U is a set of **background**, **predetermined** or **exogenous** variables, determined by factors outside the model;*
2. *V is a set $\{V_1, \ldots, V_n\}$ of **endogenous** variables, that is, they are determined by variables in $U \cup V$;*
3. *F is a set of functions $\{f_1, \ldots, f_n\}$ such that each f_i is a mapping from its domain in $U_i \cup PA_i$ to V_i, where $U_i \subseteq U$ and $PA_i \subseteq V \setminus V_i$ and the entire set F forms a mapping from U to V.*

Since F is a mapping from U to V, for each instantiation u of U the system F has a unique solution $V(u)$. Every causal model M has an associated graph $G(M)$ in which each node corresponds to a variable and the directed edges point from members of PA_i and U_i toward V_i. If $G(M)$ is acyclic, then M is acyclic and uniqueness of $V(u)$ is guaranteed. Halpern (2000) also extends axiomatization of causal models to "arbitrary theories", where equations may have multiple solutions or no solutions at all. For our purposes, the acyclic case is sufficient.

We are interested in statistical analysis of causal phenomena, so a probabilistic extension of Definition 1 is necessary. A probabilistic causal model is a pair $\langle M, P(u) \rangle$ where M is a causal model and $P(u)$ is a probability function defined over U. The operation of intervention on a variable X_i is denoted $do(X_i = x)$, and is done removing the equations $x_i = f_i(pa_i, u_i)$ and substituting all instances of X_i in the model by x. This leads to the concept of causal effect:

Definition 2 (Pearl (2009))**.** *Given two disjoint sets X and Y, the causal effect of X on Y, denoted as $P(y|do(X = x))$, $P(y|do(x))$ or, more compactly, $P(y|\hat{x})$, is a function from X to the space of probability distributions on Y. For each realization x of X, $P(y|\hat{x})$ gives the probability (or density) of $Y = y$ induced by deleting from the model $x_i = f_i(pa_i, u_i)$ all equations corresponding to variables in X and substituting $X = x$ in the remaining equations.*[11]

[11] Henceforth we will denote the intervention $do(X = x)$ also as $\hat{x}$ or $do(x)$ without distinction, only for notational convenience.

The potential response of Y in model $M = \langle U, V, F \rangle$ to action $do(X = x)$ in situation $U = u$, denoted $Y^x(u)$, is the solution for Y of the system of equations $F_x = \{f_i : V_i \notin X\} \cup \{X = x\}$ where $f_i \in F$ and $U = u$ (PEARL, 2009). This is just a formal statement of the equation substitution mechanism with fixed background $U = u$, but helps to define the important concept of counterfactual:

Definition 3 (Pearl (2009)). *Let X and Y be subsets of V in causal model $M = \langle U, V, F \rangle$. The* ***counterfactual sentence*** *"Y would be y in situation $U = u$, had X been x" is interpreted as $Y^x(u) = y$, the potential response of Y to $X = x$ in situation u.*

Letting u vary according to $P(u)$, counterfactuals may be seen as random variables Y^x, and a simple direct relation among counterfactuals and the "do" operator (PEARL; GLYMOUR; JEWELL, 2016) is

$$P(Y^x = y) = P(Y = y|do(X = x)). \tag{1.28}$$

Besides their close relation, counterfactuals and do-expressions are not the same. Counterfactuals express relations among variables that evolves in *different histories*, and do-expressions represent interventions in probability distributions of the system. This issue and the greater flexibility of counterfactuals are discussed in Pearl, Glymour and Jewell (2016, p. 98–101).

Another important property of causal models is the directionality of causality. As observed by Pearl (2009, p. 160–163), if the equation

$$y = \beta x + \epsilon \tag{1.29}$$

is regarded as structural, so the change in $E(Y)$ resulting from a unit change in X is β. If we rewrite (1.29) as

$$x = \frac{y - \epsilon}{\beta} \tag{1.30}$$

what is the interpretation of $1/\beta$? According to Pearl (2009), some authors simply deny any causal interpretation to β, concentrating on its statistical interpretation. In contrast, the econometrics standard response is that the error term in (1.30) is correlated with y, since it is not correlated with x by hypothesis in equation (1.29). But, as is well known, any pair of bivariate normal variables can be written as

$$\begin{aligned} y &= \beta x + \epsilon_y \\ x &= \alpha y + \epsilon_x \end{aligned} \tag{1.31}$$

with $Cov(X, \epsilon_x) = Cov(Y, \epsilon_y) = 0$. So, the condition $Cov(X, \epsilon_x) = 0$ does not guarantee that a variation of Y by one unit *cause* a variation of α in $E(X)$, since Equations (1.31) are not necessarily structural (causal). Notice that, observationally, they are true, but what if Y is manipulated? To think clearly about this, a simple example is helpful. Consider the following system:

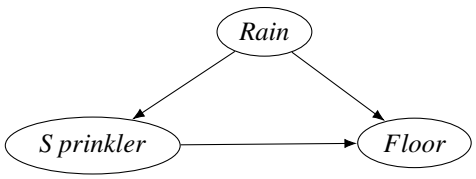

Figure 3 – DAG representing the sprinkler system described on text.

This model has only 3 variables: one tell if it is raining, another if the sprinkler is on or off and the last one represents the state of the floor (wet or dry). We suppose that the sprinkler is automatic, and turns on if weather is dry enough. The arrows represent causal relations. In the $sprinkler \rightarrow floor$ link, turn the sprinkler on wets the floor, but not the other way around. So, there is an intrinsic *asymmetry* in causal relations which is somewhat masked by the equal sign in algebraic equations, but is made clear by the arrow direction in causal graphs. One way to tackle these inconsistencies is through an *operational definition*[12] of structural equations

[12] An operational definition is a procedural way to define something, that is, a procedure to compute what is being defined.

(PEARL, 2009):

Definition 4 (Pearl (2009))**.** *An equation like* (1.29) *is said to be* ***structural*** *if in an ideal experiment where we control for $X = x$ and any other set of variables $Z = z$, Z not containing X and Y, then, the value of Y is given by $y = \beta x + \epsilon$, where ϵ is not a function of the settings x and z.*

Consequently, an operational definition for structural parameters like β in equation (1.29) is simply (PEARL, 2009):

$$\beta = \frac{\partial E[y|do(x)]}{\partial x}. \tag{1.32}$$

These considerations also provide an operational definition for the controversial structural error term (PEARL, 2009):

$$\epsilon = y - E[y|do(x)]. \tag{1.33}$$

This gives a definition to error terms that prescribes how they can be measured, avoiding worrying about how they originate. This contrasts with the widespread econometrics point of view.

One point about Definition 4 is that it prescribes how measures must be made to observe structural parameters of interest, but it does not mean that these measurements are possible. Control for $X = x$ may be impossible in practice. But, that is the point of causal analysis. Even if this hypothetical experiment is impossible, there are tools to transform equation (1.32), in some circumstances, into expressions free of do-operator, that is, expressions representing pure statistical models that may be fitted.

All in all, a causal model is a set of non-parametric structural equations representing autonomous mechanisms of variables determination that can also be represented (in the acyclic case) by a DAG (directed acyclic graph). An intervention in X is done as substitutions of some functions in the model, or as removing all parents of X in the corresponding DAG (PEARL, 2009). Indeed, the DAG is more than a simple representation. Frequently operations are easily done in DAGs than equations. Some of these operations may be used to identify causal effects using only observational data. DAGs also make the asymmetric nature of causality clear.

Counterfactuals are the response of variables of the model to interventions in a given situation. The situation, represented by $U = u$, is interpreted as an individual or experimental unit in Neyman-Rubin approach[13], which is a particular case of Definition 3.

Another related concept, which pervades econometrics and traces its origins to the Cowles Commission for Research in Economics (CHRIST, 1994), is that of exogeneity (KOOPMANS, 2005; ENGLE; HENDRY; RICHARD, 1983; LEAMER, 1985). Pearl (2009) contextualizes it and the related concepts of weak, strong and superexogeneity into the framework of SCMs together with instrumental variables and counterfactuals, showing the power of SCMs as a language for the analysis of causality in general.

Pearl (2009) gives some compelling reasons for the use of causal graphs and structural equations, at least as a common language for researchers on causality. We wish to highlight three main points in favor of his view: first, SCMs are non parametric, and so more general, a desirable property for a broad language. Second, the Neyman-Rubin potential outcome framework and SEMs, which have their own merits, are embedded in SCM framework (PEARL, 2009)[14]. Third, causal thinking is more natural to human mind than conditional independence assumptions, the basis for Neyman-Rubin framework.

Corroborating this third point, Tversky and Kahneman (1980) analyze various experiments investigating judgments about conditional probabilities. In their words:

[13] In our notation we put the "experimental condition" $X = x$ in superscript instead of subscript, as is common in Neyman-Rubin framework, because we leave the subscript for time indexing in Chapter 2

[14] Neyman-Rubin is embedded through what Pearl, Glymour and Jewell (2016) called the *fundamental law of counterfactuals*. SEMs are a particular case of SCMs.

> "It is a psychological commonplace that people strive to achieve a coherent interpretation of the events that surround them, and that the organization of events by schemas of cause-effect relations serves to achieve this goal." (TVERSKY; KAHNEMAN, 1980)

In their analysis of judgments about conditional probabilities $P(X|D)$ of a target X given evidence D, Tversky and Kahneman (1980) distinguish different types of relations between X and D. If D is perceived as a cause of X, then D is a *causal datum*. Otherwise, if X is a possible cause of D, the latter is called a *diagnostic datum*. Finally, D is *indicational* if X and D have a common cause and if they are not related, D is called *incidental*. The authors present evidence in support of causal inferences having greater efficacy than diagnostic inferences. The implication is that model construction based on causal relations is more natural and less error prone than model construction based on conditional independence assumptions.

Notice that the distinctions made in Tversky and Kahneman (1980) about the role of D are non-statistical, and the conditional probability $P(X|D)$ is the same for collected data if D is causal, diagnostic, indicational or incidental. Pearl (2009) argues that much confusion in econometrics and statistics are related to an implicit assumption of $P(X|D)$ as representing a causal relationship from D to X (see, e.g., Simpson's paradox in Pearl, Glymour and Jewell (2016) or Pearl (2009)). This is false in general, but may be true in some circumstances, something that have an important role in identification of causal effects.

1.5 Macroeconomics and causal analysis

The workhorses of econometric analysis of macroeconomic policy decisions are DSGEs, SVARs and semi-structural models. Since we are advocating the use of SCMs in macroeconomic causal reasoning, at least as a powerful complement to mainstream practice, a quick comparison among SCMs and main tools in macroeconometric modeling is necessary for a clear understanding of what SCMs can answer and what they cannot. A short review of SCMs in economics literature is also presented.

SCMs are different from DSGE models. The former models direct causal mechanisms among a set of variables and the latter models the dynamics of market general equilibrium. The invariance property assumed in SCMs is that the manipulation of a variable in the system does not affect *causal relations* among other variables in the system. In turn, DSGEs assume that the substitution of a *decision mechanism* of some agent in the system by another does not affect the *decision mechanisms* of other agents[15]. This is closely related to a causal model, but not necessarily the same. Indeed, DSGEs are silent about the *tâtonnement* mechanism driving agents to market equilibrium, and this mechanism in real world may well be a chain of causal relations. This is not exactly a problem, because DSGEs are not designed to address this issue. They are designed to explore the effects of different policy *regimes* in such a way that these changes does not affect the *decision mechanisms* of other agents. If this property is true, then the model is immune to Lucas Critique (LUCAS, 1976), one of the goals of DSGEs. Nonetheless, DSGEs may also answer questions with causal content, like its impulse response functions, which are the response of the system to *exogenous* shocks on some of its reaction mechanisms. But, this response is the equilibrium dynamics, again not modeling any intermediate step. Here we will not enter the discussion about the realism or usefulness of this modeling strategy, restricting ourselves to the key different aspects of reality that models are modeling.

VARs model the covariance structure of a set of time series and only in special conditions can answer causal questions. Their structural extension, SVARs, are designed to address this issue, and indeed answers causal questions on observational data if sufficient restrictions are imposed on the system. The main disadvantages of SVARs in face of SCMs are the linearity assumption and the absence of any guidance on

[15] Notice that we are talking about the decision *mechanism*, not the decision itself. In DSGEs, agents react optimally to optimal reactions of other agents subject to market clearance and other constaints.

the adequate restrictions and choice of variables for sound causal inference. SCMs, with the help of graphs, can actually guide the researcher on which variables are relevant for the analysis and how to identify the causal effect of interest non-parametrically. Indeed, SCMs can be used to identify SVARs and even tell how to estimate its structural parameters. Simple examples of SVAR identification with the help of SCM are given in Section 1.8 and Chapter 2.

Another line of research in SVARs is known as the sign restrictions approach and "agnostic identification". Uhlig (2017) points its origin to Dwyer et al. (1998), Faust (1998), Uhlig (1998), Canova and Pina (2000) and Uhlig (2005). We think this approach is interesting, specially for its "agnostic" viewpoint, which tries to minimize the set of assumptions, and also work with somewhat qualitative premises, since sign restrictions on impulse responses is a theoretical restriction much less stringent than other approaches. But, care must be taken. Some critiques in the literature focus on the interpretation of multiple impulse responses corresponding to different identified models, but much less is said about the consequences of bad control for confounders. Here we just point out that, if some hidden confounder is not considered in the analysis, sign restriction results are unreliable, because the premise on the number of independent shocks driving the system may fail. To see this, consider a 2 variables VAR where correlations among shocks originate not only because of contemporaneous causal effects among variables modeled in the system, but also by an unobserved common cause z. In this case, the 2 variables VAR is driven by 3 shocks, namely, one for each equation and another one named z. Actually, this critique is valid for all SVAR identification procedures, and show that causal reasoning is behind any attempt of causal inference. In sign restricted SVARs this may be worse, because IRFs with "right signs" can give an unjustified confidence on the results because of its theoretical "correctness". Much more can be said about these issues, but this is not our focus here.

Semi-structural models have features of both, structural assumptions consistent with theoretical economic behavior and reduced form features which enhance their forecasting performance (see, e.g., Angelini et al. (2019)). If its structural features are causal, they can be seen as a kind of SEM, largely used in social sciences, and are linear special cases of SCMs. But, again, SCMs serve as guide to which variables are relevant for the identification of the causal effect of interest, avoiding unnecessary modeling of various aspects of the economic system, something necessary to a large extent in semi-structural models and absolutely necessary in DSGE models. This wide system modeling strategy is too much error prone and depends on lots of simplifying assumptions to make problems tractable, which can destroy model reliability. Also, system wide modeling is complex and difficult, consuming unnecessary resources if the question can be answered by simpler causal models.

So, in our view, the main attractiveness of SCMs are: possibility of non-parametric identification of causal effects, guidance on which variables are relevant for the identification of causal effects and the possibility to identify the effects of interest without the necessity of explicit modeling and estimation of a lot of macroeconomic mechanisms. Also, traditional macroeconomic modeling techniques does not explicit which conditional independences models must obey, and so various causal conclusions are potentially non-testable and non-falsifiable. SCMs gives *all* conditional independences obeyed by the model, making it testable on various ways, and also show clearly where it is not testable and why this is so.

But, SCMs have not been adopted in economics literature, but there are exceptions. A noticeable one is Céspedes, Lima and Maka (2008), where the authors identify SVARs with the help of DAGs. Overall, much emphasis is given to conceptual discussions defending or dismissing SCMs as useful tools in economics (WHITE; CHALAK, 2006; CHALAK; WHITE, 2011; PEARL, 2013; HECKMAN; PINTO, 2015; PEARL, 2015; CUNNINGHAM, 2018; HÜNERMUND; BAREINBOIM, 2019; IMBENS, 2019). We will not enter the debate. For us, the usefulness of the tool is obvious, but this is somewhat personal. This thesis presents applications and also highlights possible flaws in traditional econometric approaches all made clear by the SCM approach.

Others may say that different approaches could give similar results. Well, that may be true, but only time can answer what is useful for a discipline on the long run. For us, SCMs make causal identification simpler, more rigorous and transparent, explicitly showing *all* causal premises adopted by the researcher, and also almost eliminates potential distractions such as implementation and estimation issues, which are statistical but not causal. Another advantage is the possibility of easily list all conditional independences obeyed by some model, making it more falsifiable. Independent of reasons behind a supposed greater resistance in economics literature towards SCMs, we restrict ourselves on presenting results using SCMs as useful tools for economic analysis.

We also think that the causal viewpoint make the distinction between identification and estimation clearer, and opens the door for all sorts of techniques which try to infer conditional distributions from data. For example, after identifying a causal effect of interest, the researcher concludes that it is necessary to estimate an equation like (1.34) or (1.36). These equations are free of "do" operators, and can be estimated by linear regressions or more sophisticated statistical and machine learning techniques, for example. This is important, because machine learning is yet viewed as just a predictive tool by economists (and even by data scientists in general) (MULLAINATHAN; SPIESS, 2017), but after the identification of which probabilistic expression represents the causal effect of interest, the estimation of this expression is very similar to a prediction problem.

1.6 Causal effects identification and do-calculus

Here we briefly review some basic SCM concepts used in this thesis. A causal effect is *identifiable* if it can be estimated from observational data. In the causal language introduced before, the causal effect $P(Y|do(X = x))$ is identifiable if this expression can be transformed in some way into an expression free of the "do" operator. From now on we adopt the following notation: the summation symbol $\sum$ represents all sorts of integration, be that a sum of discrete values or integration of continuous variables, somewhat like a Riemann–Stieltjes integral and its generalizations (SHILOV; GUREVICH, 2013).

Definition 5 (Pearl (2009))**.** *The causal effect of X on Y is **identifiable** from a graph G if the quantity $P(y|do(x))$ can be computed uniquely from any positive probability of the observed variables.*

A very useful sufficient criteria for causal effects identification in semi-Markovian models is the back-door criterion. More details in Pearl (2009) and Morgan and Winship (2015):

Definition 6 (Pearl (2009))**.** *A set of variables Z satisfies the **back-door criterion** relative to an ordered pair of variables (X_i, X_j) in a DAG G if:*

- *no node in Z is a descendant of X_i and*
- *Z blocks[16] every path between X_i and X_j that contains an arrow into X_i.*

For disjoint sets of variables X and Y in G, Z satisfies the back-door criterion relative to (X, Y) if it satisfies the criterion for any pair (X_i, Y_i) with $X_i \in X$ and $Y_i \in Y$.

Theorem 1 (**Back-door adjustment**, Pearl (2009))**.** *If a set Z satisfies the back-door criterion relative to (X, Y), then the causal effect of X on Y is identifiable and given by the adjustment for Z formula:*

$$P(y|\hat{x}) = \sum_z P(y|x, z)P(z). \tag{1.34}$$

This simple criterion is sufficient for almost all our purposes. Another concept that we will also use is the identification of plans (PEARL; ROBINS, 1995):

[16] Block in the sense of d-separation. Details in Pearl (2009).

Definition 7 (Pearl (2009)). *A **plan** is an ordered sequence $(\hat{x}_1, \ldots, \hat{x}_n)$ of value assignments to control variables $(X_1, \ldots, X_n)$.*

Below we use the notation $G_{\underline{X},\overline{Y}}$ to represent the graph G after removing all arrows emerging from nodes in X and also with the arrows pointing to nodes in Y removed. The following theorem gives a sufficient condition for plan identifiability.

Theorem 2 (Pearl (2009)). *The probability $P(y|\hat{x}_1, \ldots, \hat{x}_n)$ is identifiable if, for every $1 \leq k \leq n$, there exists a set Z_k of variables satisfying the following **sequential back-door** conditions:*

- *Z_k consists of non-descendants of $\{X_k, X_{k+1}, \ldots, X_n\}$ and*
- *$(Y \perp\!\!\!\perp X_k | X_1, \ldots, X_{k-1}, Z_1, \ldots, Z_k)_{G_{\underline{X}_k, \overline{X}_{k+1}, \ldots, \overline{X}_n}}$.*

If $P(y|\hat{x}_1, \ldots, \hat{x}_n)$ is identifiable by Theorem 2 then it is called *G-identifiable*. The following theorem (PEARL; ROBINS, 1995; ROBINS, 1997; GILL; ROBINS, 2001; PEARL, 2009) closes the formal results in causal models necessary for this thesis:

Theorem 3 (Pearl (2009)). *The probability $P(y|\hat{x}_1, \ldots, \hat{x}_n)$ is G-identifiable if and only if the following condition holds for every $1 \leq k \leq n$:*

$$(Y \perp\!\!\!\perp X_k | X_1, \ldots, X_{k-1}, W_1, \ldots, W_k)_{G_{\underline{X}_k, \overline{X}_{k+1}, \ldots, \overline{X}_n}} \tag{1.35}$$

where W_k is the set of all covariates in G that are both non-descendants of $\{X_k, X_{k+1}, \ldots, X_n\}$ and have either Y or X_k as descendants in $G_{\underline{X}_k, \overline{X}_{k+1}, \ldots, \overline{X}_n}$. Moreover, if this condition is satisfied, the plan evaluates as

$$P(y|\hat{x}_1, \ldots, \hat{x}_n) = \sum_{w_1, \ldots, w_n} \left[P(y|w_1, \ldots, w_n, x_1, \ldots, x_n) \times \prod_{k=1}^{n} P(w_k|w_1, \ldots, w_{k-1}, x_1, \ldots, x_{k-1}) \right]. \tag{1.36}$$

Our approach targets macroeconomic time series, and the causal framework above is not explicitly designed for this task. But, with some adaptations, causal analysis of time series is also possible. A paper that addresses time series inference in structural causal models context is Blondel, Arias and Gavaldà (2017). In this work, the authors generalize applications of do-calculus to what they defined as a dynamic causal network, constructed over dynamic Bayesian networks developed in the 1990s (DAGUM; GALPER; HORVITZ, 1992; DAGUM et al., 1995; MURPHY; RUSSELL, 2002; RUSSELL; NORVIG, 2016). We begin with Blondel, Arias and Gavaldà (2017) definition of dynamic causal networks:

Definition 8 (Blondel, Arias and Gavaldà (2017)). *Given random processes V_k and U_k, a **dynamic causal network** is a causal model such that $v_{k,t} = f_k(pa(V_{k,t}), u_{k,t-\alpha})$ is the time t realization of the random process V_k, that is, a realization of $V_{k,t}$. The variable $u_{k,t-\alpha}$ is the time $t - \alpha$ realization of the process U_k and $pa(V_{k,t})$ are the parents of $V_{k,t}$ in the causal model.*

A dynamic causal network may be represented as an infinite DAG. Consider the *dynamic causal model*, also called *dynamic causal system*, given by equations (1.37):

$$\begin{aligned} c_t &= 0.4c_{t-1} + 0.3c_{t-2} + 0.2y_{t-1} + \epsilon_t^c \\ x_t &= 0.4z_t - 0.3y_{t-1} + \epsilon_t^x \\ y_t &= 0.5z_t + 0.5x_t + \epsilon_t^y \\ z_t &= 0.6z_{t-1} + 0.5c_t + \epsilon_t^z \end{aligned} \tag{1.37}$$

where $\epsilon_t^k \sim N(0,1)$ for all $k \in \{c, x, y, z\}$ are serially independent and also jointly independent. In a cross sectional context, the contemporaneous model may be represented by Figure 4.

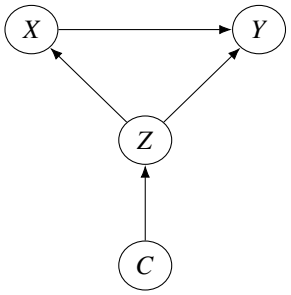

Figure 4 – DAG representing contemporaneous effects in equations (1.37).

If we could, for example, sample from the joint probability distribution of X, Y, Z and C, we should infer the causal effect of X on Y adjusting for Z, following the back-door criterion. But, this approach implicitly supposes independent random samples from $P(X, Y, Z, C)$. If these variables evolve over time as dictated by (1.37), this is not true. Observe that the sample (x_t, y_t, z_t, c_t) depends on $(x_{t-1}, y_{t-1}, z_{t-1}, c_{t-1})$ in this example. Of course, this serial dependence is the main feature of time series analysis in general. For a graphical representation of the dynamic relationships in (1.37), see Figure 5.

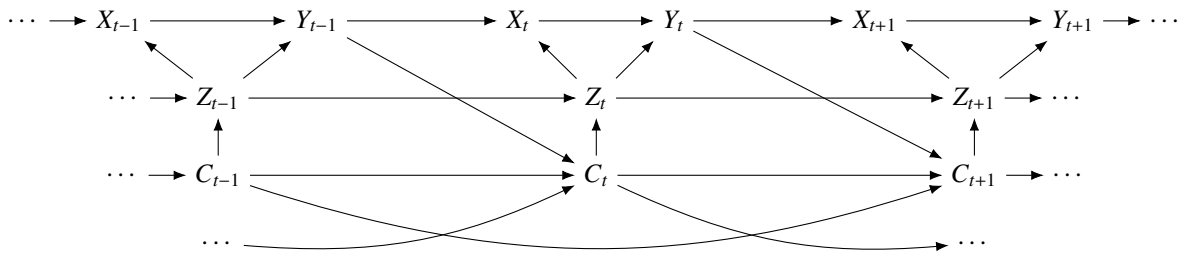

Figure 5 – Dynamic causal network representing the dynamic model in (1.37).

Looking at this figure, a critical question may be raised: What is an intervention in a time series model? By definition of stochastic process, for each t, (X_t, Y_t, Z_t, C_t) is a random variable. If we know their joint distribution (or just some suitable conditional ones) for each time sample t, some causal effects may be calculated. Blondel, Arias and Gavaldà (2017) define their intervention in a dynamic context supposing a known dynamics through Markov chains with finite state space. But, normally this dynamics is unknown and some variables may be unobserved. Also, we may be interested on some sort of "average effect" of an intervention, regardless of exactly in which moment t this intervention takes place. This can give us information about the structure of the data generating process, and is called in the literature *impulse response analysis* of the system. For time invariant linear systems this "average effect" is unambiguous since in this class of systems the impulse responses are history and shock independent (KOOP; PESARAN; POTTER, 1996). For non-linear systems this is not necessarily the case. In following sections we analyze the concept of impulse response function (IRF), including difficulties raised by non-linear systems and their relation to causal intervention on time series.

Another important relation for us is equation (1.28), relating probabilistic counterfactuals and do-expressions. But the theory goes much further than this. For example, there are three rules, named rules of do-calculus, that can be used to try to eliminate the "do" operator from causal expressions. The do-calculus is proven to be complete in acyclic models (PEARL, 1995; SHPITSER; PEARL, 2006; HUANG; VALTORTA, 2012), that is, if a causal effect is identifiable, there is a sequence of steps in do-calculus that reaches the expression for it. Conversely, if it is impossible using do-calculus to identify a causal effect, then this effect is non-identifiable. Also, these deductions may be done by a sound and complete algorithm, that is, an algorithm that finds the correct causal effect when it terminates and, if it fails to find a causal effect, this is non-identifiable (SHPITSER; PEARL, 2006). This is a remarkable result. In fact, the problem of identification of causal effects

in non-parametric recursive models is mathematically a solved problem.[17] But, as noted, this is valid for non-parametric models. With parametric assumptions some causal effects may be identified even if they are not identifiable according with do-calculus rules. This is because parametric assumptions impose restrictions on the model. The canonical example is the instrumental variables approach analyzed before.

1.7 Impulse responses

Generally speaking, the impulse response function is, according to Koop, Pesaran and Potter (1996), the outcome of a *conceptual experiment*. As pointed out by Jordà (2005), IRFs are the difference between two *forecasts*. But, as discussed before, these two visions are not necessarily the same, since the former makes reference to an intervention and the latter is observational. But, interestingly, Jordà (2005) cites the definition given by Koop, Pesaran and Potter (1996). This apparent contradiction is probably a consequence of the confusion exposed by Pearl in statistics and econometrics between correlational and causal concepts, with roots in the absence of a formal definition and notation for causality. Econometricians avoid this assuming known data generating processes, a much restrictive assumption. Indeed, despite an apparent contradiction among parlance of these authors, their formal definitions are not contradictory. Koop, Pesaran and Potter (1996) uses Definition 9 for the traditional IRF:

Definition 9 (Koop, Pesaran and Potter (1996))**.** *For a Markov multivariate process of order p with additive shocks:*

$$Y_t = F(Y_{t-1}, \ldots, Y_{t-p}) + H_t V_t, \tag{1.38}$$

*the **traditional impulse response function** is given, for $h = 1, 2, 3, \ldots$, by*

$$I_Y(h, \delta, \omega_{t-1}) = E(Y_{t+h}|V_t = \delta, V_{t+1} = 0, \ldots, V_{t+h} = 0, \omega_{t-1}) - E(Y_{t+h}|V_t = 0, V_{t+1} = 0, \ldots, V_{t+h} = 0, \omega_{t-1}), \tag{1.39}$$

where V_t is a shock in the system at time t and ω_{t-1} is the realization of the variables up to $t-1$ used to forecast Y_{t+h}. In this case, $\omega_{t-1} = \{y_{t-1}, \ldots, y_{t-p}\}$.

Jordà (2005) defines impulse responses in a similar way, but ignores zero future shocks, which is not important for his analysis. But both and virtually all the literature in econometrics concentrates on shocks over a system of equations.

Besides the lack of explicit causal content, Koop, Pesaran and Potter (1996) ideas on generalized impulse responses fit nicely to our proposal based on causal interventions. The authors define the generalized impulse response of Y at horizon h for an arbitrary current shock v_t and history ω_{t-1} as

$$GI_Y(h, v_t, \omega_{t-1}) = E[Y_{t+h}|v_t, \omega_{t-1}] - E[Y_{t+h}|\omega_{t-1}]. \tag{1.40}$$

Their idea is to see this impulse response, which is history and shock dependent, as a realization of the random variable

$$GI_Y(h, V_t, \Omega_{t-1}) = E[Y_{t+h}|V_t, \Omega_{t-1}] - E[Y_{t+h}|\Omega_{t-1}]. \tag{1.41}$$

This is interesting, since we may condition on a particular history or shock, take expectations, etc. But, conditioning on Ω_{t-1} or V_t does not ensure a causal interpretation to this impulse response. Moreover, even if (1.41) is defined on a data generating process (DGP) with clear causal content, we still have an issue on the interpretation of the shock V_t, something problematic in non additive cases, an issue explained in Chapter 2. So, we choose definitions of impulse responses which have explicit causal content. For this task, the concept of counterfactuals presented in Section 1.4 will be very useful.

[17] Recursive causal model are the ones that induce DAGs. A non-recursive model may have cycles and induce equilibrium relationships among some variables.

In Chapter 2 we regard an impulse response as a comparison between two hypothetical regimes: one where some intervention is made compared with another one where a different intervention, or no intervention at all, is made. Notice that we emphasize the interventional nature of impulse response analysis. In our opinion, the main value (and intention) of IRFs is to answer *counterfactual questions*. Indeed, a system formed by incidence of forest fires and ice cream sales may have a good forecasting performance, but is meaningless to ask for the impulse response of forest fires to ice cream sales as defined in Koop, Pesaran and Potter (1996) or Jordà (2005). Of course, for these authors, the system of equations where they define impulse responses are implicitly seem as the data generating process, or at least the history ω_{t-1} contains all relevant information for causal conclusions to be made, such as air temperature in the forest and ice cream example, much like as in Granger-causality.

Coming back to Definition 9, function F is known, so what is the interpretation of equation (1.38)? Is it simply a *description* of the process, that is, a system that generates samples of the process Y_t like a reduced form model or pure statistical model? Or is it *structural* in the sense of Section 1.4? The authors does not answer this question directly, but later in the paper we may infer that this model is probably *not* structural, since the authors admit that shocks V_t may be correlated. Actually, they restate the same reasoning that motivates structural VARs (AMISANO; GIANNINI, 2012): if the shocks are correlated, it is meaningless to infer the response from a perturbation in $V_{1,t}$ keeping $V_{2,t}$ fixed, where $V_t = (V_{1,t}, \ldots, V_{k,t})$. However, they argue that this is a weakness of traditional IRFs, instead of lack of causal identification, and call it *composition effect*.

In our opinion, the weaknesses of traditional IRFs are two: their linearity dependence to make meaningful inferences and its correlational nature. Questions regarding integration of future shocks or setting them to zero are related to plan identification and "which question the researcher want to answer", and we address them in Chapter 2. Here we argue that questions regarding causal identification must be the core of IRFs interpretation.

1.8 Falsifiability in macroeconomics

To end this chapter we review a basic concept in science which is sometimes blurred by some modeling approaches. In his seminal work entitled *The Logic of Scientific Discovery* (POPPER, 2005), the philosopher Karl Popper argues that the best *criterion of demarcation*, the division between what should be considered a scientific theory and what should not, is a property he coined *falsifiability*. Also known as *refutability*, this term means the *possibility* of a hypothesis or theory to be falsified by evidence.

So, the statement "all men are immortal" is falsifiable, since the death of one man, something observable in principle, may refute the allegation. In contrast, the statement "all men have an immortal soul which is inaccessible by physical means" is not falsifiable by construction, so cannot be scientific by Popper's criterion of demarcation. Notice that nothing is said about the veracity of the expression itself. It may be true or wrong but not falsifiable. However, if it is falsifiable, it can prove wrong in principle, even if it is true. For example, the statement "no man can live more than 200 years" is falsifiable, because the observation of one man living more than 200 years makes the statement false. The fact that a man living more than 200 years was never observed is a corroborating evidence for the truth of that statement, but not a proof of its truth. It is not possible to prove the truth of scientific allegations, but merely collect evidence in favor of or disprove it completely.

This celebrated criterion revolutionized the way philosophers and scientists see scientific method. It is not perfect, and there are critics and extensions. Kuhn (1962), in his *The Structure of Scientific Revolutions*, deals with scientific discovery on a historical perspective. He argues that scientists faces conceptual paradigms which influences the way they interpret data. Normal scientific progress goes by a process of accumulation, and the paradigms that sustains research programs are even protected against falsification by means of *ad hoc* hypothesis for explaining anomalies found. Scientific revolutions occurs with paradigm shifts. Of course

that for a revolution to occur, a sufficient number of anomalies must accumulate, and this is only possible if theories under a specific paradigm are falsifiable.

Imre Lakatos accommodates Popper and Kuhn views, arguing that scientists work on research programs, which resembles Kuhn's conceptual paradigms (LAKATOS, 1976; LAKATOS; WORRALL; CURRIE, 1982). For the author, falsification acts over those programs and not necessarily on every statement, giving margin for *ad hoc* explanations that Popper rejected. Lakatos gives science more flexibility and, somewhat paradoxically, more conservatism. Backhouse (2006) presents essays about methodology in economics viewed through the lens of philosophy of science.

Some controversial statements concerning falsifiability are those dealing with probability. Unfortunately, these are exactly the ones faced by econometrics. Popper points out that probability statements are not falsifiable. In fact, if meteorological services announce the probability of rain tomorrow as 60%, this cannot be falsified, whatever it rains or not. However, the model that computes these probabilities is statistically falsifiable, simply comparing its historical records with data and proceeding on standard statistical analysis. Gillies (2012) gives a falsifying rule for probability statements in line with statistical tests on practice. Quinn and Keough (2002) also analyses the rule of Popperian falsification in statistics.

For the purposes of this thesis, the main point concerns falsifiability, in statistical terms or not, of identification hypothesis embedded in econometric models developed for policy analysis. Consider an identification problem in econometrics unsolvable *in principle* without further assumptions on the DGP: the structural vector autoregression (SVAR). One example is:

$$Ay_t = \sum_{i=1}^{p} C_i y_{t-i} + B\epsilon_t \tag{1.42}$$

with y_t and ϵ_t $N \times 1$ vectors and A and C_i $N \times N$ matrices. Also, $\epsilon_t \sim N(0, I_N)$, where I_N is the $N \times N$ identity matrix. This is a SVAR model called by Lütkepohl (2005) an AB-model. If A is invertible, you can put it on the reduced form VAR model:

$$\begin{aligned} y_t &= \sum_{i=1}^{p} A^{-1} C_i y_{t-i} + u_t \\ u_t &= A^{-1} B\epsilon_t. \end{aligned} \tag{1.43}$$

Lets concentrate on the statistical description of u_t. Set $M = A^{-1}B$. Since $\epsilon_t \sim N(0, I_N)$, we have $u_t \sim N(0, \Sigma_u)$, where $\Sigma_u = MM'$. As is well known, for every orthogonal matrix Q, $MM' = MQQ'M' = MQ(MQ)'$ such that there are infinite matrices MQ that generate the same covariance matrix for u_t (LÜTKEPOHL, 2005; UHLIG, 2005; ARIAS; RUBIO-RAMÍREZ; WAGGONER, 2018). So, for fixed A and B, the statistical description of variables generated by (1.42) is insufficient to make inferences about A and B without further restrictions. In other words, the model in non-identified.

There are various strategies to identify A and B in (1.42), ranging from zeros imposed on A to linear or long-run restrictions. Zero restrictions have a non-parametric flavor and are directly related to causal models, where the only restrictions imposed are the presence or absence of causal links, their direction and possibly unobserved confounding. More "functional" restrictions like linear or long-run make models richer, but also raise the number of assumptions, something we think is undesirable in models of complex phenomena such as macroeconomics.

Here our concern is falsifiability. If A is just-identified, no test on restrictions is possible (LÜTKEPOHL, 2005). If the model is overidentified, so test on restrictions is possible through a LR test (LÜTKEPOHL, 2005). This is good, because makes restrictions falsifiable. But, Lütkepohl (2005, p. 359) says that in SVAR modeling it is common to rely on non-falsifiable just-identified models. We disagree with this habit. Restrictions are imposed outside the estimation procedure. Theory and other studies must guide restrictions imposed on

model structure. If these restrictions are not sufficient for model identification, the model is non-identifiable, and additional information must be collected. Restrictions imposed *ad hoc* to achieve just-identification is not good science, since this is always possible and non-falsifiable. Restrictions entailing overidentified models are falsifiable, and so preferable.

LR test tests the whole covariance matrix. Additionally, we may test conditional independences required by causal assumptions. In just-identified recursive models, conditional independences entailed by causal models are not useful either, because these models does not give testable conditions. But overidentified and some non-identifiable models may have some of its assumptions tested. Consider equation (1.42) with $p = 1$ and parameters $A = \begin{pmatrix} 1 & -0.2 & 0 & 0 \\ 0.3 & 1 & 0 & 0 \\ 0.2 & 0.2 & 1 & 0 \\ 0 & 0.1 & 0.1 & 1 \end{pmatrix}$, $B = \begin{pmatrix} 1 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 \\ 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 1 \end{pmatrix}$ and $C_1 = \begin{pmatrix} 0.7 & 0 & 0 & -0.1 \\ 0 & 0.3 & 0 & 0.1 \\ 0 & -0.3 & 0.1 & 0 \\ 0.2 & 0 & 0 & 0.4 \end{pmatrix}$ as a causal model with the following interpretation in terms of SCMs:

$$\begin{aligned} y_{1,t} &= 0.2y_{2,t} + 0.7y_{1,t-1} - 0.1y_{4,t-1} + \epsilon_{1,t} \\ y_{2,t} &= -0.3y_{1,t} + 0.3y_{2,t-1} + 0.1y_{4,t-1} + \epsilon_{2,t} \\ y_{3,t} &= -0.2y_{1,t} - 0.2y_{2,t} - 0.3y_{2,t-1} + 0.1y_{3,t-1} + \epsilon_{3,t} \\ y_{4,t} &= -0.1y_{2,t} - 0.1y_{3,t} + 0.2y_{1,t-1} + 0.4y_{4,t-1} + \epsilon_{4,t} \end{aligned} \tag{1.44}$$

A DAG representing qualitative causal knowledge about equations (1.44) at time slices $t-2$, $t-1$ and t is shown in Figure 6. Double headed dashed arrows represent confounding due to variables before $t-2$.

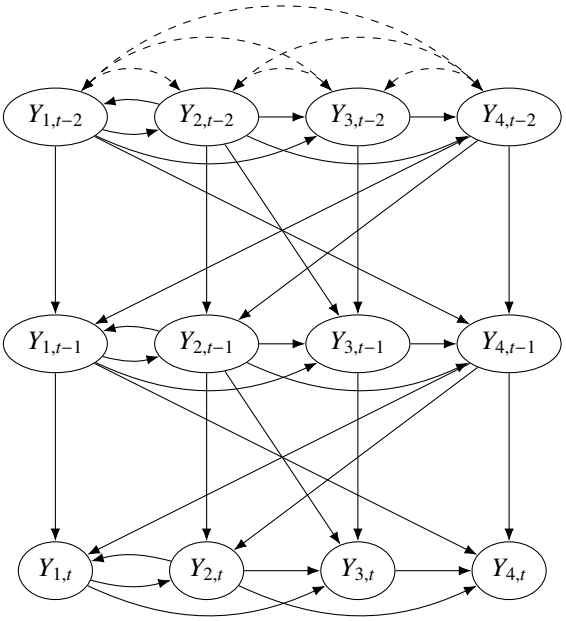

Figure 6 – Dynamic causal network representing the dynamic model in (1.44).

This DAG entails the premises of non correlated residuals and shows causal links among variables. In SVAR analysis, the arrows between time slices $t-1$ and t are normally assumed fully connected, supposing no knowledge about causal links among different time slices. This is a philosophy of "let the data speak for itself", minimizing assumptions in models to achieve identification of economic shocks, in the spirit of Sims' original proposal (SIMS, 1980; CHRISTIANO, 2012). We welcome this philosophy, but almost always data cannot speak about causal relations, and a model is necessary, even in VARs. For SVAR identification, some

premises are necessary, and the choice of variables is also important in controlling confounding, something frequently neglected. Also, with a qualitative model, some restrictions are testable.

Anyway, system (1.44) is regarded as *non identifiable*. If you try the set of restrictions[18] $A = \begin{pmatrix} 1 & * & 0 & 0 \\ * & 1 & 0 & 0 \\ * & * & 1 & 0 \\ 0 & * & * & 1 \end{pmatrix}$ and $B = \begin{pmatrix} * & 0 & 0 & 0 \\ 0 & * & 0 & 0 \\ 0 & 0 & * & 0 \\ 0 & 0 & 0 & * \end{pmatrix}$ in an econometrics package, an error or non identifiability message is thrown[19].

But, before giving up or trying to force an identification looking for "reasonable" answers, a dangerous practice according to Fernandez-Villaverde and Rubio-Ramírez (2010)[20], it is possible to test some assumptions on this model with the help of causal graphs. Conditional independences in Figure 6 for variables at time slices $t-1$ and t, obtained with the help of DAGitty (TEXTOR; van der Zander, 2016), are listed below:

I.1 $y_{1,t} \perp\!\!\!\perp y_{4,t} | y_{2,t}, y_{3,t}, y_{1,t-1}, y_{4,t-1}$

I.2 $y_{1,t} \perp\!\!\!\perp y_{2,t-1} | y_{1,t-1}, y_{4,t-1}$

I.3 $y_{1,t} \perp\!\!\!\perp y_{3,t-1} | y_{1,t-1}, y_{4,t-1}$

I.4 $y_{2,t} \perp\!\!\!\perp y_{1,t-1} | y_{2,t-1}, y_{3,t-1}, y_{1,t-2}, y_{4,t-2}$

I.5 $y_{2,t} \perp\!\!\!\perp y_{1,t-1} | y_{2,t-1}, y_{4,t-1}$

I.6 $y_{2,t} \perp\!\!\!\perp y_{3,t-1} | y_{2,t-1}, y_{4,t-1}$

I.7 $y_{3,t} \perp\!\!\!\perp y_{1,t-1} | y_{1,t}, y_{2,t}, y_{2,t-1}, y_{3,t-1}$

I.8 $y_{3,t} \perp\!\!\!\perp y_{4,t-1} | y_{1,t}, y_{2,t}, y_{2,t-1}, y_{3,t-1}$

I.9 $y_{4,t} \perp\!\!\!\perp y_{2,t-1} | y_{2,t}, y_{3,t}, y_{1,t-1}, y_{4,t-1}$

I.10 $y_{4,t} \perp\!\!\!\perp y_{3,t-1} | y_{2,t}, y_{3,t}, y_{1,t-1}, y_{4,t-1}$

These expressions can be tested against data. Consider a 1,000 sample size simulation of model (1.44). Expression **I.1** can be tested in this example with the regression shown in Table 1. Notice that the coefficient for $y_{4,t}$ is not significant, corroborating the absence of a direct causal link between y_1 and y_4 at t or, equivalently, the restrictions $A_{1,4} = A_{4,1} = 0$ in matrix A.

Additionally, it is possible to identify some elements of matrix A. Using the back-door criterion or looking directly into system (1.44), it is easy to see how inferences can be made for causal links estimation. For example, some causal effects between variables at t are shown in Table 2. We identified 4 elements of matrix A, but the feedback loop between $Y_{1,t}$ and $Y_{2,t}$ is not identifiable with this technique. Notice that regressions 1 to 4 in Table 2 are direct estimations of equations in (1.44), but regressions 5 and 6 are not, with its regressors given by the back-door criterion. Importantly, causal parameters inferred through regressions 5 and 6 can be compared with those in regressions 3 and 4. They must be (statistically) equal, and this is another possible source of falsifiability for model testing.

For the sake of completeness, to disentangle causal effects in the feedback loop, look at Figure 6. It is possible to devise some instrumental variables. Notice that, given $Y_{2,t-1}$ and $Y_{4,t-1}$, $Y_{1,t-1}$ is an instrumental

[18] The symbol "$*$" represents parameters free to vary.

[19] Package vars (PFAFF, 2008b; PFAFF, 2008a) in R (R Core Team, 2019) threw an error and JMulTi (LÜTKEPOHL; KRÄTZIG, 2004) threw a "not identified model" message.

[20] An opinion we completely agree.

	Dependent variable:
	$Y_{1,t}$
$Y_{4,t}$	0.015
	(0.028)
$Y_{2,t}$	-0.134^{***}
	(0.029)
$Y_{3,t}$	-0.203^{***}
	(0.026)
$Y_{1,t-1}$	0.629^{***}
	(0.025)
$Y_{4,t-1}$	-0.077^{***}
	(0.027)
Constant	0.003
	(0.028)
Observations	999
R^2	0.481
Adjusted R^2	0.478
Residual Std. Error	0.889 (df = 993)
F Statistic	184.133^{***} (df = 5; 993)
Note:	*p<0.1; **p<0.05; ***p<0.01

Table 1 – Test for conditional independence **I.1** implied by model (1.44).

variable for the effect $Y_{1,t} \to Y_{2,t}$. This is called a *conditional instrument* (BRITO; PEARL, 2002; BRITO; PEARL, 2012; CHEN; PEARL, 2014). Conditional instruments can be found on graphs with the help of specialized software.

We could follow the reasoning used for equations (1.27). But a better and direct approach here is to follow econometrics practice. The goal is to identify the coefficient of $Y_{1,t}$ in the second equation of system (1.44). Looking carefully for the instrumental variable definition in Wooldridge (2010, p. 89–90), we see that it is not exactly the same as the one in (BRITO; PEARL, 2002). The former looks like a conditional instrument, because the instrument must be *partially* correlated with the endogenous variable given the exogenous ones. So, in our case, using $Y_{1,t-1}$ as an instrument for $Y_{1,t}$ is sufficient to identify the effect $Y_{1,t} \to Y_{2,t}$. The reasoning is the same for $Y_{2,t} \to Y_{1,t}$. Results are shown in Tables 3 and 4. With this, all elements of matrix A were identified. Since matrix B is diagonal, it has not a direct causal interest, but is easily identified through an analysis of residuals and some sign restrictions. A point about falsifiability here: there are more instruments available in this system which can be used as overidentifying restrictions for the sake of falsifiability.

1.9 Conclusion

In this chapter we reviewed structural causal models and the concept of causality in econometrics and statistics. We also reviewed the importance of falsifiability as a scientific criterion for good models of reality and devised some potential uses of SCMs for this achievement in econometrics.

The main point is that SCMs are an adequate language for causality, being general and strictly distinguishing causal effects from correlation and associated concepts. We do not advocate SCMs as substitutes

	Causal effect	Regression	Estimate	Real value	Matrix A
1.	$Y_{2,t} \to Y_{4,t}$	$y_{4,t} \sim y_{2,t} + y_{3,t} + y_{1,t-1} + y_{4,t-1}$	-0.138^{***} (0.033)	-0.1	$A_{4,2}$
2.	$Y_{3,t} \to Y_{4,t}$	$y_{4,t} \sim y_{2,t} + y_{3,t} + y_{1,t-1} + y_{4,t-1}$	-0.138^{***} (0.029)	-0.1	$A_{4,3}$
3.	$Y_{1,t} \to Y_{3,t}$	$y_{3,t} \sim y_{1,t} + y_{2,t} + y_{2,t-1} + y_{3,t-1}$	-0.234^{***} (0.027)	-0.2	$A_{3,1}$
4.	$Y_{2,t} \to Y_{3,t}$	$y_{3,t} \sim y_{1,t} + y_{2,t} + y_{2,t-1} + y_{3,t-1}$	-0.201^{***} (0.033)	-0.2	$A_{3,2}$
5.	$Y_{1,t} \to Y_{3,t}$	$y_{3,t} \sim y_{1,t} + y_{2,t} + y_{2,t-1} + y_{4,t-1}$	-0.248^{***} (0.027)	-0.2	$A_{3,1}$
6.	$Y_{2,t} \to Y_{3,t}$	$y_{3,t} \sim y_{1,t} + y_{2,t} + y_{2,t-1} + y_{4,t-1}$	-0.197^{***} (0.033)	-0.2	$A_{3,2}$

Note: *p<0.1; **p<0.05; ***p<0.01

Table 2 – Estimation of causal parameters in model (1.44). The notation $y \sim x + z$ means the regression of y on x and z with constant term included. The parameters of interest are those multiplying exposure variables.

	Dependent variable:
	$Y_{2,t}$
$Y_{1,t}$	-0.305^{***} (0.038)
$Y_{2,t-1}$	0.298^{***} (0.030)
$Y_{4,t-1}$	0.019 (0.027)
Constant	-0.023 (0.031)
Observations	999
R^2	0.153
Adjusted R^2	0.151
Residual Std. Error	0.983 (df = 995)
Note:	*p<0.1; **p<0.05; ***p<0.01

Table 3 – IV estimation of second equation in (1.44). Instruments are $Y_{1,t-1}$, $Y_{2,t-1}$ and $Y_{4,t-1}$.

	Dependent variable:
	$Y_{1,t}$
$Y_{2,t}$	0.304^{***}
	(0.113)
$Y_{1,t-1}$	0.759^{***}
	(0.040)
$Y_{4,t-1}$	-0.074^{***}
	(0.028)
Constant	0.017
	(0.032)
Observations	999
R^2	0.347
Adjusted R^2	0.345
Residual Std. Error	0.996 (df = 995)
Note:	*p<0.1; **p<0.05; ***p<0.01

Table 4 – IV estimation of first equation in (1.44). Instruments are $Y_{2,t-1}$, $Y_{1,t-1}$ and $Y_{4,t-1}$.

of traditional modeling strategies, but as a complement. Its usefulness relies on variable selection and in what is needed for an adequate estimate of the causal effect of interest. It also facilitates finding falsifiable restrictions in assumptions made, helping to avoid wrong models. Finally, its use in conjuntion with traditional and newer techniques may prove powerful, as shown by some examples in this chapter and the following ones.

2 Causal analysis of time series

"Shallow men believe in luck or in circumstance. Strong men believe in cause and effect."

— Ralph Waldo Emerson

In Chapter 1 we reviewed some aspects of structural causal models and causality in statistics and econometrics. Besides Pearl (2009) frequently refer to time, most of the book does not explicitly target time series inference. But, with some care, applications to time series are possible and fruitful. Section 1.6 introduced the concept of dynamic causal network, which is the basis for our approach.

In this Chapter we deal with peculiarities raised by time sampled data on causal analysis and provide some examples of causal inference through Monte Carlo experiments. New concepts of impulse responses, more meaningful for policy analysis, are presented. These new definitions also address complications raised by non-linear dynamics.

Specifically, in Section 2.1 we define the counterfactual impulse response function and derive from it two kinds of generalized impulse responses with causal interpretation and their respective expected values. In Section 2.2 we apply our concepts to a linear model in a Monte Carlo simulation, using artificial data to infer impulse responses. In Section 2.3 we proceed to the same exercise, but with a non-linear model. In Section 2.4 we define the causal impulse response to an intervention not just at t, but also at $t+1, t+2, \ldots, t+h$, allowing the analysis of policy plans. Finally, in Section 2.5 we discuss the causal interpretation of the Lucas critique and its implications for our approach.

2.1 Causal impulse response functions

This section presents our first main contributions, which are causal definitions of impulse response functions.

Definition 10. *The **counterfactual impulse response function (CIRF)** on horizon h of Y in a dynamic causal system among interventions $do(X_t = x_1)$ and $do(X_t = x_0)$ is given by*

$$CIRF_Y^{x_1,x_0}(h, \omega_{t-1}, \epsilon_{t,h}) \equiv Y_{t+h}^{x_1}(\omega_{t-1}, \epsilon_{t,h}) - Y_{t+h}^{x_0}(\omega_{t-1}, \epsilon_{t,h}), \tag{2.1}$$

where $Y_{t+h}^{x}(\omega_{t-1}, \epsilon_{t,h})$ is the potential response of Y_{t+h} given $X_t = x$, history ω_{t-1} and shocks realization $\epsilon_{t,h}$.

This definition uses counterfactuals, presented in Chapter 1. The situation $U = u$ here is $(\Omega_{t-1}, E_{t,h}) = (\omega_{t-1}, \epsilon_{t,h})$, where Ω_{t-1} represents all variables in the dynamic system up to time $t-1$ and $E_{t,h}$ all exogenous disturbances on the system up to time $t+h$.

Definition 10 is a comparison between two hypothetical stories which differs only by the intervention performed in X at time t. The *base counterfactual*, $Y_{t+h}^{x_0}(\omega_{t-1}, \epsilon_{t,h})$, is the value Y_{t+h} would have had if an intervention at t given by $do(X_t = x_0)$ had been done with all other relevant endogenous and exogenous variables realization given by ω_{t-1} and $\epsilon_{t,h}$. The *main counterfactual*, $Y_{t+h}^{x_1}(\omega_{t-1}, \epsilon_{t,h})$, is the value Y_{t+h} would have had if an intervention at t given by $do(X_t = x_1)$ had been done with everything else being equal.

Obviously, this CIRF is not observable, unless the exact same situations occur and exogenous interventions are feasible. In practical time series analysis, both situations are impossible or almost that. But, conceptually, Definition 10 is the basis for the construction of causal impulse responses that may be inferred from observational data.

How Definition 10 resembles counterfactuals as defined in Newman-Rubin potential outcomes approach? Here, set $X_t = x_1$ is to give the treatment status to "individual" (or unit of treatment) $\{\omega_{t-1}, \epsilon_{t,h}\}$ and set $X_t = x_0$ is to give the control status to *the same* "individual". In structural causal models framework, an "individual" is a particular realization of the exogenous variables, as seen in Chapter 1.

From Definition 10, we follow Koop, Pesaran and Potter (1996) and let the background variables vary, treating them as random variables. So, we may ask about (2.1) for arbitrary realizations ω_{t-1} of Ω_{t-1} and $\epsilon_{t,h}$ of $E_{t,h}$, which leads to Definition 11:

Definition 11. *The **generalized counterfactual impulse response function (GCIRF)** on horizon h of Y in a dynamic causal system among interventions $do(X_t = x_1)$ and $do(X_t = x_0)$ is given by*

$$GCIRF_Y^{x_1,x_0}(h, \Omega_{t-1}, E_{t,h}) \equiv Y_{t+h}^{x_1}(\Omega_{t-1}, E_{t,h}) - Y_{t+h}^{x_0}(\Omega_{t-1}, E_{t,h}), \tag{2.2}$$

where $Y_{t+h}^{x}(\Omega_{t-1}, E_{t,h})$ is the random variable Y_{t+h} given the random variable history Ω_{t-1}, the random variable shocks $E_{t,h}$ and intervention $do(X_t = x)$.

Of course, CIRFs are particular realizations of GCIRFs. From Definition 11 we see that GCIRF is a random variable, and one may define different responses of interest based on properties of this distribution. In particular, we define the *expected generalized counterfactual impulse response function*:

Definition 12. *The **expected generalized counterfactual impulse response function (EGCIRF)** on horizon h of a dynamic causal system between interventions $do(X_t = x_1)$ and $do(X_t = x_0)$ is given by*

$$EGCIRF_Y^{x_1,x_0}(h) \equiv E\left[GCIRF_Y^{x_1,x_0}(h, \Omega_{t-1}, E_{t,h})\right] = E\left[Y_{t+h}^{x_1}(\Omega_{t-1}, E_{t,h}) - Y_{t+h}^{x_0}(\Omega_{t-1}, E_{t,h})\right], \tag{2.3}$$

where the expectation on GCIRF is taken relative to Ω_{t-1} and $E_{t,h}$.

Notice that the EGCIRF is history independent and have a clear counterfactual interpretation. Indeed, we may see it as the expected value of the difference of Y_{t+h} between two conceptual experiments: setting $X_t = x_1$ and $X_t = x_0$. In other words, it represents the average difference of Y_{t+h} after a manipulation of the system setting $X_t = x_1$ against setting $X_t = x_0$, averaged over all relevant variables.

EGCIRF is the difference between two hypothetical interventions. Besides its usefulness, especially in comparing different potential policy decisions, regularly policy makers are interested on the direct effects of their actions compared with no action. One possible impact measure of interest is what would be the response of Y to an intervention in X compared with no intervention, regardless the state of all variables involved. We may achieve this goal comparing the effect of an intervention with what would have been the trajectory of the system without the intervention. This is easily done through GCIRF not setting x_0, which leads to the following definition:

Definition 13. *The **causal generalized impulse response (CGI)** on horizon h of a dynamic causal system after intervention $do(X_t = x)$ is given by*

$$CGI_Y^{x}(h, \Omega_{t-1}, E_{t,h}) \equiv Y_{t+h}^{x}(\Omega_{t-1}, E_{t,h}) - Y_{t+h}(\Omega_{t-1}, E_{t,h}), \tag{2.4}$$

where $Y_{t+h}(\Omega_{t-1}, E_{t,h})$ is the value of Y_{t+h} given history Ω_{t-1} and shocks $E_{t,h}$.

As before, this is a random variable. Again we may define its expected value:

Definition 14. *The **expected causal generalized impulse response (ECGI)** on horizon h of a dynamic causal system after intervention $do(X_t = x)$ is given by*

$$ECGI_Y^{x}(h, \Omega_{t-1}, E_{t,h}) \equiv E\left[Y_{t+h}^{x}(\Omega_{t-1}, E_{t,h}) - Y_{t+h}(\Omega_{t-1}, E_{t,h})\right], \tag{2.5}$$

where $Y_{t+h}(\Omega_{t-1}, E_{t,h})$ is the value of Y_{t+h} given history Ω_{t-1} and shocks $E_{t,h}$.

Each of these definitions have its own interest and potential applications. In next sections we apply these concepts together with causal calculus to make inferences based on Monte Carlo simulations of various systems.

2.2 Impulse responses and causal interventions in linear systems

Lets begin with linear models. In this class of models, impulse responses are *history independent*, that is, independent of ω_{t-1}. Suppose we want the response of Y_{t+h} for various horizons h to a unitary shock in X_t in the model given by (1.37). Lets reproduce its equations and causal graph here for convenience:

$$\begin{aligned} c_t &= 0.4c_{t-1} + 0.3c_{t-2} + 0.2y_{t-1} + \epsilon_t^c \\ x_t &= 0.4z_t - 0.3y_{t-1} + \epsilon_t^x \\ y_t &= 0.5z_t + 0.5x_t + \epsilon_t^y \\ z_t &= 0.6z_{t-1} + 0.5c_t + \epsilon_t^z \end{aligned} \tag{2.6}$$

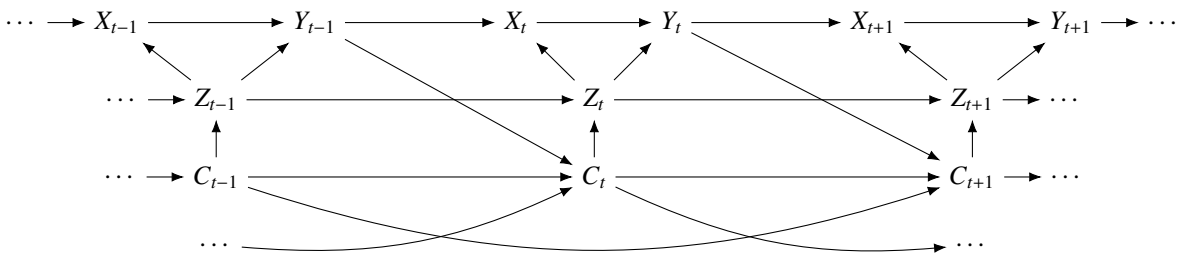

Figure 7 – Dynamic causal network representing the dynamic model in (2.6).

In the language of structural causal models, if we assume (2.6) as the DGP, the diagram in Figure 7 represents its causal relations. This network is an infinite DAG.

To make things interesting, consider C_t a non-observable variable in this system. Looking for equations (2.6), it is clear that functions f_k from Definition 8 are time invariant. Also, for each t, if we adjust for Y_{t-1} and Z_t we can compute the causal effect from X_t to any horizon Y_{t+h}, $h \in \mathbb{N}$. Actually, we may write:

$$P[Y_{t+h}|do(X_t = \delta)] = \sum_{Y_{t-1},Z_t} P(Y_{t+h}|X_t = \delta, Y_{t-1}, Z_t)P(Y_{t-1}, Z_t), \ \forall t \in \mathbb{Z} \tag{2.7}$$

by the back door criterion. Multiplying (2.7) by Y_{t+h} and summing, gives:

$$E[Y_{t+h}|do(X_t = \delta)] = \sum_{Y_{t-1},Z_t} E(Y_{t+h}|X_t = \delta, Y_{t-1}, Z_t)P(Y_{t-1}, Z_t). \tag{2.8}$$

Take the difference of (2.8) for $\delta = 1$ and $\delta = 0$:

$$E[Y_{t+h}|do(X_t = 1)] - E[Y_{t+h}|do(X_t = 0)] = \sum_{Y_{t-1},Z_t} [E(Y_{t+h}|X_t = 1, Y_{t-1}, Z_t) - E(Y_{t+h}|X_t = 0, Y_{t-1}, Z_t)]P(Y_{t-1}, Z_t). \tag{2.9}$$

Remembering that, by (1.28), $E\left[Y_{t+h}^1(\Omega_{t-1}, E_{t,h}) - Y_{t+h}^0(\Omega_{t-1}, E_{t,h})\right] = E[Y_{t+h}|do(X_t = 1)] - E[Y_{t+h}|do(X_t = 0)]$, equation (2.9) could have been deduced directly from (2.3), showing that we are identifying the EGCIRF for this system. Since this model is linear, the conditional expectations may be written as

$$E(Y_{t+h}|X_t, Y_{t-1}, Z_t) = \alpha_h + \beta_h^{X_t} X_t + \beta_h^{Y_{t-1}} Y_{t-1} + \beta_h^{Z_t} Z_t. \tag{2.10}$$

Substituting (2.10) into (2.9) gives

$$E[Y_{t+h}|do(X_t = 1)] - E[Y_{t+h}|do(X_t = 0)] = \beta_h^{X_t}. \tag{2.11}$$

Hence, the causal effect on the expected value of Y_{t+h} by setting $X_t = 1$ against setting $X_t = 0$ is given by (2.11), which can be estimated by OLS from (2.10). This approach is reminiscent of impulse responses estimation by local projections (JORDÀ, 2005), but here we explicitly address the causal nature of impulse responses.

Figure 8 shows a sample simulation of (2.6). Figure 9 shows the theoretical traditional IRF of (2.6) along with estimates using (2.11) from simulations of (2.6).

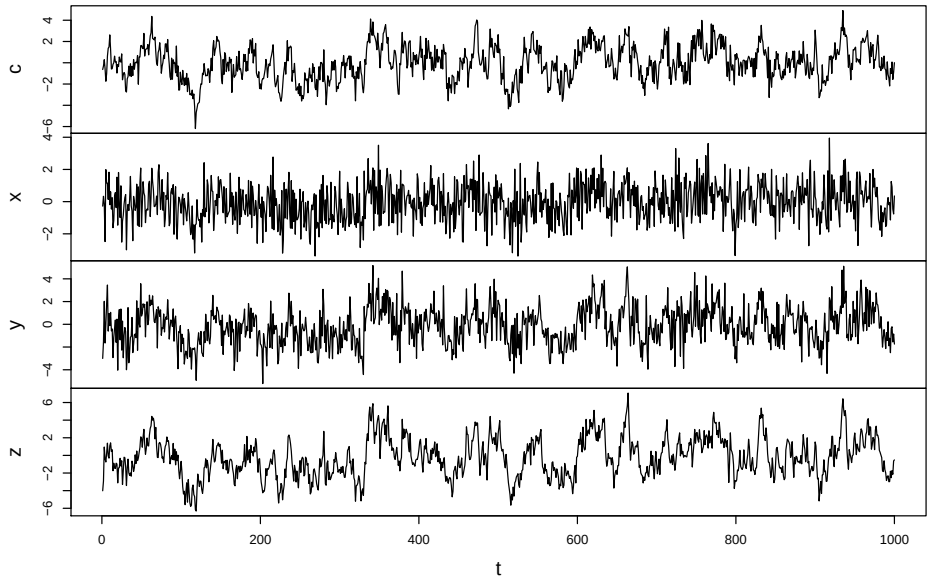

Figure 8 – Sample size 1,000 simulation of process (2.6). Burn-in period is 100 time samples.

Now observe that this model can be put in SVAR form:

$$A\psi_t = B_1\psi_{t-1} + B_2\psi_{t-2} + \epsilon_t, \tag{2.12}$$

where $\psi_t = \begin{pmatrix} c_t \\ x_t \\ y_t \\ z_t \end{pmatrix}$, $A = \begin{pmatrix} 1 & 0 & 0 & 0 \\ 0 & 1 & 0 & -0.4 \\ 0 & -0.5 & 1 & -0.5 \\ -0.5 & 0 & 0 & 1 \end{pmatrix}$, $B_1 = \begin{pmatrix} 0.4 & 0 & 0.2 & 0 \\ 0 & 0 & -0.3 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0.6 \end{pmatrix}$, $B_2 = \begin{pmatrix} 0.3 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$ and $\epsilon_t = \begin{pmatrix} \epsilon_t^c \\ \epsilon_t^x \\ \epsilon_t^y \\ \epsilon_t^z \end{pmatrix}$.

Lütkepohl (2005) calls a model like (2.12) an A-model. Its *structural* IRFs are given by (LÜTKEPOHL, 2005):

$$\Theta_j = \Phi_j A^{-1} \tag{2.13}$$

where Φ_j are recursively given by

$$\begin{aligned} \Phi_0 &= I_4 \\ \Phi_1 &= A^{-1}B_1 \\ \Phi_j &= \Phi_{j-1}A^{-1}B_1 + \Phi_{j-2}A^{-1}B_2 \text{ if } j \geq 2. \end{aligned} \tag{2.14}$$

It is straightforward to verify that (2.13) gives the same IRFs as (1.39).

Consider the SVAR approach estimates shown in Figure 10. Here we adopt the “correct” SVAR, estimating an unrestricted order 2 VAR and identifying the A matrix in (2.12) restricting all but the instantaneous

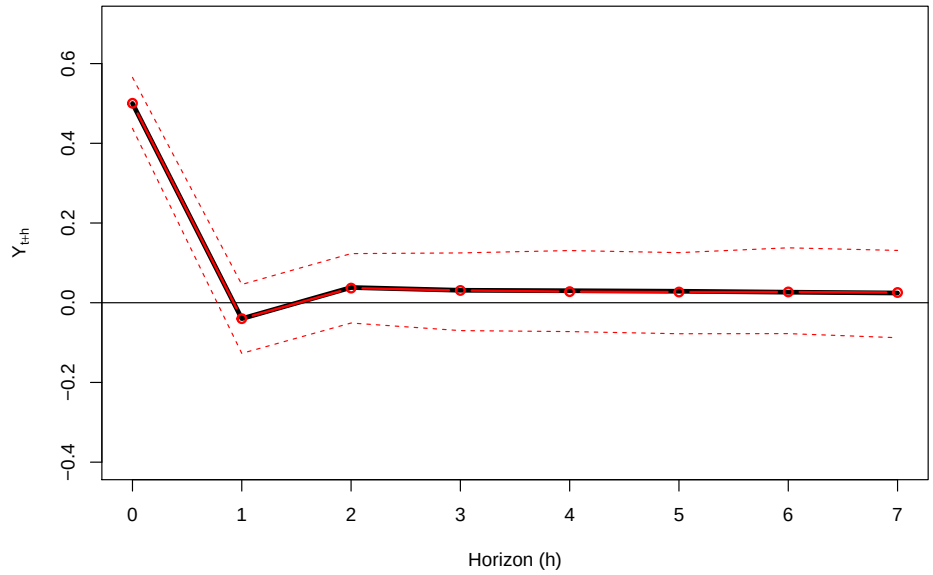

Figure 9 – The thick black line represents theoretical traditional IRF of Y_t in (2.6) for a unit shock in X_t. The red doted line represents the mean of 1,000 estimates using (2.11) estimated from 1,000 simulations of system (2.6). Each simulation generates series of length 1,000 after a burn-in period of 100 time samples. Dashed red lines represent percentiles 2.5% and 97.5% of $\beta_h^{X_t}$ estimates.

coefficients. More important, we assumed C_t observable, because we could not estimate this SVAR without this variable. Notice that estimates are strikingly similar to Figure 9 until $h = 2$. For higher h SVAR estimates became more precise. This is so because SVAR estimates are much more restricted and correctly specified. Also, SVAR IRFs assume all shocks after X_t as zero, as seem in (1.39). Our estimates does not assume this restriction, and IRF estimates remain unbiased because future shocks have zero mean and the system is linear.

Our results are untouched by the observability status of C_t. But SVARs will not work if C_t is non-observable. It may be argued that equations for x_t and y_t in (2.6) can be estimated by OLS, but the equation for z_t, which is essential to the dynamics of x_t and y_t, cannot. Some econometricians may suggest an instrumental variable approach, which depends on the existence of a suitable instrument. It is possible to infer from equations (2.6) that adjusting for y_{t-1} is sufficient to estimate the IRF, but this is not as obvious as is just looking to Figure 7. And this is the main reason why Pearl (2009) insists on a specific language for causality and the use of causal graphs: simplicity and intuitive appeal. Another advantage is to be a common language for identification of causal effects in general, which helps to connect ideas apparently unrelated.

To be fair, the SVAR approach is still useful in identifying the IRF from X_t to Y_t, especially if we choose the restrictions in the structural matrix based on causal information provided by Figure 7, showing the usefulness of causal models even for traditional approaches. Consider again a SVAR, but now with only variables X_t, Y_t and Z_t. It is clear that contemporaneous restrictions over A are given by Figure 4. Also, effects through the unobservable variable C_t come from longer lags than only 2, because we cannot control for C_t anymore. This sort of confounder is called a *dynamic confounder* by Blondel, Arias and Gavaldà (2017). We control for it with two more lags in the reduced form VAR. The IRFs are shown in Figure 11.

Our linear example does not show the full power of causal reasoning, since it is tractable by SVARs. But, as mentioned, the observability status of a confounder variable such as C_t may make the analysis based

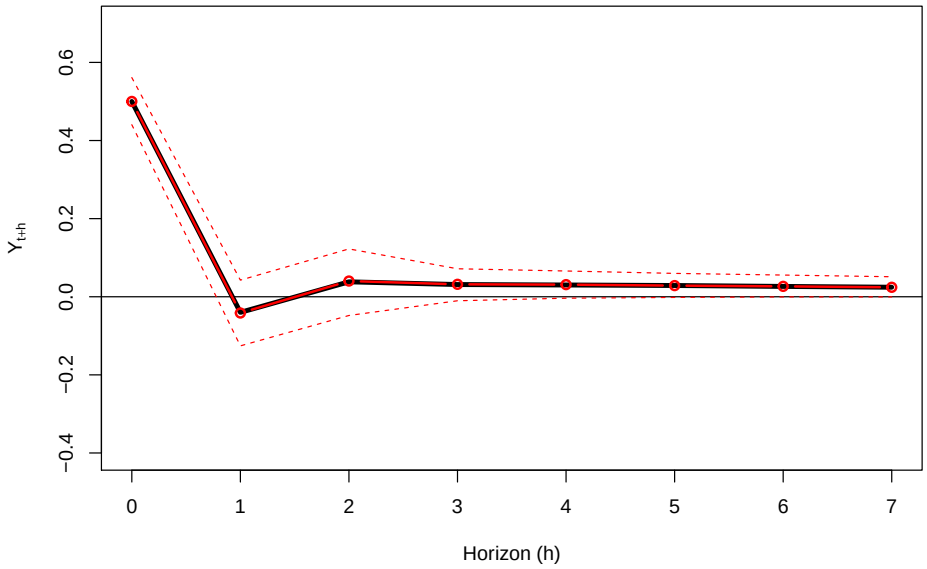

Figure 10 – The black thick line represents theoretical traditional IRF of Y_t in (2.6) for a unit shock in X_t. The red doted line represents the mean of 1,000 estimates using SVAR identification estimated from 1,000 simulations of system (2.6). Each simulation generates series of length 1,000 after a burn-in period of 100 time samples. Dashed red lines represent percentiles 2.5% and 97.5% of SVAR IRF estimates.

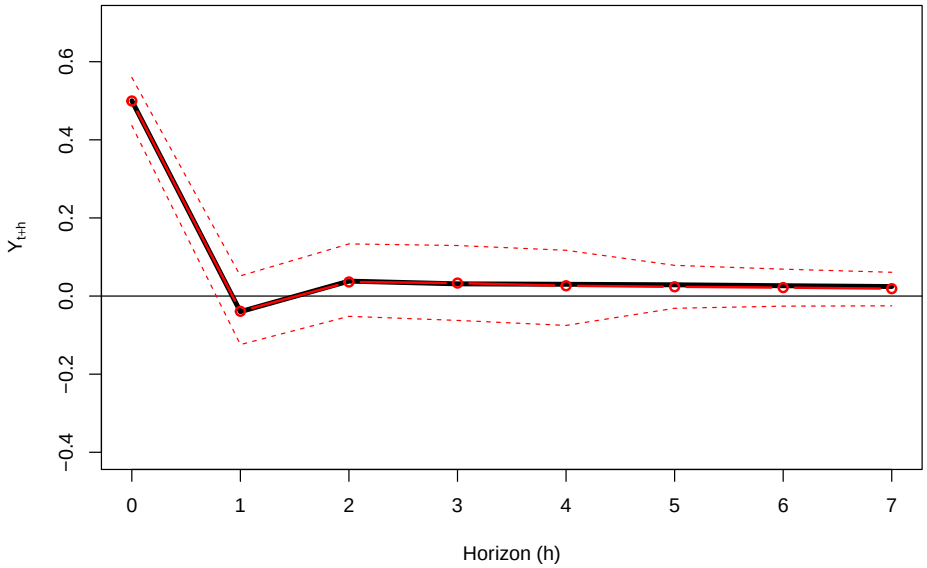

Figure 11 – The black thick line represents theoretical traditional IRF of Y_t in (2.6) for a unit shock in X_t. The red doted line represents the mean of 1,000 estimates using the 3 variable SVAR identification estimated from 1,000 simulations of system (2.6). Each simulation generates series of length 1,000 after a burn-in period of 100 time samples. Dashed red lines represent percentiles 2.5% and 97.5% of SVAR IRF estimates.

on automatic application of SVARs problematic, and causal reasoning based on DAGs may also help even in SVAR identification, as shown. The non-linear case is much more complicated, since the concept of traditional impulse response function itself loses its meaning. This will be discussed in next section.

2.3 Impulse responses and causal interventions in non-linear systems

Koop, Pesaran and Potter (1996) introduce the concept of *generalized impulse response function*, which try to overcome difficulties raised by non-linear systems. As seen in Section 2.1, we built our analysis over their ideas, but emphasizing the causal nature of IRFs. The authors define the *traditional* impulse response function as in Definition 9, which is the most popular form in econometric analysis according to them. They also discuss its limitations and motivate their generalization.

The main difficulty raised by non-linear systems stems from its history and shock dependence. In Section 2.2, the specific values of X_t, Y_t, Z_t and C_t at the moment of the shock does not have any effect over the IRF. The size of the shock may also be rescaled to any size desired, simply multiplying the response by the same amount. These properties have important consequences for the interpretation of what *is* an IRF. For example, in Section 2.2 we treated the operation $do(X_t = 1)$ as if it was a unit shock in X_t. But, in model (2.6), a unit shock in X_t is to set $\epsilon_t^x = 1$, according to prevalent econometric jargon. Nonetheless, both affirmations are the same in linear models, since the deterministic part of X_t, namely, $0.4z_t - 0.3y_{t-1}$, is already given, and when we take the difference in Equation (1.39) it disappears, leaving only the shock ϵ_t^x. So, in linear models both interventions are the same.

However, in non-linear models this is not true. Writing the equation for X_t in a more general form, like

$$X_t = f(pa(X_t), \epsilon_t^x), \tag{2.15}$$

for arbitrary f and exogenous (as in the linear case) ϵ_t^x, the difference between system responses for $do(X_t = 1)$ and $do(X_t = 0)$ are not necessarily the same as the difference setting $\epsilon_t^x = 1$ against $\epsilon_t^x = 0$. Furthermore, the shock interpretation becomes obscured because $f(pa(X_t), \epsilon_t^x)$ is now history dependent, and the quantity $E(X_t|pa(X_t), \epsilon_t^x = 1) - E(X_t|pa(X_t), \epsilon_t^x = 0)$ is a function of $pa(X_t)$, since its effect do not necessarily cancel as in the linear case. So, for different values of $pa(X_t)$, $\epsilon_t^x = x$ induces different values for X_t depending on the system history. Meanwhile, the "do" operation remains unambiguous, since it imposes the substitution of (2.15) by $X_t = x$, setting unequivocally X_t.

Of course that it may be of interest the effect of a shock given X_t, such as the effect of raising X_t by one unit. This situation is still complicated in (2.15) for the shock approach, since ϵ_t^x does not enter into this equation as an additive term. Indeed, Koop, Pesaran and Potter (1996) define their system as (1.38), with additive shocks, probably to avoid this sort of complication. The "do" operator also cannot solve this problem, but counterfactuals deal with it naturally. For details, see Pearl (2009) and Pearl, Glymour and Jewell (2016).

As an example of application of our concepts in non-linear case, consider a non-linear version of model (2.6) given by equations (2.16):

$$\begin{aligned} c_t &= 0.4c_{t-1} + 0.3c_{t-2} + 0.2y_{t-1} + \epsilon_t^c \\ x_t &= 0.4z_t - 0.3y_{t-1} + \epsilon_t^x \\ y_t &= 4\cos(0.5z_t + 0.5x_t) + \epsilon_t^y \\ z_t &= 0.6z_{t-1} + 0.5c_t + \epsilon_t^z \end{aligned} \tag{2.16}$$

where $\epsilon_t^k \sim N(0,1)$ for all $k \in \{c, x, y, z\}$ are serially independent and also jointly independent, as before. The infinite DAG representing this system is the same as in Figure 7, but now the system is non-linear because of the cosine in the equation for Y_t. Figure 12 shows some traditional IRFs for this system given different initial

values. Notice how different these IRFs are, even being produced by the same shocks on the same model. Also, Figure 13 shows traditional IRFs for this system given equal initial values and different shocks. Notice how responses are not proportional to shocks. Indeed, shocks with opposite signs give responses with equal signs.

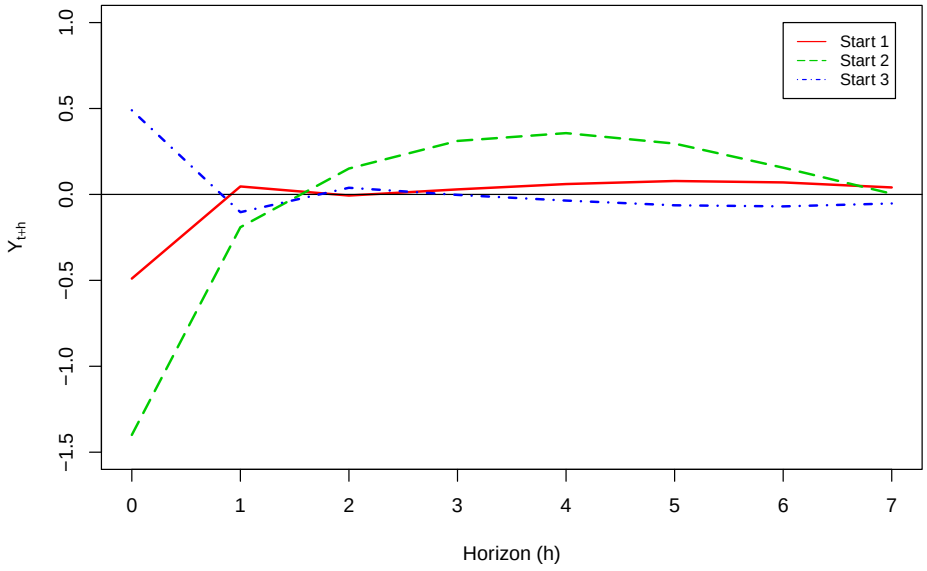

Figure 12 – Traditional IRFs for Y_{t+h} setting $X_t = 1$ against $X_t = 0$ for different starting values of system (2.16). Solid red line represents starting values $C_{t-2} = 0$, $C_{t-1} = 0$, $Y_{t-1} = 0$, $Z_{t-1} = 0$. Dashed green line represents starting values $C_{t-2} = 0.85469449$, $C_{t-1} = 0.85469449$, $Y_{t-1} = 1.30697124$, $Z_{t-1} = 1.06806908$, obtained as the respective averages for 100,000 simulations of (2.16) after a 100 burn-in period. Dotted dashed blue line represents starting values $C_{t-2} = 0$, $C_{t-1} = 0$, $Y_{t-1} = 0$, $Z_{t-1} = -\frac{5}{3}$.

One way to simulate GCIRF (2.2) for model (2.16) is the following:

1. Simulate the system for a set of random shocks ϵ^i and initial values ω_0^i setting $X_t = x_1$ for some sufficiently large t and collect Y_{t+h} for all h, here denoted $Y_{t+h}^{x_1}(i)$;
2. Repeat the simulation *for the same set of shocks and initial values* of item 1, but setting $X_t = x_0$ on the same t and collect Y_{t+h} for all h, now called $Y_{t+h}^{x_0}(i)$;
3. Collect the difference $Y_{t+h}^{x_1}(i) - Y_{t+h}^{x_0}(i)$, which will be one sample of $GCIRF_Y^{x_1,x_0}(h, \Omega_{t-1}, E_{t,h})$;
4. Repeat steps 1, 2 and 3 until collect sufficient samples of $GCIRF_Y^{x_1,x_0}(h, \Omega_{t-1}, E_{t,h})$.

Figure 14 shows the mean and percentiles 2.5% and 97.5% of 10,000 samples of $GCIRF_Y^{x_1,x_0}(h, \Omega_{t-1}, E_{t,h})$ with $x_1 = 1$ and $x_0 = 0$ for process (2.16).

Notice that the thick solid line represents the expected generalized counterfactual impulse response function (EGCIRF) for this system. We may estimate it by the following procedure: from equation (2.3), we have

$$EGCIRF_Y^{x_1,x_0}(h) = E\left[Y_{t+h}^{x_1}(\Omega_{t-1}, E_{t,h}) - Y_{t+h}^{x_0}(\Omega_{t-1}, E_{t,h})\right] = E[Y_{t+h}|do(X_t = x_1)] - E[Y_{t+h}|do(X_t = x_0)], \quad (2.17)$$

with $x_1 = 1$ and $x_0 = 0$. By the back-door criterion,

$$EGCIRF_Y^{1,0}(h) = \sum_{Y_{t-1},Z_t} [E(Y_{t+h}|X_t = 1, Y_{t-1}, Z_t) - E(Y_{t+h}|X_t = 0, Y_{t-1}, Z_t)]P(Y_{t-1}, Z_t). \quad (2.18)$$

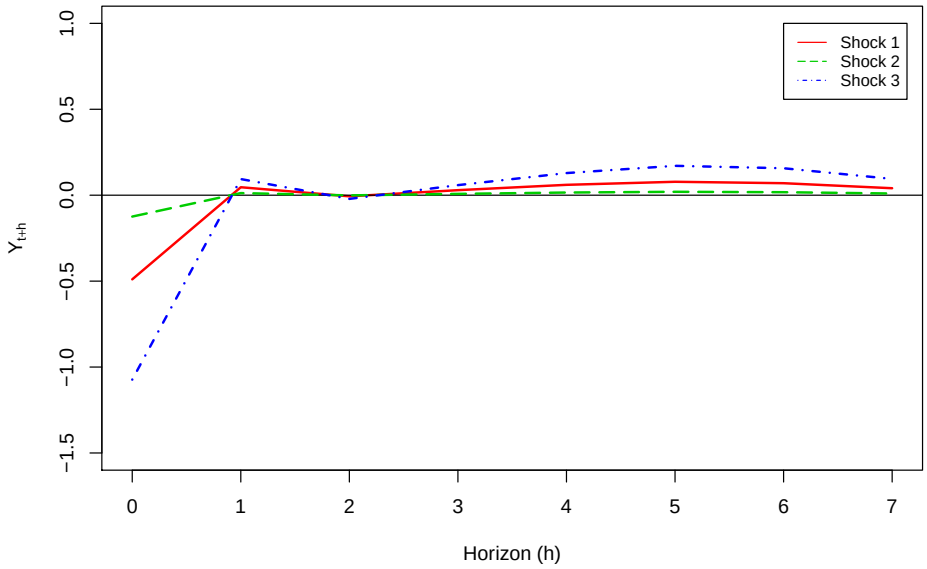

Figure 13 – Traditional IRFs for Y_{t+h} for starting values of system (2.16) given by $C_{t-2} = 0$, $C_{t-1} = 0$, $Y_{t-1} = 0$, $Z_{t-1} = 0$. Solid red line represents shock $X_t = 1$ against $X_t = 0$. Dashed green line represents shock $X_t = 0.5$ against $X_t = 0$. Dotted dashed blue line represents shock $X_t = -1.5$ against $X_t = 0$.

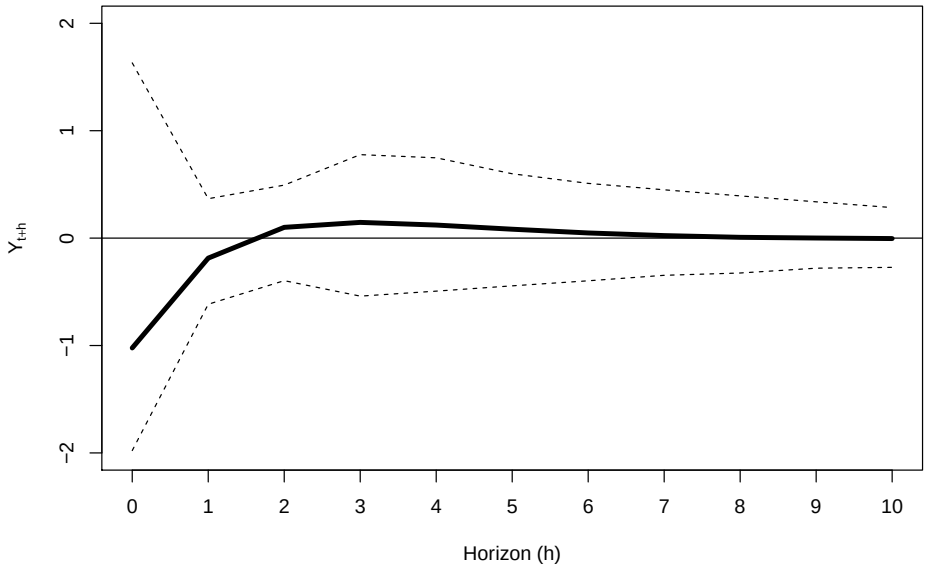

Figure 14 – Generalized counterfactual impulse response function (GCIRF) for Y_{t+h} with $x_1 = 1$ and $x_0 = 0$. Thick solid line is the mean and dashed lines are percentiles 2.5% and 97.5% of 10,000 samples.

To estimate (2.18) we may first estimate the conditional expectation function $f(x, Y_{t-1}, Z_t) = E(Y_{t+h}|X_t = x, Y_{t-1}, Z_t)$. Then, we estimate the expected value of $g(Y_{t-1}, Z_t) = f(1, Y_{t-1}, Z_t) - f(0, Y_{t-1}, Z_t)$, substituting sample values for Y_{t-1} and Z_t and taking the average.

Of course this procedure is much simpler for linear f, leading to equation (2.11). This linear approach is shown in Figure 15. Notice that estimates are biased, especially for $h = 0$. This is because (2.16) is non-linear.

The non-linearity of the model may be inferred from data through standard diagnostics of regression estimates. This is important, because separating causal identification from estimation makes easier know from data if we have a misspecification due to a bad causal model (which can be tested through conditional independence implications of the causal diagram) or to a bad statistical model (which can be tested through residual analysis and lots of other tools available from statistics).

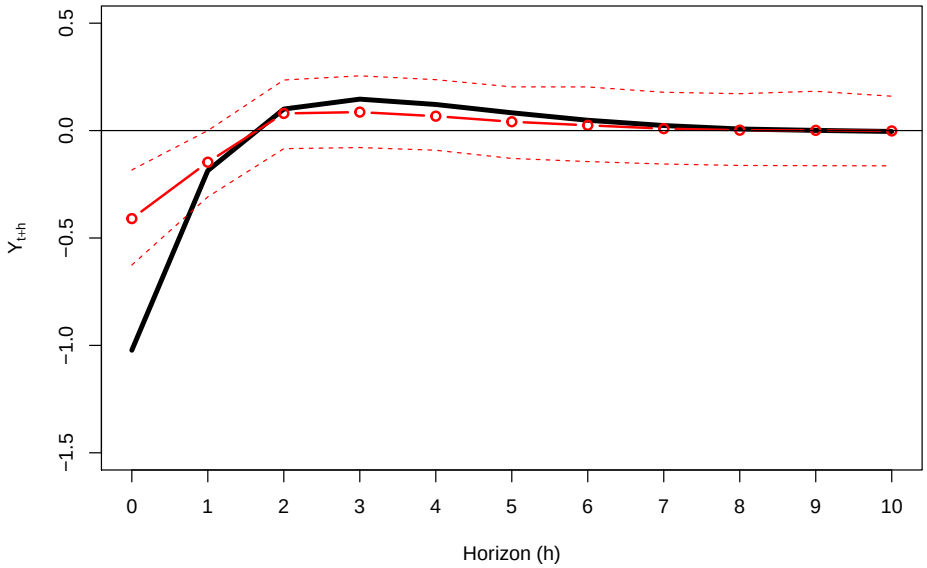

Figure 15 – Thick solid line is the expected generalized counterfactual impulse response function (EGCIRF) for Y_{t+h} in model (2.16) with $x_1 = 1$ and $x_0 = 0$. Red dotted dashed line is the mean of 1,000 estimates of EGCIRF based on linear conditional expectation estimates of 1,000 simulations of (2.16). Red dashed lines are percentiles 2.5% and 97.5% for these estimates. Each simulation have 1,000 data points after a burn-in period of 100.

As an example, consider one of the regressions fitted estimating $EGCIRF_Y^{1,0}(h)$ for $h = 0$ used to plot Figure 15. Table 5 shows results for this regression.

At first sight this table is fine. But looking to residuals we see that this model is probably misspecified. Figure 16 shows its fitted values against residuals, a standard plot for functional form misspecification diagnostic. As can be seen, the model is badly specified, and this plot suggest a non-linear relationship among regressors and regressand, as expected. To deal with this we may add higher order terms on the regression, for example. However, here we choose a different approach, through the use of generalized additive models (GAM) (HASTIE; TIBSHIRANI, 1987; HASTIE, 2017; WOOD, 2017).

We choose GAMs for three main reasons. First, it is flexible and powerful. Second, its application is straightforward in R with package *mgcv* (R Core Team, 2019; WOOD, 2017). Third, with GAMs we show how different techniques may be used to estimate conditional expectations that we already know how to interpret. This is possible because of the separation between causal identification and model estimation greatly facilitated by the structural causal model approach advocated in this thesis. For a brief presentation of GAMs and some details on the model used here, see Appendix A. Figure 17 shows the same EGCIRF with $g(Y_{t-1}, Z_t) = f(1, Y_{t-1}, Z_t) - f(0, Y_{t-1}, Z_t)$ estimated by GAMs. The fit is much better, almost without bias. Notice that in our analysis no knowledge about the DGP beyond that in Figure 7 was used. This knowledge is the same for the linear and non-linear versions, because causal links are abstracted from functional forms. Furthermore, we treat C_t as a non-observable confounder.

	Dependent variable:
	Y_t
X_t	-0.496^{***}
	(0.070)
Z_t	-0.555^{***}
	(0.054)
Y_{t-1}	0.169^{***}
	(0.033)
Constant	1.886^{***}
	(0.099)
Observations	999
R^2	0.343
Adjusted R^2	0.341
Residual Std. Error	2.178 (df = 995)
F Statistic	173.224^{***} (df = 3; 995)
Note:	*p<0.1; **p<0.05; ***p<0.01

Table 5 – Regression results for $EGCIRF_Y^{1,0}(h)$ with $h = 0$ estimated from one simulation of (2.16). The causal effect of interest is the coefficient for X_t.

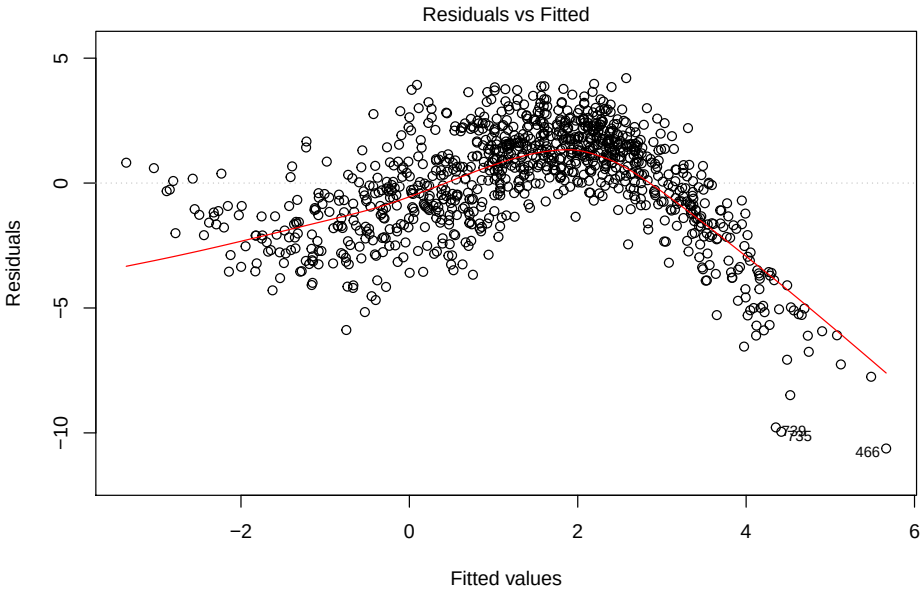

Figure 16 – Scatter plot of fitted values vs residuals for model in Table 5. Clearly there is a non-linear relationship among variables.

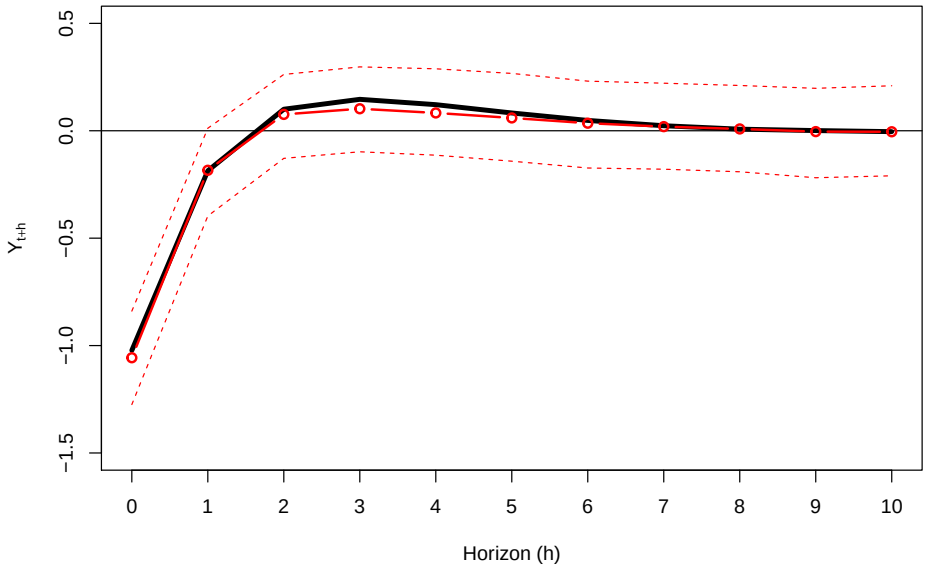

Figure 17 – Thick solid line is the expected generalized counterfactual impulse response function (EGCIRF) for Y_{t+h} in model (2.16) with $x_1 = 1$ and $x_0 = 0$. Red dotted dashed line is the mean of 1,000 estimates of EGCIRF based on generalized additive models conditional expectation estimates of 1,000 simulations of (2.16). Red dashed lines are percentiles 2.5% and 97.5% for these estimates. Each simulation have 1,000 data points after a burn-in period of 100.

Now lets see the CGI, presented in Definition 13, for this model. CGI is also a random variable. We may sample it in a way similar to what was done on GCIRF. Enough for that is just let x_0 free to vary. Figure 18 shows the mean and percentiles 2.5% and 97.5% of 10,000 samples of $CGI^x_Y(h, \Omega_{t-1}, E_{t,h})$ with $x = -1$ for process (2.16).

Notice again that the thick black line in Figure 18 represents the ECGI for system (2.16). We may estimate it using GAMs, as before. The result for 1,000 simulations is shown in Figure 19.

These results are remarkable because of the assumptions substantiating them. Basically, we just need a *qualitative* causal model, given by a causal graph like that of Figure 7, and, even with unobserved confounders and non-linear dynamics, we where able to estimate with high precision some impulse responses, here defined as exogenous interventions of interest. Our assumptions were weaker than those normally presumed in econometric evaluation of policy interventions in time series.

Our estimating procedures concentrated on average impulse responses defined in general models because of its simplicity. It is also possible to extend our analysis for estimation of other moments or full distributions such as GCIRF or CGI, for example. In principle, distributional problems are conceptually the same regarding identification, and distribution estimation is feasible through dozens of statistical and machine learning procedures available in the specialized literature. Another possible extension with great interest for policy analysis is the evaluation of conditional forecasts.

2.4 Sequential plan response

In Chapter 1 we discussed limitations of impulse responses defined without causal content. On previous sections we presented alternative definitions which not only address the lack of causal meaning on previous popular IRF definitions in the literature, but also face difficulties raised by non-linear models.

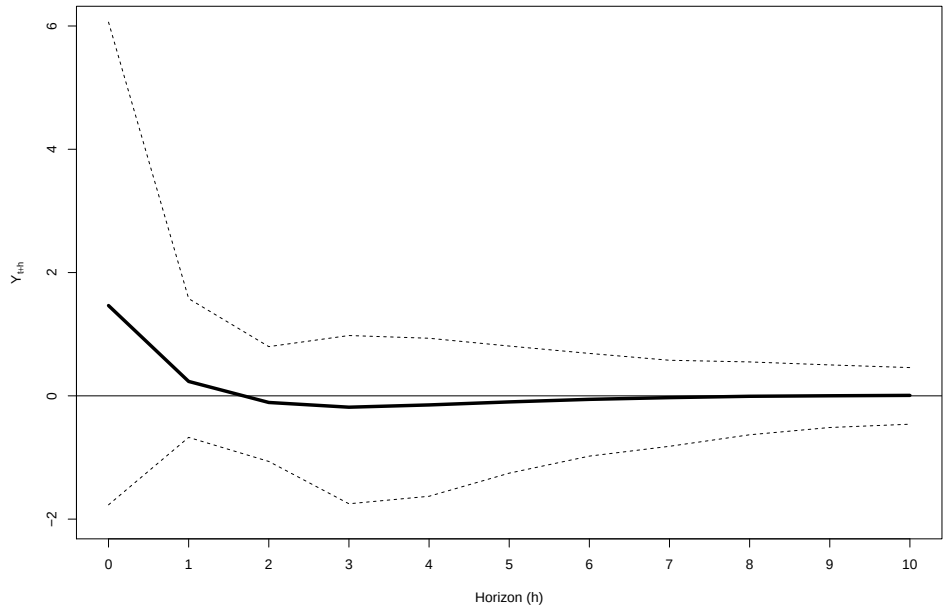

Figure 18 – Causal generalized impulse response (CGI) for Y_{t+h} with $x = -1$. Thick solid line is the mean and dashed lines are percentiles 2.5% and 97.5% of 10,000 samples.

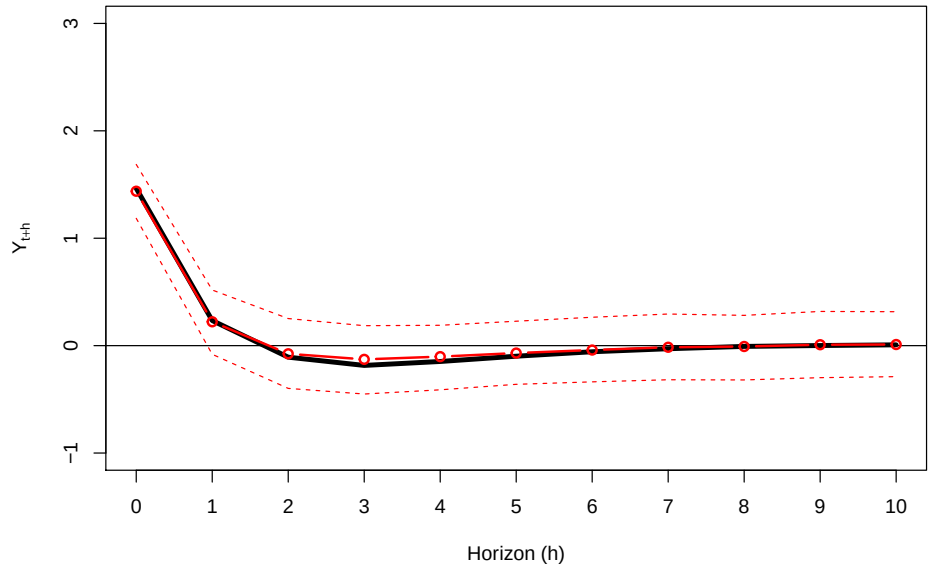

Figure 19 – Thick solid line is the expected causal generalized impulse response (ECGI) for Y_{t+h} in model (2.16) with $x = -1$. Red dotted dashed line is the mean of 1,000 estimates of ECGI based on generalized additive models conditional expectation estimates of 1,000 simulations of (2.16). Red dashed lines are percentiles 2.5% and 97.5% for these estimates. Each simulation have 1,000 data points after a burn-in period of 100.

Here we recover the discussion about future shocks. Remember that in Definition 9, future shocks such as V_{t+1}, V_{t+2}, etc., are set to zero. In linear models with zero expectations exogenous shocks this is almost immaterial. Actually, since our interest is on inference about counterfactual interventions, namely, a comparison between two hypothetical experiments leaving *all else equal*, including future shocks, in models where shocks are additive, the difference between two hypothetical histories would cancel all future shocks. As we have seen, this is not true in general. Also, in our framework, interventions are made directly on policy variables. So, instead of talking about future shocks, we talk about future interventions. The methods developed in earlier sections predict the response of the system to an exogenous manipulation of the policy variable at t, leaving this policy variable free to vary endogenously after the intervention. Now we are interested in the effect of a sequence of exogenous interventions through time.

A very useful result in discussing future interventions is Theorem 3 on identification of plans. Actually, a plan consisting of a sequence of interventions is exactly what happens when we set not just the shock at time t, but also at $t+1, t+2, \ldots$. Consider that each w_i in Theorem 3 consists of a set of variables $\{w_{i,1}, w_{i,2}, \ldots, w_{i,l_i}\}$. Then, under conditions outlined in Theorem 3, the non-parametric causal effect on horizon h of a sequence of interventions $\{\hat{x}_1, \ldots, \hat{x}_h\}$ is:

$$\begin{aligned} P(y_h|\hat{x}_1, \ldots, \hat{x}_h) = & \sum_{w_{1,1},\ldots,w_{h,l_h}} P(y_h|w_{1,1}, \ldots, w_{h,l_h}, x_1, \ldots, x_h) \\ & \times \prod_{k=1}^{h} P(w_{k,1}, \ldots, w_{k,l_k}|w_{1,1}, \ldots, w_{k-1,l_{k-1}}, x_1, \ldots, x_{k-1}). \end{aligned} \tag{2.19}$$

Multiplying both sides by y_h and summing through this variable, we arrive at the non-parametric expected causal effect:

$$\begin{aligned} E(y_h|\hat{x}_1, \ldots, \hat{x}_h) = & \sum_{w_{1,1},\ldots,w_{h,l_h}} E(y_h|w_{1,1}, \ldots, w_{h,l_h}, x_1, \ldots, x_h) \\ & \times \prod_{k=1}^{h} P(w_{k,1}, \ldots, w_{k,l_h}|w_{1,1}, \ldots, w_{k-1,l_{k-1}}, x_1, \ldots, x_{k-1}). \end{aligned} \tag{2.20}$$

We may approximate conditional expectations in (2.20) by linear functions, such that we may have non-biased estimates of these approximations through OLS. Moreover, this procedure can be iterated in such a way that we get an estimator for (2.20). In fact, this is the content of the following proposition:

Proposition 1. *The expected causal effect in* (2.20) *may be approximated by the equation*

$$\begin{aligned} E(y_h|\hat{x}_1, \ldots, \hat{x}_h) = & \alpha^{y_h} + \sum_{m=2}^{h} \sum_{n=1}^{l_m} \beta_{m,n}^{y_h} \alpha(m-1)^{m,n} \\ & + \sum_{j=1}^{l_1} \left[\beta_{1,j}^{y_h} + \sum_{m=2}^{h} \sum_{n=1}^{l_m} \beta_{m,n}^{y_h} \beta(m-1)_{1,j}^{m,n} \right] E(w_{1,j}) \\ & + \sum_{i=1}^{h-1} \left[\gamma_i^{y_h} + \sum_{m=i+1}^{h} \sum_{n=1}^{l_m} \beta_{m,n}^{y_h} \gamma(m-1)_i^{m,n} \right] x_i + \gamma_h^{y_h} x_h, \end{aligned}$$

where α^{y_h}, $\beta_{m,n}^{y_h}$ *e* $\gamma_m^{y_h}$ *are given by*

$$E(y_h|w_{1,1}, \ldots, w_{h,l_h}, x_1, \ldots, x_h) = \alpha^{y_h} + \sum_{m=1}^{h} \sum_{n=1}^{l_m} \beta_{m,n}^{y_h} w_{m,n} + \sum_{m=1}^{h} \gamma_m^{y_h} x_m$$

and $\alpha(m-1)^{m,n}$, $\beta(m-1)^{m,n}_{1,j}$ *and* $\gamma(m-1)^{m,n}_{i}$ *are recursively calculated by the following equations:*

$$\begin{aligned}
\alpha(q+1)^{m,n} &= \alpha(q)^{m,n} + \sum_{p=1}^{l_{m-q}} \beta(q)^{m,n}_{m-q,p}\alpha(1)^{m-q,p} \\
\beta(q+1)^{m,n}_{i,j} &= \beta(q)^{m,n}_{i,j} + \sum_{p=1}^{l_{m-q}} \beta(q)^{m,n}_{m-q,p}\beta(1)^{m-q,p}_{i,j} \\
\gamma(q+1)^{m,n}_{i} &= \gamma(q)^{m,n}_{i} + \sum_{p=1}^{l_{m-q}} \beta(q)^{m,n}_{m-q,p}\gamma(1)^{m-q,p}_{i},
\end{aligned}$$

where $1 \leq q \leq m-1$ *and* $\alpha(1)^{m,n}$, $\beta(1)^{m,n}_{i,j}$ *and* $\gamma(1)^{m,n}_{i}$ *are given by*

$$E(w_{m,n}|w_{1,1},\ldots,w_{m-1,l_{m-1}},x_1,\ldots,x_{m-1}) = \alpha(1)^{m,n} + \sum_{i=1}^{m-1}\sum_{j=1}^{l_i}\beta(1)^{m,n}_{i,j}w_{i,j} + \sum_{i=1}^{m-1}\gamma(1)^{m,n}_{i}x_i.$$

Proof. Consider the expected causal effect given by (2.20). We may approximate the conditional expectation by:

$$E(y_h|w_{1,1},\ldots,w_{h,l_h},x_1,\ldots,x_h) = \alpha^{y_h} + \sum_{m=1}^{h}\sum_{n=1}^{l_m}\beta^{y_h}_{m,n}w_{m,n} + \sum_{m=1}^{h}\gamma^{y_h}_{m}x_m. \tag{2.21}$$

Substituting (2.21) into (2.20), we have:

$$\begin{aligned}
E(y_h|\hat{x}_1,\ldots,\hat{x}_h) &= \alpha^{y_h} + \sum_{m=1}^{h}\gamma^{y_h}_{m}x_m + \\
&+ \sum_{m=1}^{h}\sum_{n=1}^{l_m}\beta^{y_h}_{m,n}\left[\sum_{w_{1,1},\ldots,w_{h,l_h}} w_{m,n}\prod_{k=1}^{h}P(w_{k,1},\ldots,w_{k,l_k}|w_{1,1},\ldots,w_{k-1,l_{k-1}},x_1,\ldots,x_{k-1})\right].
\end{aligned} \tag{2.22}$$

Now, it is necessary evaluate the term inside square brackets in (2.22):

$$W^{h}_{m,n} = \sum_{w_{1,1},\ldots,w_{h,l_h}} w_{m,n}\prod_{k=1}^{h}P(w_{k,1},\ldots,w_{k,l_k}|w_{1,1},\ldots,w_{k-1,l_{k-1}},x_1,\ldots,x_{k-1}). \tag{2.23}$$

To do so, we adopt the same strategy and substitute conditional expectations by linear functions. First, note that (2.23) may be rewritten, for $m > 1$, in the following way:

$$\begin{aligned}
W^{h}_{m,n} = &\sum_{w_{1,1},\ldots,w_{h,l_h}} w_{m,n}\prod_{k=1}^{h}P(w_{k,1},\ldots,w_{k,l_k}|w_{1,1},\ldots,w_{k-1,l_{k-1}},x_1,\ldots,x_{k-1}) = \\
&\sum_{w_{1,1},\ldots,w_{m,l_m}} w_{m,n}\prod_{k=1}^{m}P(w_{k,1},\ldots,w_{k,l_k}|w_{1,1},\ldots,w_{k-1,l_{k-1}},x_1,\ldots,x_{k-1}) = \\
&\sum_{w_{1,1},\ldots,w_{m-1,l_{m-1}}}\prod_{k=1}^{m-1}P(w_{k,1},\ldots,w_{k,l_k}|w_{1,1},\ldots,w_{k-1,l_{k-1}},x_1,\ldots,x_{k-1}) \\
&\qquad\times E(w_{m,n}|w_{1,1},\ldots,w_{m-1,l_{m-1}},x_1,\ldots,x_{m-1}).
\end{aligned} \tag{2.24}$$

Substituting conditional expectations gives

$$\begin{aligned}
W^{h}_{m,n} = &\sum_{w_{1,1},\ldots,w_{m-1,l_{m-1}}}\prod_{k=1}^{m-1}P(w_{k,1},\ldots,w_{k,l_k}|w_{1,1},\ldots,w_{k-1,l_{k-1}},x_1,\ldots,x_{k-1}) \\
&\qquad\times\left[\alpha(1)^{m,n} + \sum_{i=1}^{m-1}\sum_{j=1}^{l_i}\beta(1)^{m,n}_{i,j}w_{i,j} + \sum_{i=1}^{m-1}\gamma(1)^{m,n}_{i}x_i\right],
\end{aligned} \tag{2.25}$$

where we adopt the following notation for $E(w_{m,n}|w_{1,1},\ldots,w_{m-1,l_{m-1}},x_1,\ldots,x_{m-1})$ linear approximations:

$$\alpha(1)^{m,n}+\sum_{i=1}^{m-1}\sum_{j=1}^{l_i}\beta(1)_{i,j}^{m,n}w_{i,j}+\sum_{i=1}^{m-1}\gamma(1)_i^{m,n}x_i. \tag{2.26}$$

Here, $\alpha(1)^{m,n}$ is the constant term, $\beta(1)_{i,j}^{m,n}$ is the coefficient of $w_{i,j}$ and $\gamma(1)_i^{m,n}$ is the coefficient of x_i. The number 1 inside brackets is an index used for the induction argument.

Suppose that

$$\begin{aligned}W_{m,n}^h=&\sum_{w_{1,1},\ldots,w_{m-q,l_{m-q}}}\prod_{k=1}^{m-q}P(w_{k,1},\ldots,w_{k,l_k}|w_{1,1},\ldots,w_{k-1,l_{k-1}},x_1,\ldots,x_{k-1})\\&\times\left[\alpha(q)^{m,n}+\sum_{i=1}^{m-q}\sum_{j=1}^{l_i}\beta(q)_{i,j}^{m,n}w_{i,j}+\sum_{i=1}^{m-1}\gamma(q)_i^{m,n}x_i\right].\end{aligned} \tag{2.27}$$

for any q, $1\le q\le m-1$. Summing through $w_{m-q,1},\ldots,w_{m-q,l_{m-q}}$, we have:

$$\begin{aligned}W_{m,n}^h=&\sum_{w_{1,1},\ldots,w_{m-(q+1),l_{m-(q+1)}}}\prod_{k=1}^{m-(q+1)}P(w_{k,1},\ldots,w_{k,l_k}|w_{1,1},\ldots,w_{k-1,l_{k-1}},x_1,\ldots,x_{k-1})\\&\times\left[\alpha(q)^{m,n}+\sum_{i=1}^{m-(q+1)}\sum_{j=1}^{l_i}\beta(q)_{i,j}^{m,n}w_{i,j}+\sum_{i=1}^{m-1}\gamma(q)_i^{m,n}x_i\right.\\&\left.+\sum_{p=1}^{l_{m-q}}\beta(q)_{m-q,p}^{m,n}\left(\alpha(1)^{m-q,p}+\sum_{i=1}^{m-(q+1)}\sum_{j=1}^{l_i}\beta(1)_{i,j}^{m-q,p}w_{i,j}+\sum_{i=1}^{m-1}\gamma(1)_i^{m-q,p}x_i\right)\right],\end{aligned} \tag{2.28}$$

where the term inside parenthesis is the linear approximation of

$$E(w_{m-q,p}|w_{1,1},\ldots,w_{m-(q+1),l_{m-(q+1)}},x_1,\ldots,x_{m-(q+1)}), \tag{2.29}$$

given by (2.26). Rearranging and grouping (2.28) gives

$$\begin{aligned}W_{m,n}^h=&\sum_{w_{1,1},\ldots,w_{m-(q+1),l_{m-(q+1)}}}\prod_{k=1}^{m-(q+1)}P(w_{k,1},\ldots,w_{k,l_k}|w_{1,1},\ldots,w_{k-1,l_{k-1}},x_1,\ldots,x_{k-1})\\&\times\left[\alpha(q+1)^{m,n}+\sum_{i=1}^{m-(q+1)}\sum_{j=1}^{l_i}\beta(q+1)_{i,j}^{m,n}w_{i,j}+\sum_{i=1}^{m-1}\gamma(q+1)_i^{m,n}x_i\right],\end{aligned} \tag{2.30}$$

where we define:

$$\begin{aligned}\alpha(q+1)^{m,n}&=\alpha(q)^{m,n}+\sum_{p=1}^{l_{m-q}}\beta(q)_{m-q,p}^{m,n}\alpha(1)^{m-q,p}\\\beta(q+1)_{i,j}^{m,n}&=\beta(q)_{i,j}^{m,n}+\sum_{p=1}^{l_{m-q}}\beta(q)_{m-q,p}^{m,n}\beta(1)_{i,j}^{m-q,p}\\\gamma(q+1)_i^{m,n}&=\gamma(q)_i^{m,n}+\sum_{p=1}^{l_{m-q}}\beta(q)_{m-q,p}^{m,n}\gamma(1)_i^{m-q,p}.\end{aligned} \tag{2.31}$$

So, by induction, we conclude that (2.27) is valid for $1\le q\le m-1$. Thus, substituting q by its maximum, we have:

$$\begin{aligned}W_{m,n}^h=&\sum_{w_{1,1},\ldots,w_{h,l_h}}w_{m,n}\prod_{k=1}^{h}P(w_{k,1},\ldots,w_{k,l_k}|w_{1,1},\ldots,w_{k-1,l_{k-1}},x_1,\ldots,x_{k-1})=\\&\sum_{w_{1,1},\ldots,w_{1,l_1}}P(w_{1,1},\ldots,w_{1,l_1})\times\left[\alpha(m-1)^{m,n}+\sum_{j=1}^{l_1}\beta(m-1)_{1,j}^{m,n}w_{1,j}+\sum_{i=1}^{m-1}\gamma(m-1)_i^{m,n}x_i\right],\end{aligned} \tag{2.32}$$

which reduces to

$$W^h_{m,n} = \sum_{w_{1,1},\ldots,w_{h,l_h}} w_{m,n} \prod_{k=1}^{h} P(w_{k,1},\ldots,w_{k,l_k}|w_{1,1},\ldots,w_{k-1,l_{k-1}},x_1,\ldots,x_{k-1}) = \\ \alpha(m-1)^{m,n} + \sum_{j=1}^{l_1} \beta(m-1)^{m,n}_{1,j} E(w_{1,j}) + \sum_{i=1}^{m-1} \gamma(m-1)^{m,n}_{i} x_i. \tag{2.33}$$

Since $q \geq 1$, equation (2.33) is valid for $m \geq 2$. If $m = 1$, equation (2.23) reduces to $E(w_{1,n})$. Considering this and substituting (2.33) into (2.22), we have

$$E(y_h|\hat{x}_1,\ldots,\hat{x}_h) = \alpha^{y_h} + \sum_{j=1}^{l_1} \beta^{y_h}_{1,j} E(w_{1,j}) + \sum_{i=1}^{h} \gamma^{y_h}_i x_i \\ + \sum_{m=2}^{h} \sum_{n=1}^{l_m} \beta^{y_h}_{m,n} \left[\alpha(m-1)^{m,n} + \sum_{j=1}^{l_1} \beta(m-1)^{m,n}_{1,j} E(w_{1,j}) + \sum_{i=1}^{m-1} \gamma(m-1)^{m,n}_{i} x_i \right], \tag{2.34}$$

which may be rearranged[1] in the following way:

$$E(y_h|\hat{x}_1,\ldots,\hat{x}_h) = \alpha^{y_h} + \sum_{m=2}^{h} \sum_{n=1}^{l_m} \beta^{y_h}_{m,n} \alpha(m-1)^{m,n} \\ + \sum_{j=1}^{l_1} \left[\beta^{y_h}_{1,j} + \sum_{m=2}^{h} \sum_{n=1}^{l_m} \beta^{y_h}_{m,n} \beta(m-1)^{m,n}_{1,j} \right] E(w_{1,j}) \\ + \sum_{i=1}^{h-1} \left[\gamma^{y_h}_i + \sum_{m=i+1}^{h} \sum_{n=1}^{l_m} \beta^{y_h}_{m,n} \gamma(m-1)^{m,n}_{i} \right] x_i + \gamma^{y_h}_h x_h, \tag{2.35}$$

where α^{y_h}, $\beta^{y_h}_{m,n}$ and $\gamma^{y_h}_m$ are given by (2.21) and $\alpha(m-1)^{m,n}$, $\beta(m-1)^{m,n}_{1,j}$ e $\gamma(m-1)^{m,n}_{i}$ are recursively given by (2.31). □

So, we may use OLS regressions to estimate (2.21) and (2.26) and plug these results into (2.35). Notice that in (2.35), the control variable is a vector $x = (x_1,\ldots,x_h)$. So, it is meaningless to ask about the average response to a unit variation in x. We may ask about the response to a *plan* $(x_1,\ldots,x_h)$. For example, we may ask about the response to a unitary shock in x_1 and zero shocks from x_2 to x_h. Another interesting possibility is to ask about the sequence $(x_1,\ldots,x_h)$ which maximizes (or minimizes) response variation. Another possibility is to find an optimal plan, for some optimality criterion. Finally, we supposed x continuous, but proposition 1 may be easily adapted to discrete case.

Now, consider the model:

$$\begin{aligned} x_t &= x_{t-1} + \epsilon^x_t \\ y_t &= \frac{1}{\sum_{i=0}^{8} \exp[-(-2+i/2)^2]} \sum_{i=0}^{8} e^{-(-2+i/2)^2} e_{t-i} \\ z_t &= x_t + y_t, \end{aligned} \tag{2.36}$$

where shocks ϵ^x_t are given by:

$$\epsilon^x_t \sim N\left(0, 0.2^2\right) \tag{2.37}$$

[1] In deducing (2.35), we applied the following summation inversion formula:

$$\sum_{m=2}^{h} \sum_{i=1}^{m-1} f(m,i) = \sum_{i=1}^{h-1} \sum_{m=i+1}^{h} f(m,i).$$

The proof is easy using Iverson brackets.

and policy shocks e_t are given by:

$$e_t \sim N\left[\frac{-1}{140}\sum_{i=1}^{7} i^2 \Delta z_{t+i-8}, \frac{1}{6}\sum_{i=1}^{7}\left(\Delta z_{t+i-8} - \frac{1}{7}\sum_{i=1}^{t}\Delta z_{t+i-8}\right)^2\right]. \tag{2.38}$$

Suppose that only z_t and the policy variable e_t are observable. Also, notice that e_t is endogenous and given by past values of z_t. Indeed, (2.38) is a *stochastic policy rule* that tries to smooth z_t, giving negative shocks to the system if z_t grows too fast and vice versa. A typical realization of this system is shown in Figure 20.

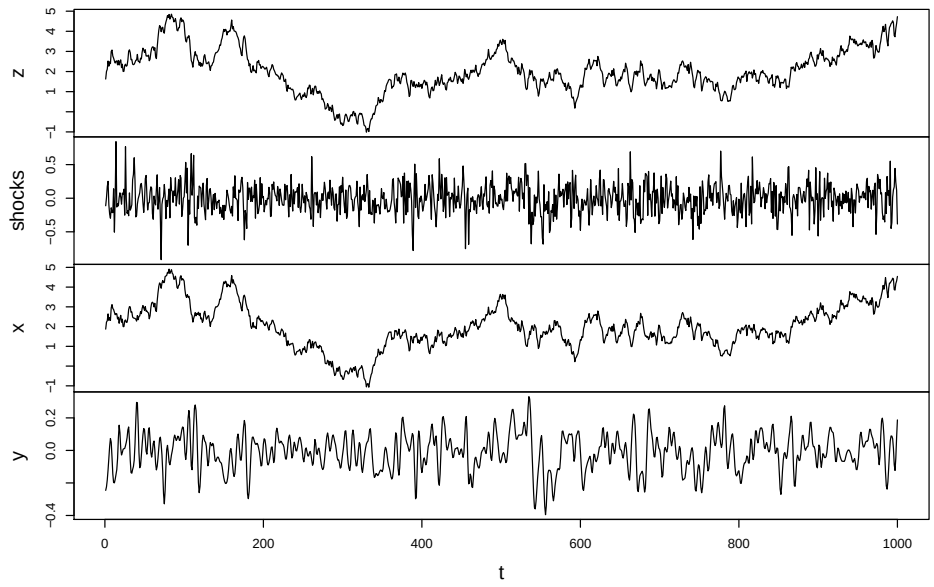

Figure 20 – Size 1,000 sample simulation of process (2.36). Burn-in period is 100 time samples.

Notice that long run movements are driven by random walk x_t while short run deviations are driven by stochastic policy e_t, called shocks in Figure 20. A DAG representing part of this model is given in Figure 21. We do not represent all connections because they are not relevant for our argument. The main point of Figure 21 is that setting $X_{k+i} = e_{t+i}$ and $W_t = Z_{t-1}$ in Theorem 3, the conditions for G-identifiability are fulfilled and our results are directly applicable.

The expected generalized counterfactual impulse response function (EGCIRF) for this system between interventions $do(e_t = 1)$ and $do(e_t = 0)$ is given by

$$EGCIRF_Z^{1,0}(h) = E[Z_{t+h}|do(e_t = 1)] - E[Z_{t+h}|do(e_t = 0)], \tag{2.39}$$

which raises some problems. Actually, Z_t is clearly non-stationary, a fact that may be inferred from Figure 20 and unit roots tests. So, equation (2.39) does not makes sense, since Z_t does not have a well defined expected value. A common approach to overcome this is to take the first difference of the series. But this raises other problems. Even if the identification of causal effects is not affected, we will measure the effect on the variation of Z over one single period, which is probably a weak effect very difficult to estimate without a lot of data. Also, generally this is not the effect of interest for policy makers. A more meaningful measure in terms of policy and statistics is the total variation over the horizon h, $\Delta_h Z_t = Z_{t+h} - Z_t$, which has a well defined expected value[2] for

[2] This can be easily validated through stationarity tests and also by the properties of random walks.

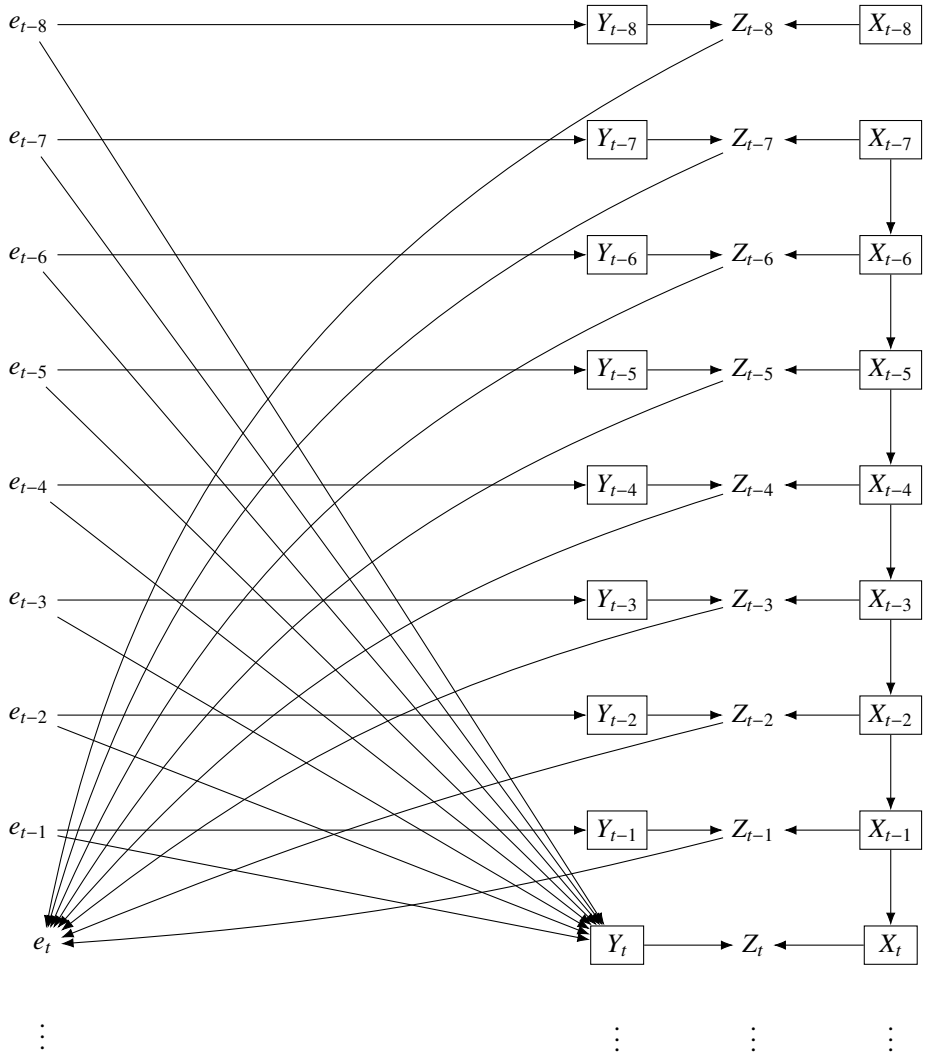

Figure 21 – Dynamic causal network representing relevant aspects of the dynamic model in (2.36).

each t. Also, the presence of Z_t in the response variable does not affect the identification of causal effects in this case. So, the EGCIRF of interest is

$$EGCIRF^{1,0}_{\Delta_h Z}(h) = E[\Delta_h Z_t | do(e_t = 1)] - E[\Delta_h Z_t | do(e_t = 0)], \tag{2.40}$$

which can be simulated as before, and results are shown in Figure 22.

A remarkable feature of Figure 22 is the oscillatory pattern shown by $\Delta_h Z_t$ after a unitary shock $do(e_t = 1)$ compared with a $do(e_t = 0)$ shock. This is due to the endogeneity of e_t. Since we are considering here only interventions at t, future shocks are free to vary. It is somewhat like a shock in traditional econometric sense, such as a Taylor rule. The policy variable is shocked at t, but follows its own policy rule afterwards. In our case, a positive shock in e_t causes Z_t to go up gradually. But e_{t+k} responds negatively to increasing $Z_{t+k-1}, Z_{t+k-2}, \ldots$. This negative response in turn decelerates Z, which induces positive responses in e and so on, causing the oscillatory pattern.

Since this model is linear, we may estimate its EGCIRF as before, through equation (2.11). This is shown in Figure 23. The most salient feature here, besides the unbiasedness of the estimates, is the increasingly less precise estimation as the horizon h goes larger. This is so because the model is non-stationary, and the variance of $\Delta_h Z_t$ raises linearly with h.

A common type of impulse response in econometric literature is the traditional IRF of Definition 9. In

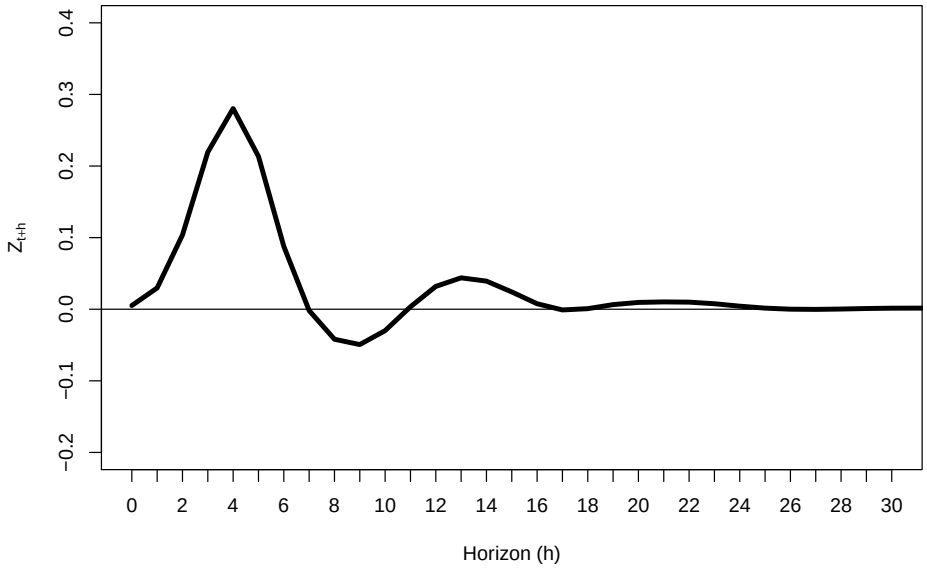

Figure 22 – EGCIRF over $\Delta_h Z_t$ for model (2.36) with intervention $do(e_t = 1)$ against $do(e_t = 0)$.

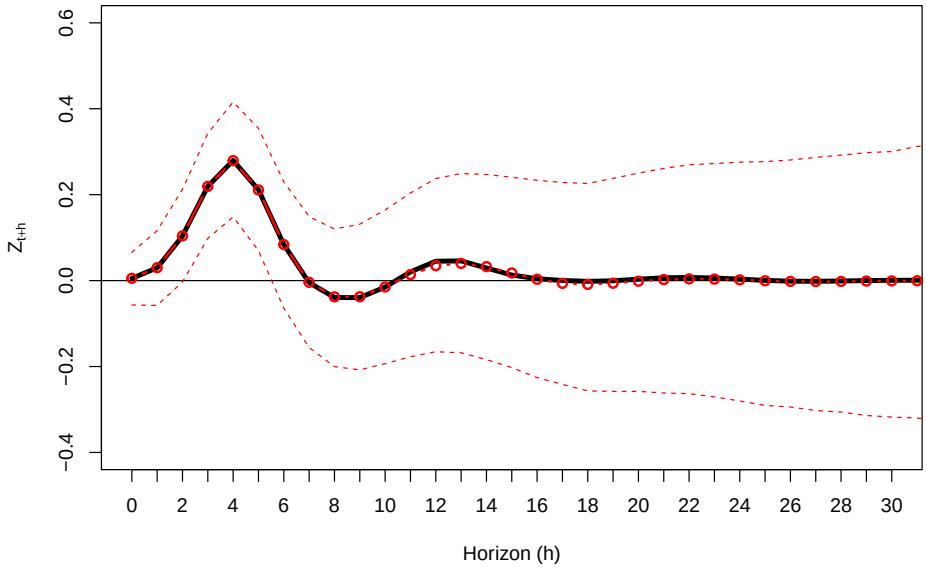

Figure 23 – Thick black line is the EGCIRF for model (2.36) for $do(e_t = 1)$ against $do(e_t = 0)$. Red dotted dashed line is the mean of estimates based on Equation 2.40. Red dashed lines represent percentiles 2,5% and 97,5%. Estimates are based on 10,000 model simulations with sample size 1,000 each after a burn-in period of 100.

our counterfactual definitions of IRFs, the comparison is made among two hypothetical interventions, leaving *all else equal*, including non-manipulated stochastic shocks. So, for linear models with *additive zero mean independent* shocks, set future shocks to zero is immaterial, since all cancel out and, because of independence, a manipulation of a shock at time t does not affect any future shock. This is not the case for the model defined by equations (2.36), (2.37) and (2.38), where policy shocks are endogenous. Thus, set future shocks in this model have a non trivial effect over its response.

Lets evaluate the EGCIRF for this model between interventions $do(X_t = 1, X_{t+1} = 0, \ldots, X_{t+h} = 0)$ and $do(X_t = 0, X_{t+1} = 0, \ldots, X_{t+h} = 0)$. We call its general form the expected counterfactual difference sequential plan response (ECDSP):

Definition 15. *The **expected counterfactual difference sequential plan response (ECDSP)** of a dynamic causal system between interventions* $\hat{x}^c = do(X_t = x_0^c, X_{t+1} = x_1^c, \ldots, X_{t+h} = x_h^c)$ *and* $\hat{x} = do(X_t = x_0, X_{t+1} = x_1, \ldots, X_{t+h} = x_h)$ *is given by*

$$ECDSP_Y^{x^c,x}(h) \equiv E[Y_{t+h}^{x^c}(\Omega_{t-1}, E_{t,h}) - Y_{t+h}^{x}(\Omega_{t-1}, E_{t,h})]. \tag{2.41}$$

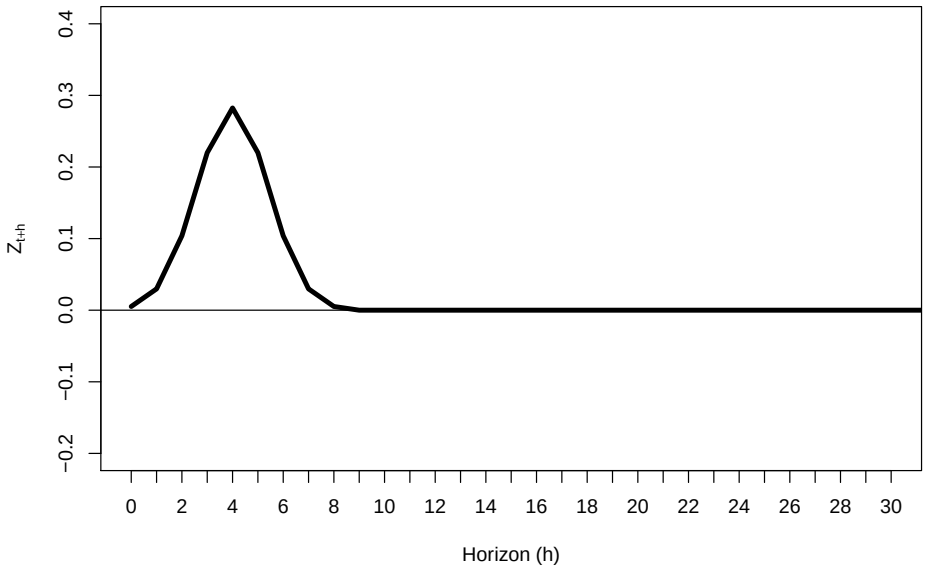

Figure 24 – ECDSP over $\Delta_h Z_t$ for model (2.36) with intervention $do(e_t = 1, \ldots, e_{t+30} = 0)$ against $do(e_t = 0, \ldots, e_{t+30} = 0)$.

Figure 24 shows the ECDSP for $\Delta_h Z_t$ in model (2.36) for interventions $do(e_t = 1, e_{t+1} = 0, \ldots, e_{t+30} = 0)$ and $do(e_t = 0, e_{t+1} = 0, \ldots, e_{t+30} = 0)$. Notice that this figure does not show the oscillatory pattern present in Figure 22. This is so because setting e_{t+k} for all k from zero to 30 "destroys" the autocorrelated structure of e_t, forcing all policy shocks to predetermined values.

The estimation of this impulse response from observational data is more cumbersome than previous examples, but can be done with the help of Theorem 3 or, more generally, through the rules of do-calculus. Here we just apply our Proposition 1, derived from Theorem 3, which fits nicely on linear models.

First, notice that, as before

$$ECDSP_{\Delta_h Z}^{e^c,e}(h) = E[\Delta_h Z_t | do(e^c)] - E[\Delta_h Z_t | do(e)], \tag{2.42}$$

where $e^c = (e_t = 1, e_{t+1} = 0, \ldots, e_{t+30} = 0)$ and $e = (e_t = 0, e_{t+1} = 0, \ldots, e_{t+30} = 0)$. The expected values in equation (2.42) can be estimated with the help of Proposition 1. It is easy to see from Figure 21 that conditions of Theorem 3 are fulfilled by all Z_{t+h} and e_{t+k}, and so the proposition applies. Results are shown in Figure 25.

The results are almost unbiased, the variance grows as before, and the oscillatory pattern is not present in the estimation, as expected.

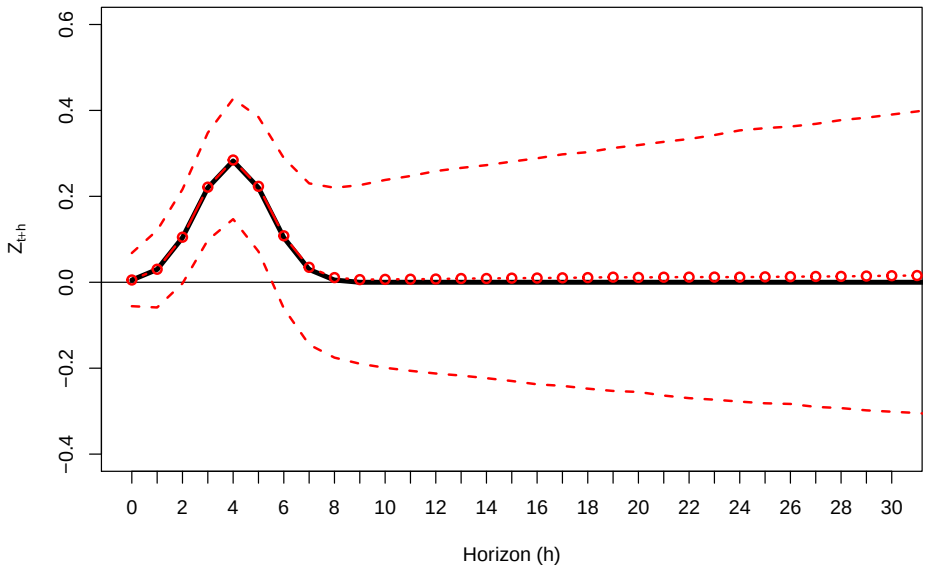

Figure 25 – Thick black line is the ECDSP over $\Delta_h Z_t$ for model (2.36) with intervention $do(e_t = 1, \ldots, e_{t+30} = 0)$ against $do(e_t = 0, \ldots, e_{t+30} = 0)$. Red dotted dashed line is the mean of estimates based on Proposition 1. Red dashed lines represent percentiles 2,5% and 97,5%. Estimates are based on 10,000 model simulations with sample size 1,000 each after a burn-in period of 100.

2.5 The Lucas critique

Here we briefly discuss the implications of Lucas critique for our approach and expose our opinion about traditional approaches as well.

Throughout 1970s, the United States experienced some of its highest levels of inflation along with elevated rates of unemployment. These facts defied the concept of Phillips Curve as well as Keynesian macroeconomic theory in general. Robert Emerson Lucas Jr. argued, in his most famous paper (LUCAS, 1976), that traditional macroeconometric approach at the time was fundamentally flawed with respect to policy analysis, despite its relative success in forecasting economic time series (LUCAS, 1976; GOUTSMEDT et al., 2015). Lucas received the Nobel Memorial Prize in Economic Sciences in 1995, mainly for his research on rational expectations theory and economic policy, whose fundamentals are present in Lucas (1976) [3].

According to Goutsmedt et al. (2015), Lucas targeted Klein and Goldberger (1955) and Tinbergen (1952) models. Lucas (1976) claims that traditional macroeconometric approach ignored that changes in economic policy *regimes* also change agents behavior. This, in turn, shifts parameter values that are not "structural".

[3] Advanced information. NobelPrize.org. Nobel Media AB 2019. Mon. 30 Sep 2019. <https://www.nobelprize.org/prizes/economic-sciences/1995/advanced-information/>

Here, some clarifications are necessary, because these concepts are not always clear, and are subject to debates and different interpretations (CHARI; KEHOE; MCGRATTAN, 2009; GOUTSMEDT et al., 2015; SERGI, 2018). By *economic policy* we mean some sort of intervention, usually by a government, in the economy. An *economic policy regime* is a set of *rules* which in some way determines economic policy. These rules may be deterministic or stochastic and not necessarily consciously followed by the policy maker. Examples of economic policies are monetary policy and fiscal policy. Examples of policy regimes are the gold standard for monetary policy or fixed exchange rates on currency markets.

We say that x, which may be possibly a set, is a *policy instrument* or *policy variable* if it is the economic variable available for *direct manipulation* by policy makers in pursuing their goals. The variable x is manipulated according to policy rule to achieve policy objectives. Notice the causal language in this setting, with reference to variable manipulation, and also a somewhat paradoxical concept of regime, where rules determine x manipulation. This is not a contradiction, since policy makers may behave as if they are following rules, an idea which is behind Taylor (1993) conclusions.

We agree that Lucas (1976) conclusions are serious and possibly relevant, but further developments left the impression that there is only one way to overcome difficulties raised by his arguments. The conclusions about bad econometric practices in his age are right, but the broadly adopted microfounded models incorporating rational expectations are just one possible way to try overcome these difficulties, being not absent of its own problems. Another possibility, advocated here by us, is econometric modeling based on causal models, which is, of course, not absent of difficulties, but may assist or even substitute (in simple queries) the development of microfounded models, be they DSGEs, ABMs[4] or other sort of microfounded model.

It is fair to mention that Lucas cannot be guilt of what has been made from his ideas. Quoting himself:

> My paper, “Econometric Policy Evaluation: A Critique” was written in the early 70s. Its main content was a criticism of specific econometric models — models that I had grown up with and had used in my own work. [...] But the term “Lucas critique” has survived, long after that original context has disappeared. It has a life of its own and means different things to different people. Sometimes it is used like a cross you are supposed to use to hold off vampires: Just waving it an opponent defeats him. Too much of this, no matter what side you are on, becomes just name calling. (LUCAS et al., 2012)

Goutsmedt et al. (2015) interpret Lucas (1976) as a *positive* and as a *prescriptive* statement. The former is an effort to explain the stagflation of the 1970s in United States, while the latter is a methodological prescription for macroeconometricians. Here we focus on the prescriptive statement.

The description of an economic system in Lucas (1976) is made by a difference equation as

$$y_{t+1} = F(y_t, x_t, \theta(\lambda), \epsilon_t), \tag{2.43}$$

where y_t is a vector of state variables, x_t is a vector of *forcing* policy disturbances, ϵ_t is a vector of exogenous shocks and $\theta(\lambda)$ are parameters of F which are functions of policy parameters λ.

Policy is a stochastically distributed function of the state of the system:

$$x_t = G(y_t, \lambda, \eta_t), \tag{2.44}$$

where λ are parameters of G, and η_t are exogenous shocks. Equations (2.43) and (2.44) closely resembles functional equations described as structural causal models in Chapter 1. In Lucas (1976), these equations

[4] ABM - Agent-based models.

does not represent, necessarily, causal relations, but if we have a causal model represented by (2.43) and (2.44), his critique applies to our approach as well. Remembering dynamic causal models defined in Chapter 1, one basic premise is the invariability of F and G, which is not maintained with a non constant θ.

One possible way out this conundrum in causal models is to add λ as a variable of the system. To see why, notice that implicit in Lucas' argument is the assumption that F and G are fixed given θ and λ, respectively. Since θ is a function of λ, only the latter have substantive causal content. So, equations (2.45) and (2.46) below can be seen as representing a SCM:

$$y_{t+1} = H(y_t, x_t, \lambda, \epsilon_t) \tag{2.45}$$

and

$$x_t = G(y_t, \lambda, \eta_t). \tag{2.46}$$

where F is absorbed into H incorporating the function $\theta(\cdot)$. This can be represented as the diagram in Figure 26. To make things simple, we assumed no unobserved confounding, not important for our argument. We also

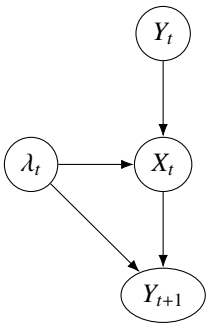

Figure 26 – Graph representing equations (2.45) and (2.46).

explicitly set λ as time varying. Figure 26 shows that Lucas critique can be seen as a confounding problem. If λ is observable, there is hope for identification. But, since λ represent parameters of a function that describes the economy, there is little hope to observe it. However, we may argue that λ represents the behavior of policy makers, and is caused by other variables such as different central bank chairmans, or alternative monetary policy regimes, for example. Representing these variables as C_t, the diagram is now that of Figure 27.

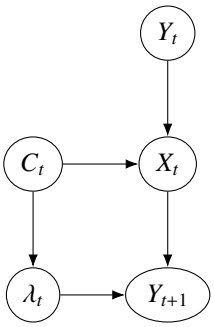

Figure 27 – Graph representing equations (2.45) and (2.46) with a policy behavior variable C_t causing λ_t and X_t.

So, in principle, controlling for C_t solves our problem. But, Lucas critique is more than this. In the context of our example, he argues that economic agents may anticipate changes in C_t, changing their behavior accordingly. From the econometrist's viewpoint, this may be problematic.

Suppose that C_t is constant in all its history, but will change from now on and economic agents know it. A possibility is an announced change in policy regime. Without variation in C_t, there is no room for statistical

inference, even if we know that C_t will change and economic agents also adapt behavior. This is the basic argument in favor of microfounded models, where parameters represent optimizing behavior and so on.

Coming back to Lucas (1976), he argues that interest must lie on estimating $\theta(\lambda)$, and our problem of C_t variation boils down to λ variation. Again, $\theta(\lambda)$ can not be estimated only from data if λ does not vary sufficiently. In this situation, an estimated model is immune to Lucas critique only if it makes assumptions beyond information contained in data alone. We stressed before that some causal assumptions are necessary for causal identification on the majority of situations where we face observational data, but this is valid for other extra data information too.

So, hardly a model estimated from data, especially aggregated data, can avoid Lucas critique in the case of an anticipated change in policy regime never observed. This is valid for our approach and virtually all macroeconometric approaches, even microfounded ones, because these are estimated from aggregate data, which does not inform about λ variations. But, if λ varies across samples, there is hope for controlling for the right variables, or make estimations on only selected time periods to see if there is significant change in policy makers or economic agents behavior.

3 Causal effects of monetary policy: The case of Brazil

"Myth No. 1: Monetary policy causes booms and busts. ..."

— Edward C. Prescott

This chapter presents results about causal effects of monetary policy in Brazil. The main point here is to show simple practical applications of previously developed techniques to a classical policy evaluation problem in macroeconomics.

3.1 Identifying causal effects of monetary policy in Brazil

The analysis of monetary policy in Brazil is specially suited for our method because this country adopted an inflation target regime with explicit target values for inflation (IPCA) and overnight interest rate (Selic rate) in mid-1999 (BOGDANSKI; TOMBINI; WERLANG, 2000), with Selic rate as the main policy instrument. So, we have an explicit manipulation of a variable (the overnight interest rate) and want to evaluate its causal effects.

To do so, we use the EGCIRF defined in Chapter 2. Control for confounding is essential in this context. Past inflation influences central bank decisions and also affects future inflation through inertial mechanisms, for example. Other potential confounders for inflation are expectations and real activity. Actually, several macroeconomic variables influences both, central bank decisions and future inflation, and are confounders for our problem.

Fortunately, central bankers are not omniscient beings, and must base their decisions on observable data and their own expert judgment. So, we take advantage of the inflation reports issued by Banco Central do Brasil (BCB) since the adoption of inflation targeting to take clues and data to control for direct causes of Selic rate variations, and so identify causal effects of these variations on economic aggregates.

An approach close to ours, in the spirit of causal analysis of monetary policy, is Angrist, Jordà and Kuersteiner (2018). In this work the authors try to infer causal effects of monetary policy using US data, but since their goal is to evaluate non-linear effects, they use propensity score matching for inference. Our approach resembles the one in Romer and Romer (2004). Their method regresses intended changes on fed funds rate by FOMC, inferred from the *Weekly Report of the Manager of Open Market Operations*, against internal forecasts of the Federal Reserve on inflation and real activity, extracted from the "Greenbook", and then take the residuals of this regression as a measure of monetary policy shocks. Their causal reasoning is clear: if we admit intended changes in fed funds rate as actions of FOMC and control these actions for confounders that are immediate causes of FOMC intentions, the residuals are, we hope, free of counfounding effects, being a suitable measure of exogenous monetary policy shocks.

Our goal is easier, since Selic rate is strictly manipulated and its target publicly available, freeing us from inferring intended funds rate from reports, a potential source of inaccuracies. Moreover, we regress the response variable of interest, inflation for example, directly against Selic rate and confounders composed of BCB forecasts and other relevant information available on inflation reports. The relation to Romer and Romer (2004) becomes clearer if we remember that regress the response against residuals of the regression of Selic rate on confounders, like in Romer and Romer (2004), is the same as our approach (in linear models) by the Frisch-Waugh-Lovell theorem (FRISCH; WAUGH, 1933; LOVELL, 1963; HAYASHI, 2000). Moreover, to

do things in our way facilitates regression diagnostics, confidence interval computations and also non-linear expansions in a more direct and general way than Romer and Romer (2004) approach.

The causal diagram representing our identification strategy is shown in Figure 28. The main idea is that whatever variables in economy affects response and central bank decisions, the latter is always done through some picture of reality mediated by observable data and possibly unobservable judgment. So, these are the parents of Selic rate decision in our causal diagram. Assuming that possible judgment bias remains constant on average along all sample period, conditioning on observable data used by central bankers for decision making is sufficient for causal effects identification by the back-door criterion. Also, regressions are done for each horizon h after meeting time t exactly like the various causal impulse responses presented in Chapter 2.

One word about judgment bias. They are represented in SCMs by exogenous variables and/or functions f_i in Definition 1. A systematic change in policy decision behavior represents a change on these mechanisms and are related to Lucas critique, as explained in Section 2.5. Here we will assume no systematic change in policy behavior both for simplicity and weak empirical evidence, but we recognize the possibility of distinct regimes, specially under different BCB governors.

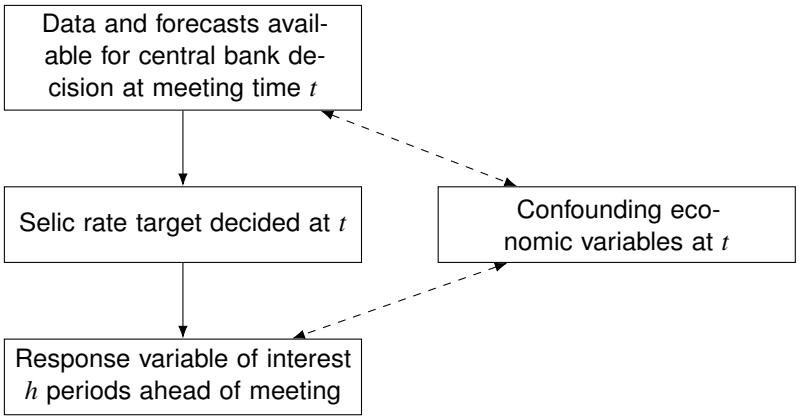

Figure 28 – Graph representing manipulation of Selic rate by Banco Central do Brasil.

3.2 Data

Before showing which series are used for inference, we explain how data is organized for estimations. The method regresses response variables h periods ahead meeting time t against policy instrument and available data for policy makers at t. Since meetings are not regularly spaced along time, some care must be taken. Suppose the response variable is Y_{t+h}, the policy variable at meeting time t is X_t, and data available for policy decision is C_t. Notice that C_t may contain variables realized before t, like the last available inflation index and its lags, for example. Then, for response h periods ahead, we regress Y_{t+h} against X_t and C_t. Notice that h is equally spaced over time, representing h months ahead, for example. It does not coincide, necessarily, with meeting frequency, which actually change over time. Data were also interpolated to coincide with meeting days. More details in Appendix B.

That being said, we have chosen suitable variables extracted mainly from BCB inflation reports, common macroeconomic aggregates and market projections collected by the Focus survey carried out by BCB. Since we control for parents of Selic rate in Figure 28, all specifications are the same, independent of the response. So, after choosing which direct causes control for, these same regressors may be used for the estimation of *any causal effect of Selic rate manipulation*, provided that all premises of the causal model and adequate regularity conditions are fulfilled. The core specification for regressors is given in Tables 6 and 7.

Variables in Tables 6 and 7 are not “cherry-picked” as might look at first glance. They are reminiscent of a selection process over a wider set of variables. We choose a set of meaningful observable determinants of BCB decisions, computed the causal impulse responses of interest, and proceeded on a process of variable exclusion in a way that minimally affected the causal impulse responses. The rationale behind this procedure is the strong correlations among regressors, because of redundant information carried by them. This correlation is not particularly important, since the only coefficient of interest is the one for policy variable, but less variables means more degrees of freedom and more precise or, at least, more reliable estimates. All coefficients but the policy variable are there just for conditioning, and almost all does not have any causal interpretation. This is natural, since is fairly intuitive that BCB internal forecasts, for example, does not have a *direct* causal impact on response variables such as inflation or GDP. Their causal impact is all mediated by the policy variable, as is clear from diagram in Figure 28[1].

Another important practical aspect is the stationarity of all variables involved. For a regression to represent an estimate of a conditional expectation, some regularity conditions must be fulfilled. And this is very important, because we may use OLS for almost any data we put in a computer, but if your conditional expectation does not exist, the results are meaningless (see discussion about Z_t non stationarity in Section 2.4). Also, since we are dealing with time series data, ergodicity is a main concern (HAMILTON, 1994), since this property guarantees that temporal averages converge to ensemble averages asymptotically in time dimension. So, non-stationary variables like GDP expectations have to be transformed, taking differences, for example. Taking only first differences monthly may not be sufficient, because policy makers may look for longer horizons, like annual, semiannual or quarterly variations. Also, annual variations may behave like non-stationary data even if it is stationary in the long run, because of short sample. So, we use some artifices on data differentiation and transformations to maintain stationarity without losing too much information on the process. These artifices are quite common in the literature, but may cause collinearity problems, and so we proceed to remove some collinear variables as described before. More details in Appendix C.

Finally, regression residuals are autocorrelated mainly because response variables are autocorrelated, a common feature in time series regression. This is unavoidable, since our regressions only have their causal meaning specified as we do. Certainly, for some response variable Y at $t+h$, if we control for Y_{t+h-1}, the regression fit would be better, but its causal meaning is lost. To see why, think about the causal path linking the policy variable S_t to Y_{t+h}. If this causal link exists, so we probably also have a causal link from S_t to Y_{t+h-1}, and controlling for this last variable would block the causal link $S_t \to Y_{t+h-1} \to Y_{t+h}$, invalidating our estimates of total causal effects. So, we use Newey-West HAC estimators to compute confidence intervals (NEWEY; WEST, 1986; BIERENS, 1996; HAMILTON, 1994).

3.3 Main results

From Figure 28 it is clear that if we control for direct determinants of Selic rate, we have its causal effects by the back-door criterion. So, we proceed as in Chapter 2 and compute EGCIRFs for the following response variables: real GDP gap, 3 months Selic rate variation, 12 months IPCA and real US dollar total percent variation from t to $t+h$, where h is the horizon of the EGCIRF. The policy variable is Selic rate variation, the counterfactual regimes are a 25 basis points (0.25 p.p.) uprise against no variation and the covariates chosen are those in Tables 6 and 7. The real GDP gap is computed by the HP filter in the usual way, and all

[1] These observations highlight the main difference between our procedure and the traditional structural equations estimation carried over in econometrics. Our regression equations are NOT structural. They represent *conditional expectations*, not mechanisms of variable determination. But, behind its justification there is a Structural Causal Model, pictured in a simplified way by the DAG in Figure 28. The economic mechanisms are hidden in the confounding economic variables at t, but we may avoid modeling these mechanisms, because in our problem we can block the back-door paths from policy variable to response variables controlling for direct causes of the policy variable. So, besides our equations are not structural in the mechanistic sense, the coefficient of the policy variable *does have a causal meaning*, made clear by the SCM approach.

Regressor	Description	Source
S	Target Selic rate variation. This is the policy variable, determined on each COPOM meeting. Its coefficient is the causal effect of interest.	BCB - Copom Meetings
R_1	12 months difference on target Selic rate	BCB - SGS
R_2	Last 6 months variation of inflation index	BCB - SGS
R_3	Last 12 months variation of inflation index	BCB - SGS
R_4	Median of Focus IPCA 12 months ahead projections - deseasonalized	BCB - Market Expectations System
R_5	Difference between reference scenario IPCA forecast by BCB for meeting day and 12 months accumulated IPCA	BCB - Inflation Reports
R_6	Difference of reference scenario IPCA forecast by BCB for 6 months ahead and BCB forecast for 3 months ahead both adjusted for inflation target	BCB - Inflation Reports
R_7	Difference of reference scenario IPCA forecast by BCB for 9 months ahead and BCB forecast for 6 months ahead both adjusted for inflation target	BCB - Inflation Reports
R_8	Difference of reference scenario IPCA forecast by BCB for 12 months ahead and BCB forecast for 9 months ahead both adjusted for inflation target	BCB - Inflation Reports
R_9	Monthly variation of median 12 months ahead Focus Selic rate forecasts	BCB - Market Expectations System
R_{10}	Difference between the median of Focus annualized 1 quarter accumulated GDP forecasts for meeting day and accumulated 12 months GDP	BCB - Market Expectations System
R_{11}	Difference between the median of Focus annualized 1 quarter accumulated GDP forecasts for 3 quarters ahead and accumulated 12 months GDP	BCB - Market Expectations System
R_{12}	Real GDP 3 months log variation	BCB - SGS
R_{13}	Real exchange rate 6 months log variation - dollar	BCB - SGS

Table 6 – Regressors used in main estimates, part I.

Regressor	Description	Source
R_{14}	Focus end of month expected exchange rate variation - US dollar	BCB - Market Expectations System
R_{15}	Focus 6 months expected exchange rate variation - US dollar	BCB - Market Expectations System
R_{16}	Focus 12 months expected exchange rate variation - US dollar	BCB - Market Expectations System
R_{17}	Monthly variation of Focus end of month expected exchange rate variation - US dollar	BCB - Market Expectations System
R_{18}	Monthly variation of Focus 6 months expected exchange rate variation - US dollar	BCB - Market Expectations System
R_{19}	Monthly variation of Focus 12 months expected exchange rate variation - US dollar	BCB - Market Expectations System
R_{20}	Focus expected industrial production 6 months ahead minus 1 year industrial production variation	BCB - Market Expectations System
R_{21}	Focus expected industrial production 9 months ahead minus 1 year industrial production variation	BCB - Market Expectations System
R_{22}	Three months difference of reference scenario IPCA forecast by BCB for meeting day	BCB - Inflation Reports
R_{23}	Three months difference of reference scenario IPCA forecast by BCB for 3 months ahead	BCB - Inflation Reports
R_{24}	Three months difference of reference scenario IPCA forecast by BCB for 12 months ahead	BCB - Inflation Reports
R_{25}	Difference of market scenario IPCA forecast by BCB for 12 months ahead and same BCB forecast for 6 months ahead both adjusted for inflation target	BCB - Inflation Reports

Table 7 – Regressors used in main estimates, part II.

variables are suitably transformed or interpolated to have values at meeting day t and monthly figures from there. We also add as regressors 4 lags of the policy variable and lags 2 to 4 of GDP gap for reasons that will become clear later.

Details on data transformations are in Appendix B. Regressions diagnostics and robustness checks are in Appendix C. The EGCIRFs are shown in Figures 29, 30, 31 and 32.

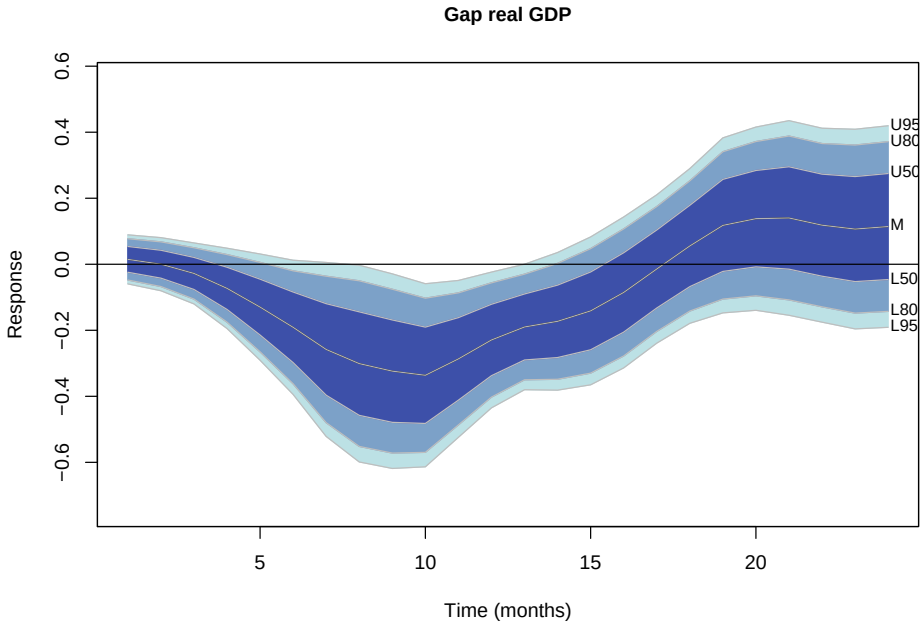

Figure 29 – EGCIRF of real GDP gap for 25 basis points uprise in Selic rate against no variation. This response considers 4 lags of policy variable and lags 2 to 4 of GDP gap as confounders.

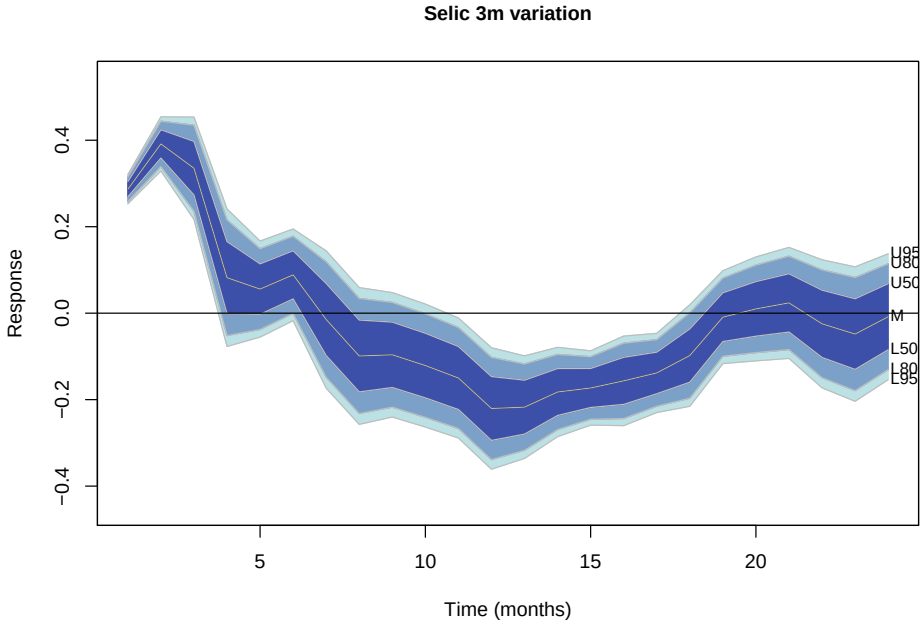

Figure 30 – EGCIRF of 3 months Selic rate variation for 25 basis points uprise in Selic rate against no variation. This response considers 4 lags of policy variable and lags 2 to 4 of GDP gap as confounders.

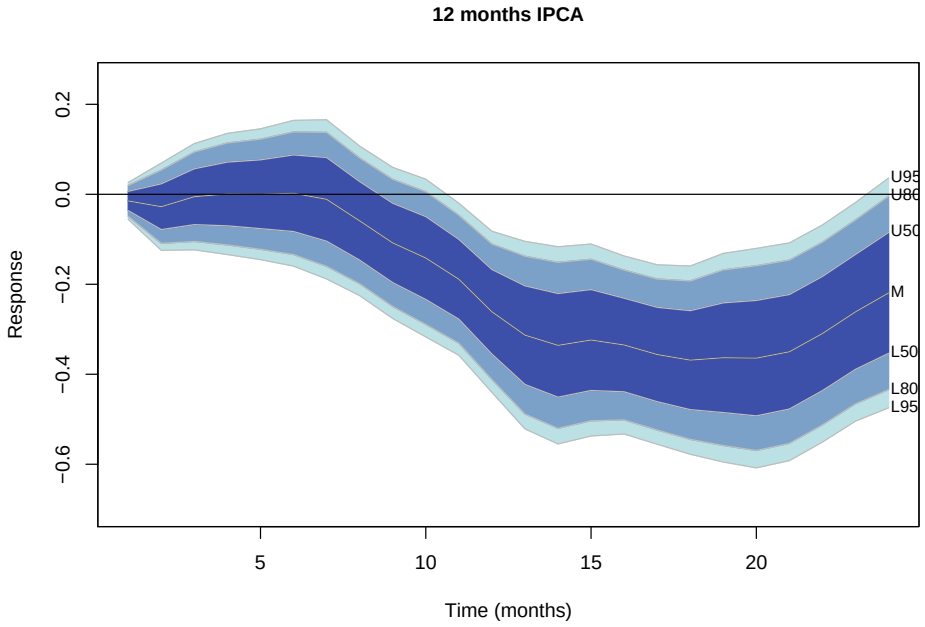

Figure 31 – EGCIRF of 12 months IPCA for 25 basis points uprise in Selic rate against no variation. This response considers 4 lags of policy variable and lags 2 to 4 of GDP gap as confounders.

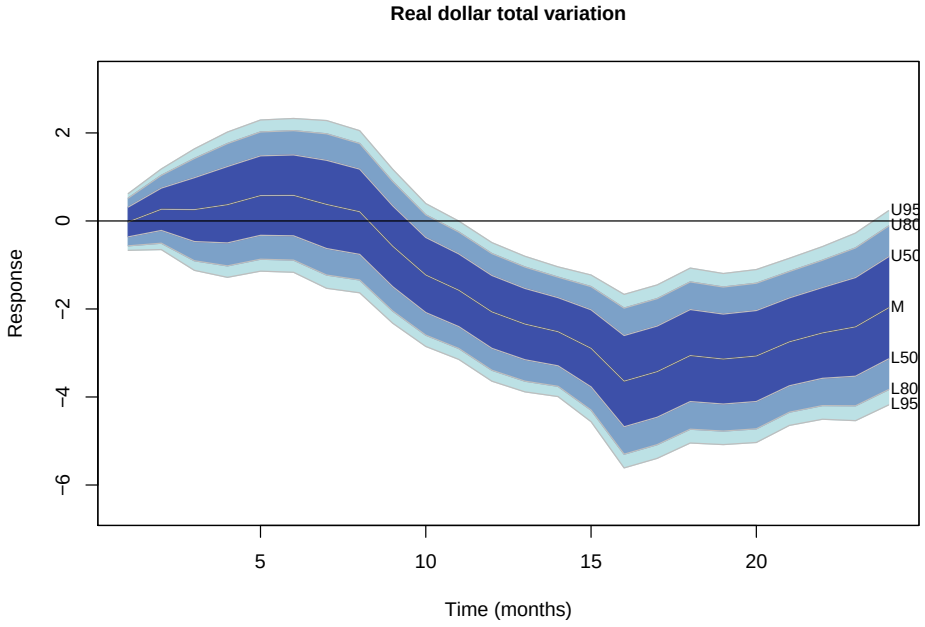

Figure 32 – EGCIRF of real dollar total variation for 25 basis points uprise in Selic rate against no variation. This response considers 4 lags of policy variable and lags 2 to 4 of GDP gap as confounders.

These Figures show the expected behavior from a theoretical viewpoint and also agrees to some degree with findings in the literature. Notice that covariates are the same for all 4 estimates, since we changed only the response variable. Lets analyze them separately.

3.3.1 Real GDP gap

Figure 29 shows the response of output gap in percent to a 25 basis points increase in Selic rate. As expected, this response is negative, reaching its minimum 10 months after the shock, with minimum expected value of -0.37%. Notice that the real GDP gap is hardly observable at t, especially the one computed with HP filter, because this technique suffers from severe end point bias and various other problems (HAMILTON, 2018). However, we may add the gap at $t-2$ to $t-4$ as a proxy to the GDP gap at t, and so we did it. Without these covariates the EGCIRF becomes that of Figure 33.

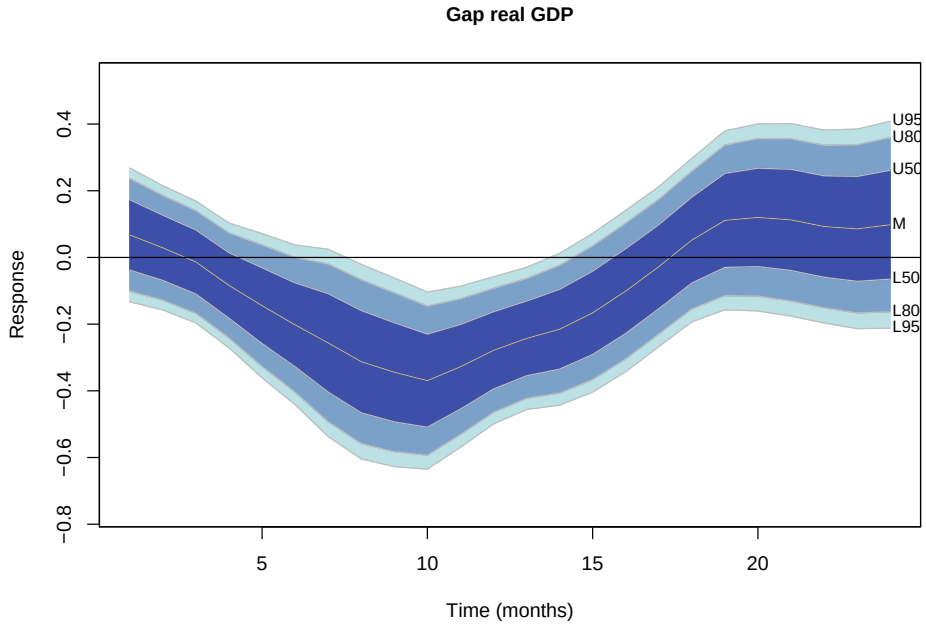

Figure 33 – EGCIRF of real GDP gap for 25 basis points uprise in Selic rate against no variation.

The effect of adding output gap as a covariate is to set closer to zero the effect at t and increase the estimate precision in some horizons close to the shock, having no visible effect on horizons beyond 6 months. Overall, the main conclusions remain intact. Also, the addition of output gap as a covariate have had no effect in all other EGCIRFs presented.

The literature of GDP response to monetary policy shocks in Brazil is comprised mainly by estimations made through VARs, semi-structural models and DSGEs, as expected. Overall, results point to a negative effect over GDP by a contractionary shock, as our estimates, but the size of the effect and its timing vary.

Costa Filho (2017) uses monetary policy shocks inspired by Romer and Romer (2004) and includes these shocks in VARs to compute impulse responses. For GDP, his results point to a maximal effect of -0.5% reached before 5 months horizon, contrasting with our -0.37% in 10 months. But, the majority of impulse responses presented in Costa Filho (2017) are not statistically significant, a common issue in VAR analysis, and other variables present some problems in some specifications, such as significant “price puzzles” on IPCA. The author argues that cost channels of monetary policy in Brazil may explain these findings. We recognize the possibility of these cost channels, but Figure 31 shows no price puzzle. Actually, all our specifications does not show significant price puzzles, but if some key confounders are not controlled for, price puzzles become strong

and significant, showing that misspecification caused by inadequate control of confounding is a main concern on this issue. In Chapter 1 we discuss some pitfalls related to VARs that may explain this recurrent behavior.

Mendonça, Medrano and Sachsida (2010) uses the SVAR with sign restrictions "agnostic" approach presented in Uhlig (2005) to estimate impulse responses. Their results point to 65% of probability of a negative response of GDP after a one standard deviation monetary shock, with 20% probability for negative variations between 0 and -0.5%. They also find a local minimum 10 months after the shock, but their confidence intervals are huge and this is the reason for their analysis to show results as probabilities, since they are not significantly different from zero. The agnostic approach is more free of assumptions, but have its issues, which we commented briefly in Chapter 1.

A semi-structural approach may be found in Minella and Souza-Sobrinho (2013). Their results point to a maximum reduction in GDP around 3 quarters after a 25 basis points per quarter shock, and the size of the effect is around -0.2%. Overall, their results are close to ours, but effects are weaker. There are not confidence bands for precision comparison.

A DSGE approach may be found in Castro et al. (2011). This is the main DSGE model used by BCB in monetary policy analysis, creatively called SAMBA (Stochastic Analytical Model with a Bayesian Approach), and in its original form, their results point to a -0.25% minimal contraction of GDP after a monetary policy shock occurring after roughly 3 quarters, very close to our findings. More recently, in a revision of the SAMBA model (Special Study n.39/2019, issued in Inflation Report Boxes, march 2019), their GDP response is stronger, with minimum around -0.65% in a two quarters horizon.

Results vary greatly, but the message is the same: monetary policy does cause real effects on GDP. We claim that our approach is more easily interpretable, have less critical assumptions, and is more precise except for the DSGE approach, although this one is the more rigid in its assumptions, generating more debatable results. Anyway, we do not believe in silver bullets in science. For us, our approach is a clean way to access meaningful impulse response for policy decisions and can be a reference for more mechanistic models.

3.3.2 3 months Selic rate variation

The next response we analyze is the 3 months Selic rate variation, shown in Figure 30. This is very important for our work because this thesis is mainly focused in methodology and not economic analysis, and this EGCIRF may give us some clues about the adequacy and interpretation of our results. The first noticeable feature of Figure 30 is that, after a positive 25 basis points hike in Selic rate, subsequent shocks remain positive and after roughly seven months they change their sign. Remember from Chapter 2 that the EGCIRF is the response to an exogenous shock at t, but subsequent shocks are free to vary, different of the Sequential Plan Response ECDSP of Section 2.4, where we evaluate the response to a shock at t with subsequent shocks constrained to zero.

So, our results suggest that a contractionary period of monetary policy held by BCB is followed by a period of monetary expansion, much like the impulse response shown in Figure 22, where we only considered an intervention at t, leaving future shocks free to vary. This points to a conservative action of BCB, where interest rates are raised excessively to drive inflation to its target, forcing a period of monetary expansion to accommodate the excess. This behavior predicts an oscillatory pattern in Selic rate, which is indeed the case if we look for historical Selic rate as shown in Figure 34.

Methodologically, an important issue is the response of 3 months Selic variation after 1 month. This value is roughly 25 basis points according to Figure 30, as expected. But if we do not use lags of Selic rate variation as regressors, the result is the one in Figure 35.

Notice that the response after 1 month is roughly 40 basis points in this case, with remaining

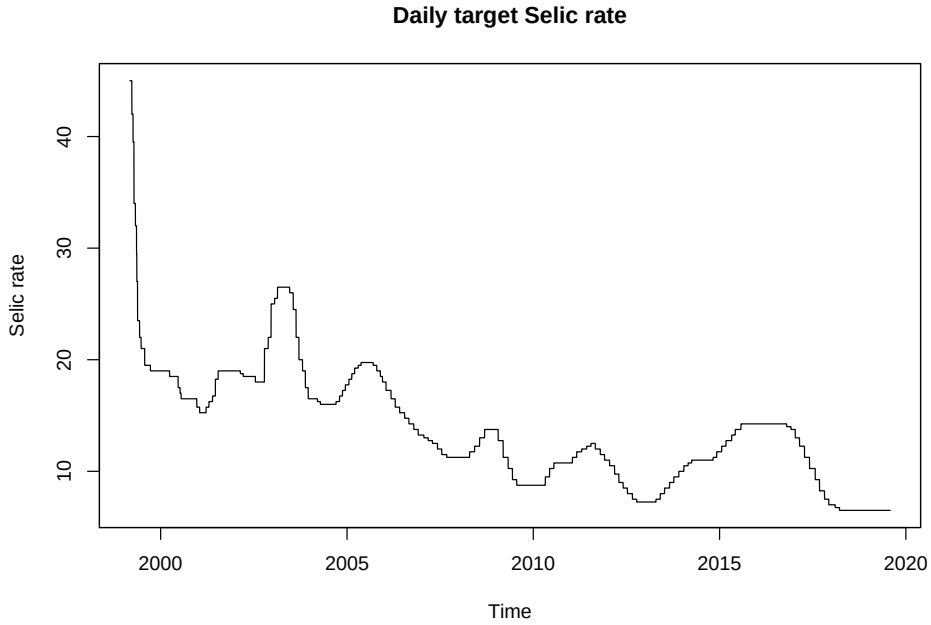

Figure 34 – Daily target Selic rate.

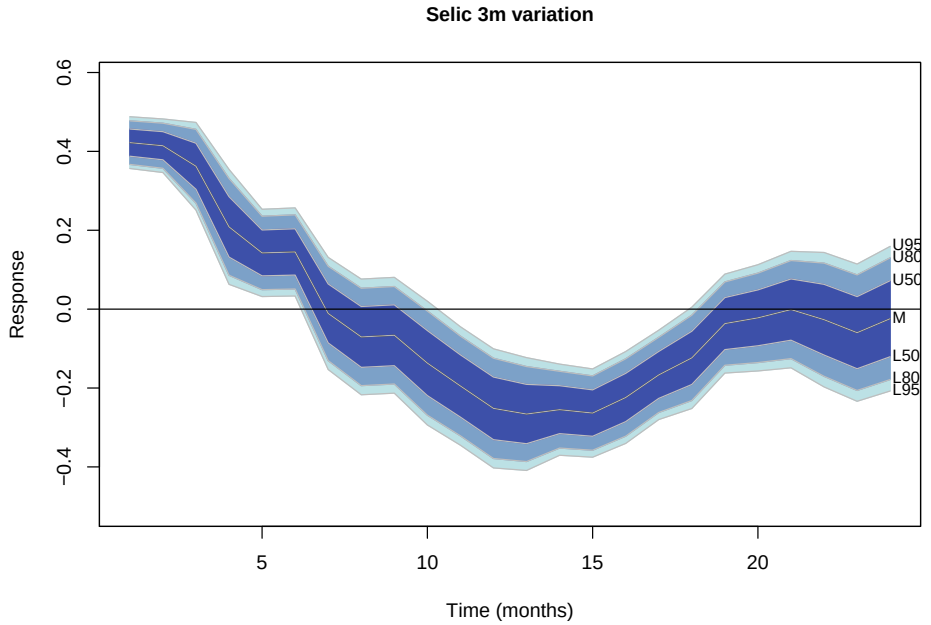

Figure 35 – EGCIRF of 3 months Selic rate variation for 25 basis points uprise in Selic rate against no variation.

periods very close to our main result. This suggests that correlations among policy decision and the already accumulated Selic rate before meeting are being accounted in the impulse response, and so it is not causal, because previous Selic rate variations are acting as confounders. Indeed, Selic rate variation between meetings is strongly autocorrelated. Part of this autocorrelation may be explained by the autocorrelation of variables that determine policy decisions, but our analysis point to a residual "pure" autocorrelation that must be controlled for. We can achieve this adding lags of the policy variable (Selic rate variation). Adding 4 or more lags leads to identical results, and so we choose to add 4 lags.

This autocorrelation in Selic rate variation explainable by more than the autocorrelation of its other determinants suggests that policy makers choose to slowly vary the Selic rate. Moreover, this slow variation is carried out by no other reason than simply make things in a cautious way, acting in cycles of monetary contraction and expansion, clearly visible in Figure 34. This is reasonable and corroborates with human behavior under uncertainty. Comparing with results in the literature, the most noticeable feature is a similar behavior from Selic response found in Minella and Souza-Sobrinho (2013) in terms of timing, because their response change sign after roughly 3 quarters, and also in terms of the intensity of the effect.

3.3.3 12 months accumulated IPCA

Figure 31 shows the EGCIRF for 12 months accumulated IPCA after a 25 basis points uprise in Selic rate against no variation. Some interesting features of this result are: no significant price puzzle and inflation reduction is stronger after real GDP contraction, corroborating conventional wisdom. The maximum effect occurs at roughly 15 to 20 months and has an expected value around -0.42%.

Costa Filho (2017) showed price puzzle for almost all specifications, something we attribute to poor control of confounding. Actually, the traditional price puzzle "solution", as explained by the author, is to control for commodity prices hoping to anticipate future inflation, because it is believed that the puzzle is caused by anticipation of future inflation by central bank, which raises interest rates before inflation rise. Well, in terms of causal models this is typical confounding, resembling the police-crime example in Section 1.2. What happens is that there are some variables which causes more inflation in the future, and looking for those variables, central bankers anticipate inflation hike raising interest rates. Controlling for those variables or its consequences for central bankers, like forecasts indicating more inflation, hopefully vanishes the puzzle. Our results point to this conclusion.

Mendonça, Medrano and Sachsida (2010) point to a 35% probability of a -0,10% fall in IPCA six months after the shock. But, as before, their estimates lacks precision, with most results statistically non-significant.

Minella and Souza-Sobrinho (2013) point to a roughly -0.6% IPCA fall 5 to 6 quarters after shock, and Castro et al. (2011) points to approximately -0.25% IPCA fall after 4 quarters. The 2019 updated SAMBA version, mentioned in Section 3.3.1, points to a roughly -0.5% IPCA fall after 4 quarters.

These results are less variable, and our figures are relatively close to Minella and Souza-Sobrinho (2013), with significant stronger effects over inflation on a longer horizon than other estimates.

3.3.4 Real dollar total variation

This is the response of real exchange rate between currencies Brazilian real and US dollar, measured in reals per dollar adjusted for respective inflation indexes. It is shown in Figure 32. Notice that the appreciation effect is delayed months ahead, something probably related to the delayed effect of Selic rate over IPCA. Indeed, their timing approximately coincide, as can be seen comparing Figures 31 and 32. However, some care must be taken in this comparison, because IPCA is measured as 12 months variation, and real exchange rate is total accumulated since shock. The maximum real appreciation of domestic currency occurs around 16

months after shock and corresponds to a US dollar real total depreciation of -3.9% on average.

The reaction of real exchange rate to monetary contractions is a subject of intense debate in the literature, and puzzles are found for developed and emergent economies. Kohlscheen (2014) discuss some of these issues. Zettelmeyer (2004) studies the impact of monetary policy shocks on exchange rate of some small open economies, namely, Australia, Canada and New Zealand during the 1990s. His findings point to a 2-3% appreciation of the exchange rate after a 100 basis points hike in 3 months interest rates. Kohlscheen (2014) analiyzes Brazil, Mexico and Chile, and looking for 1 day after policy shock the author does not find support for the predictions of standard small open economies models of an appreciation of the exchange rate. Our findings also does not find short run effects over real exchange rate, and even show some depreciation effect, but not statistically significant at 5% level. But, on longer horizons the appreciation is clear and significant, as shown in Figure 32.

In more model dependent approaches, Costa Filho (2017) shows some weak evidence in favor of appreciation. Mendonça, Medrano and Sachsida (2010) found puzzling effects, but non-significant. Minella and Souza-Sobrinho (2013) point to a maximum 1.75% appreciation in 3 quarters, while Castro et al. (2011) shows immediate effects of about 1% real appreciation of domestic currency.

Results on real exchange rate are less consistent in the literature, and our approach may help shed some light on these questions. A point favoring our results is its consistency among different responses. Remember that regressors are the same for all our estimates, and the others results are less controversial than real exchange rate and are somewhat appealing. Of course that confounders relevant for real exchange rate may be missing, since internal forecasts of BCB used in our analysis seeks only inflation. But, market expectations about exchange rates are considered, and these are probably contemplated by BCB policy makers. Indeed, BCB forecasts contain scenarios considering Focus expected exchange rate, and we control for these scenarios.

3.3.5 Final considerations

There is room to improvements, like non-linear effects, other confounders not considered in our analysis and so on. Our goal was to make things as simple as possible, with a direct application of techniques developed in Chapter 2 to show their usefulness in practical problems. Our results are reasonable by a theoretical viewpoint, agrees in general terms to previous findings in the literature, are robust (see Appendix C) and open the doors to more sophisticated approaches.

Possible improvements are a better shrinking of covariates using LASSO (TIBSHIRANI, 1996), ridge regression (SALEH; ARASHI; KIBRIA, 2019), principal components regression (AMEMIYA, 1985) or partial least squares (HAENLEIN; KAPLAN, 2004), for example. Actually, since it is possible to translate the estimation of conditional distributions into prediction problems through what Angrist and Pischke (2008) calls the CEF decomposition property and CEF prediction property, and since identifiable causal effects can be asserted as expressions composed of conditional distributions by the SCM approach, a full set of modern prediction techniques in machine learning may be applied to causal inference, despite widespread beliefs that these techniques can only express correlations (MULLAINATHAN; SPIESS, 2017). Indeed, the SCM approach is all about expressing causal effects by means of correlations inferable from observable data.

4 Conclusion

In this thesis we reviewed the concept of causality in statistics and econometrics in Chapter 1, and applied the Structural Causal Model approach developed since the 1980s to time series data, discussing the meaning of causal inference in this sort of problem. We developed tools in Chapter 2 to express and estimate causal impulse responses, showing their effectiveness by means of Monte Carlo simulations. We also applied these tools to real world macroeconomic problems in Chapter 3, obtaining reasonable results. This kind of analysis is useful not just for inferential purposes, but also to put economic theories to test, enhancing the falsifiability of complicated structural models, which contributes to their development.

Our main contributions are the development of impulse responses with explicit causal content, something missing in the literature, also developing a technique for evaluating a sequential plan of interventions through Proposition 1, which opens the doors for a policy plan comprising various events of policy decisions, for example. Our techniques also bring to the causal identification world arbitrary approaches for conditional distributions estimations, as explicitly shown in the example using GAMs in Chapter 2. This also weakens common critics about the correlational nature of machine learning if these routines are applied looking for conditional distributions estimations with causal content attested by SCMs. This is very important to bring to the most interesting economic problems techniques used very successfully in other domains.

Also, our macroeconomic applications showed how the effects of interventions of interest for policy makers may be assessed by simple techniques with a reduced set of premises in large part testable. This makes our impulse responses more reliable and falsifiable then the ones from complicated mechanistic structural or computational models, which is good for model testing and development. As stated before, we do not believe in silver bullets, and because convoluted models may give more precise results and also more freedom to test complicated hypothesis, develop them is always useful, and our approach may be viewed as a guideline to what to expect from these models in specific situations, enhancing their reliability and contributing for their development.

A Generalized additive models

In Section 2.3 we analyzed non-linear systems. In the estimation procedure, generalized additive models to deal with non-linearities were adopted. References for techniques used are Wood (2017), Hastie (2017) and Hastie and Tibshirani (1987).

Generally speaking, a GAM is a model of the form

$$g(\mu_i) = A_i\theta + f_1(x_{1i}) + f_2(x_{2i}) + f_{3,4}(x_{3i}, x_{4i}) + \ldots \tag{A.1}$$

where g is a link function, $\mu_i = E(Y_i)$ with $Y_i \sim EF(\mu_i, \phi)$ a response variable distributed according to an exponential family distribution, $A_i\theta$ represents strictly parametric model components and functions f_k are smooth functions of the covariates x_k.

In all specifications of Section 2.3 we adopted an identity link, Gaussian response and one multivariate smooth function embracing all covariates. For estimation purposes we adopted the dimension of the basis of the smoother as 200. Details on GAM estimation are available in Wood (2017). So, our models have the form

$$E(Y_{t+h}) = f(X_t, Y_{t-1}, Z_t), \tag{A.2}$$

where variables are those in equations (2.16). We run (A.2) to estimate conditional expectations in equation (2.18). The procedure adopted for EGCIRF and ECGI computations is explained in Section 2.3.

B Data

Data source and transformations are shown in Tables 6 and 7. Since meeting days are irregular and does not coincide exactly with available data points, we made daily linear interpolations in all time series and matched these interpolations with meeting days. A more complicated scheme were drawn for variables predicting future. Focus data is not an issue because data are daily available, but BCB projections in inflation reports are not, and so we proceeded with a different interpolation scheme.

Table 8 represents data on internal forecasts made in days t, $t+1$, $t+2$, etc. These are represented in each line of the table. Also, each entry of the table, labeled in row-column convention as $(t+k, t+l)$ represents the forecast made in date $t+k$ for the variable at $t+l$. Forecasts represented by "O" are available data, and the ones represented by "X" are unavailable and we want to interpolate then. So, from the table is implicit that forecasts are made every k days and are also made for every k days in the future. Notice that forecast data is empty for $(t+k, t+l)$ where $l < k$ because this represents a prediction of past data.

	t	$t+1$	$t+2$	$\dots$	$t+k$	$t+k+1$	$t+k+2$	$t+k+3$	$\dots$	$t+2k$
t	O	X	X	$\dots$	O	X	X	X	$\dots$	O
$t+1$		X	X	$\dots$	X	X	X	X	$\dots$	X
$t+2$			X	$\dots$	X	X	X	X	$\dots$	X
$\vdots$				$\ddots$	$\vdots$	$\vdots$	$\vdots$	$\vdots$	$\ddots$	$\vdots$
$t+k$					O	X	X	X	$\dots$	O
$t+k+1$						X	X	X	$\dots$	X
$t+k+2$							X	X	$\dots$	X
$t+k+3$								X	$\dots$	X
$\vdots$									$\ddots$	$\vdots$
$t+2k$										O

Table 8 – Schematic representation of BCB internal forecast data.

For each line where there is forecast data we linearly interpolate then daily. So, we arrive at Table 9, where H represents a linear horizontal interpolation.

	t	$t+1$	$t+2$	$\dots$	$t+k$	$t+k+1$	$t+k+2$	$t+k+3$	$\dots$	$t+2k$
t	O	H	H	$\dots$	O	H	H	H	$\dots$	O
$t+1$		X	X	$\dots$	X	X	X	X	$\dots$	X
$t+2$			X	$\dots$	X	X	X	X	$\dots$	X
$\vdots$				$\ddots$	$\vdots$	$\vdots$	$\vdots$	$\vdots$	$\ddots$	$\vdots$
$t+k$					O	H	H	H	$\dots$	O
$t+k+1$						X	X	X	$\dots$	X
$t+k+2$							X	X	$\dots$	X
$t+k+3$								X	$\dots$	X
$\vdots$									$\ddots$	$\vdots$
$t+2k$										O

Table 9 – Schematic representation of BCB internal forecast data. "H" represents horizontally linearly interpolated data.

To interpolate periods where there are not projections things got trickier. We may interpolate then vertically, but columns where there is not data will became empty at bottom points, or must be extrapolated. A better option is to interpolate diagonally, but this is going to put information of future forecasts in interpolations,

something plausible, since forecasters probably have more information close to forecast announcement, but debatable. For example, the interpolated forecast at date $t+k-1$ for $t+k$ will be very close to forecast available at $t+k$ to $t+k$. A possible critique on this procedure is that the forecast at $t+1$ for $t+k$ is probably closer to the forecast made at t to $t+k$, and this is not being considered. So, we combine both ideas.

Consider the diagonal linear interpolation made after the horizontal one. This is represented in Table 10. Consider now an auxiliary variable α which varies linearly between t and $t+k$, $t+k$ and $t+2k$, etc., such that $\alpha(t+nk)=0$ and $\alpha(t+(n+1)k)=1$, $n=0,1,\ldots$. Let $A_{t+i,t+j}$ be the element $(t+i,t+j)$ of Table 10 and $I_{t+i,t+j}$ be the element $(t+i,t+j)$ of our final interpolation. So, we proceed in the following way: $I_{t+i,t+j}=\alpha(t+i)A_{t+i,t+j}+[1-\alpha(t+i)]A_{h,t+j}$ such that h is the maximum row value with $h<t+i$ and containing original data.

	t	$t+1$	$t+2$	$\ldots$	$t+k$	$t+k+1$	$t+k+2$	$t+k+3$	$\ldots$	$t+2k$
t	O	H	H	$\ldots$	O	H	H	H	$\ldots$	O
$t+1$		D	D	$\ldots$	D	D	D	D	$\ldots$	D
$t+2$			D	$\ldots$	D	D	D	D	$\ldots$	D
$\vdots$				$\ddots$	$\vdots$	$\vdots$	$\vdots$	$\vdots$	$\ddots$	$\vdots$
$t+k$					O	H	H	H	$\ldots$	O
$t+k+1$						D	D	D	$\ldots$	D
$t+k+2$							D	D	$\ldots$	D
$t+k+3$								D	$\ldots$	D
$\vdots$									$\ddots$	$\vdots$
$t+2k$										O

Table 10 – Schematic representation of BCB internal forecast data. "H" represents horizontally linearly interpolated data and "D" represents diagonally linearly interpolated data.

In this way we maintain interpolations of forecasts closer to real ones when dates are close. This procedure is just a coherence exercise, because in practice, only longer horizon forecasts are relevant for our estimates, and the interpolation procedure have very low impact on results.

C Robustness checks and regressions diagnostics

C.1 Robustness check - changing regressors set

In Tables 6 and 7 we present the covariates used in Chapter 3 regressions. A wider set of variables, presented in Tables 11, 12 and 13, were chosen from Inflation Reports of BCB, Focus survey and macroeconomic aggregates. This wider set does not present all possible variables because lots of them simply does not have any affect on impulse responses.

We can compare the EGCIRFs computed in Chapter 3 with the ones computed using covariates of Tables 11, 12 and 13 together with 4 lags of policy variable and lags 2 to 4 of real GDP gap, as explained in Chapter 3. This comparison is shown in Figures 36, 37, 38 and 39.

Results are remarkably similar, and are not statistically different. For the sake of robustness, results presented in Chapter 3 uses less 20 variables, and these variables may be added or removed without significant change in EGCIRFs. Actually, even various variables in Tables 6 and 7 can be removed or redefined without drastically changing conclusions.

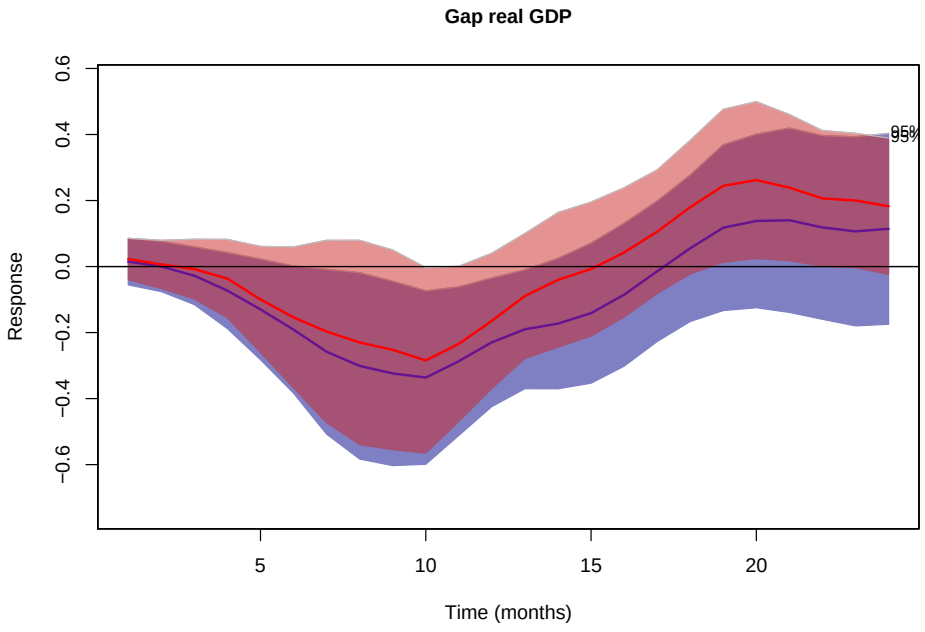

Figure 36 – EGCIRF of real GDP gap for 25 basis points uprise in Selic rate against no variation. Blue results are the ones in Chapter 3 and red results correspond to estimates using Tables 11, 12 and 13 covariates.

Regressor	Description	Source
S	Target Selic rate variation. This is the policy variable, determined on each COPOM meeting. Its coefficient is the causal effect of interest.	BCB - Copom Meetings
R_1	3 months difference on target Selic rate	BCB - SGS
R_2	12 months difference on target Selic rate	BCB - SGS
R_3	Last 3 months variation of inflation index	BCB - SGS
R_4	Last 6 months variation of inflation index	BCB - SGS
R_5	Last 12 months variation of inflation index	BCB - SGS
R_6	Median of Focus IPCA 1 month ahead projections - deseasonalized	BCB - Market Expectations System
R_7	Median of Focus IPCA 6 months ahead projections - deseasonalized	BCB - Market Expectations System
R_8	Median of Focus IPCA 12 months ahead projections - deseasonalized	BCB - Market Expectations System
R_9	Difference between reference scenario IPCA forecast by BCB for meeting day and 12 months accumulated IPCA	BCB - Inflation Reports
R_{10}	Difference of reference scenario IPCA forecast by BCB for 3 months ahead and BCB forecast for meeting day both adjusted for inflation target	BCB - Inflation Reports
R_{11}	Difference of reference scenario IPCA forecast by BCB for 6 months ahead and BCB forecast for 3 months ahead both adjusted for inflation target	BCB - Inflation Reports
R_{12}	Difference of reference scenario IPCA forecast by BCB for 9 months ahead and BCB forecast for 6 months ahead both adjusted for inflation target	BCB - Inflation Reports
R_{13}	Difference of reference scenario IPCA forecast by BCB for 12 months ahead and BCB forecast for 9 months ahead both adjusted for inflation target	BCB - Inflation Reports
R_{14}	Monthly variation of median meeting day Focus Selic rate forecasts	BCB - Market Expectations System
R_{15}	Monthly variation of median 6 months ahead Focus Selic rate forecasts	BCB - Market Expectations System

Table 11 – Regressors used in preliminary estimates, part I.

Regressor	Description	Source
R_{16}	Monthly variation of median 12 months ahead Focus Selic rate forecasts	BCB - Market Expectations System
R_{17}	Difference between the median of Focus annualized 1 quarter accumulated GDP forecasts for meeting day and accumulated 12 months GDP	BCB - Market Expectations System
R_{18}	Difference between the median of Focus annualized 1 quarter accumulated GDP forecasts for 1 quarter ahead and accumulated 12 months GDP	BCB - Market Expectations System
R_{19}	Difference between the median of Focus annualized 1 quarter accumulated GDP forecasts for 2 quarters ahead and accumulated 12 months GDP	BCB - Market Expectations System
R_{20}	Difference between the median of Focus annualized 1 quarter accumulated GDP forecasts for 3 quarters ahead and accumulated 12 months GDP	BCB - Market Expectations System
R_{21}	Real GDP 3 months log variation	BCB - SGS
R_{22}	Real GDP 6 months log variation	BCB - SGS
R_{23}	Real GDP 9 months log variation minus real GDP 6 months log variation	BCB - SGS
R_{24}	Real GDP 12 months log variation minus real GDP 9 months log variation	BCB - SGS
R_{25}	Real exchange rate 1 month log variation - dollar	BCB - SGS
R_{26}	Real exchange rate 6 months log variation minus real exchange rate 1 month log variation - dollar	BCB - SGS
R_{27}	Real exchange rate 12 months log variation minus real exchange rate 6 months log variation - dollar	BCB - SGS
R_{28}	Nominal exchange rate 1 month log variation - dollar	BCB - SGS
R_{29}	Nominal exchange rate 6 months log variation - dollar	BCB - SGS
R_{30}	Nominal exchange rate 12 months log variation - dollar	BCB - SGS
R_{31}	Focus end of month expected exchange rate variation - US dollar	BCB - Market Expectations System
R_{32}	Focus 6 months expected exchange rate variation - US dollar	BCB - Market Expectations System

Table 12 – Regressors used in preliminary estimates, part II.

Regressor	Description	Source
R_{33}	Focus 12 months expected exchange rate variation - US dollar	BCB - Market Expectations System
R_{34}	Monthly variation of Focus end of month expected exchange rate variation - US dollar	BCB - Market Expectations System
R_{35}	Monthly variation of Focus 6 months expected exchange rate variation - US dollar	BCB - Market Expectations System
R_{36}	Monthly variation of Focus 12 months expected exchange rate variation - US dollar	BCB - Market Expectations System
R_{37}	Focus expected industrial production 1 month ahead minus 1 year industrial production variation	BCB - Market Expectations System
R_{38}	Focus expected industrial production 6 months ahead minus 1 year industrial production variation	BCB - Market Expectations System
R_{39}	Focus expected industrial production 9 months ahead minus 1 year industrial production variation	BCB - Market Expectations System
R_{40}	Three months difference of reference scenario IPCA forecast by BCB for meeting day	BCB - Inflation Reports
R_{41}	Three months difference of reference scenario IPCA forecast by BCB for 3 months ahead	BCB - Inflation Reports
R_{42}	Three months difference of reference scenario IPCA forecast by BCB for 6 months ahead	BCB - Inflation Reports
R_{43}	Three months difference of reference scenario IPCA forecast by BCB for 9 months ahead	BCB - Inflation Reports
R_{44}	Three months difference of reference scenario IPCA forecast by BCB for 12 months ahead	BCB - Inflation Reports
R_{45}	Difference of market scenario IPCA forecast by BCB for 12 months ahead and same BCB forecast for 6 months ahead both adjusted for inflation target	BCB - Inflation Reports

Table 13 – Regressors used in preliminary estimates, part III.

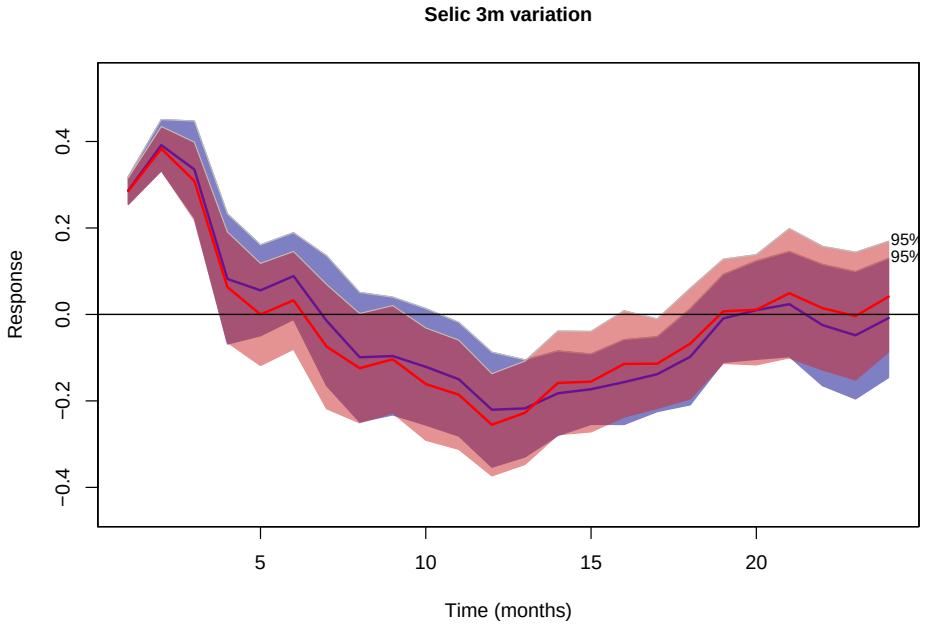

Figure 37 – EGCIRF of 3 months Selic rate variation for 25 basis points uprise in Selic rate against no variation. Blue results are the ones in Chapter 3 and red results correspond to estimates using Tables 11, 12 and 13 covariates.

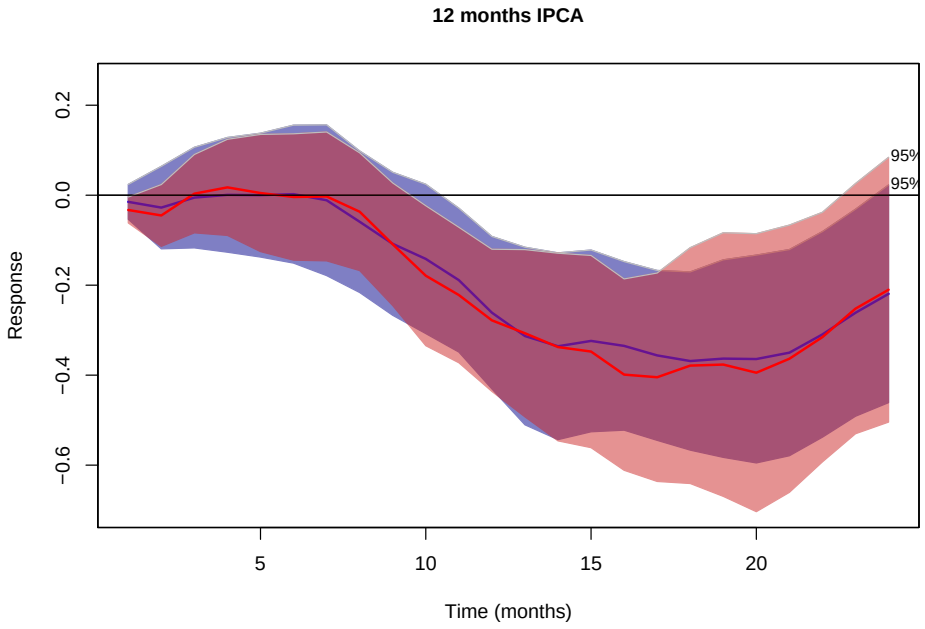

Figure 38 – EGCIRF of 12 months IPCA for 25 basis points uprise in Selic rate against no variation. Blue results are the ones in Chapter 3 and red results correspond to estimates using Tables 11, 12 and 13 covariates.

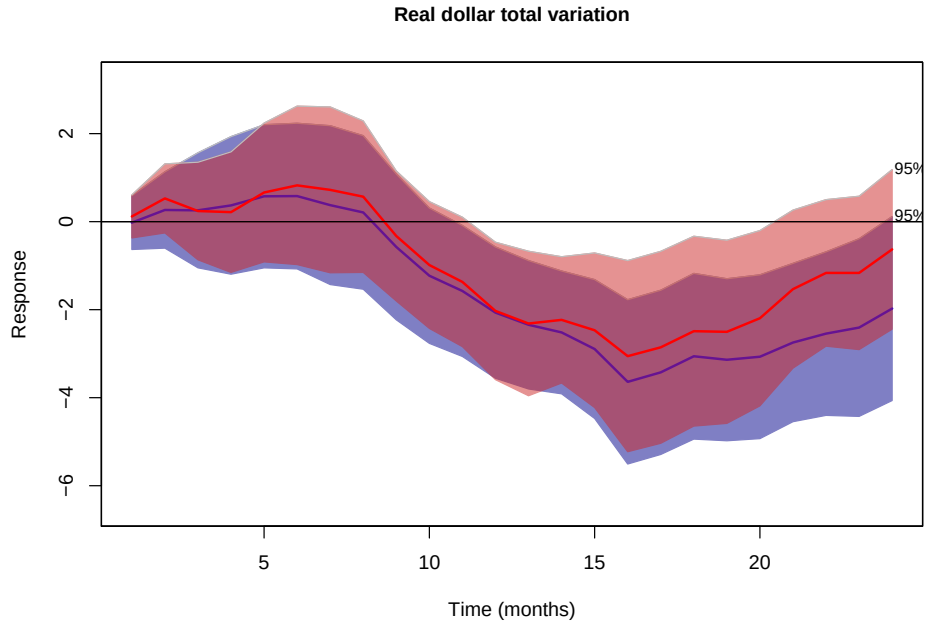

Figure 39 – EGCIRF of real dollar total variation for 25 basis points uprise in Selic rate against no variation. Blue results are the ones in Chapter 3 and red results correspond to estimates using Tables 11, 12 and 13 covariates.

C.2 Robustness check - changing time sample

Another robustness exercise is to change the sample size, computing EGCIRFs for shorter time samples. This additionally gives clues about possible changes in policy makers or economic agents behavior. Final results presented in Figures 29, 30, 31 and 32 ranges from 2003-02-19 to 2019-02-06 in their first horizon[1]. Cutting all data beyond 2014 gives a sample from 2003-02-19 to 2014-12-03, reducing the estimation period by more than 4 years from the end. Figures 40, 41, 42 and 43 shows comparisons among final results and reduced sample estimates. Notice that results are reasonably similar and, indeed, not statistically different.

Things got worse if we delete data from beginning of sample, starting from 2007-07-18 instead of 2003-02-19, in a more than 4 years cut from original starting point. Confidence intervals got much bigger and results behave more wildly. This happens probably because the period 2002-2003 were turbulent for monetary policy in Brazil. Markets feared the election of Luiz Inácio Lula da Silva, and strong depreciations of domestic currency were experienced, accompanied of higher inflation and interest rates. So, our results are particularly sensitive to this period. But, looking for comparisons in Figures 44, 45, 46 and 47, they are not so different, specially considering the confidence intervals, maybe except for GDP.

It can be argued that, besides economic turbulence, the BCB is yet looking for inflation control, trying to put it on target, and did not fundamentally changed its behavior, only rising Selic rate strongly because of stronger inflationary forces. One point corroborating this view is the presence of much bigger confidence intervals for the short samples estimations cutting data before 2007 in comparison with the previous time sample cut exercise. The causal interpretation of the regressions carried on in our analysis depends on the existence of unpredictable behavior from policy makers. Indeed, more unpredictability increases estimation precision, other things equal. This is so because causal estimations based on SCM may be interpreted as IPTW (Inverse Probability of Treatment Weighting) estimators, as shown in (PEARL, 2009), and so with more

[1] Different horizons may comprise different sample sizes because the regression for each horizon uses all available data. Since for longer horizons less data is available, data availability changes for longer horizons. So, we use as reference the sample for horizon 1.

variation on treatment choice (Selic rate determination), results may be more precise, and maybe this is what happens in our analysis. Of course that this can be investigated more deeply, but we do not do this here. After all, considering the confidence intervals, results in Figures 44, 45, 46 and 47 are yet reasonably close to our main results.

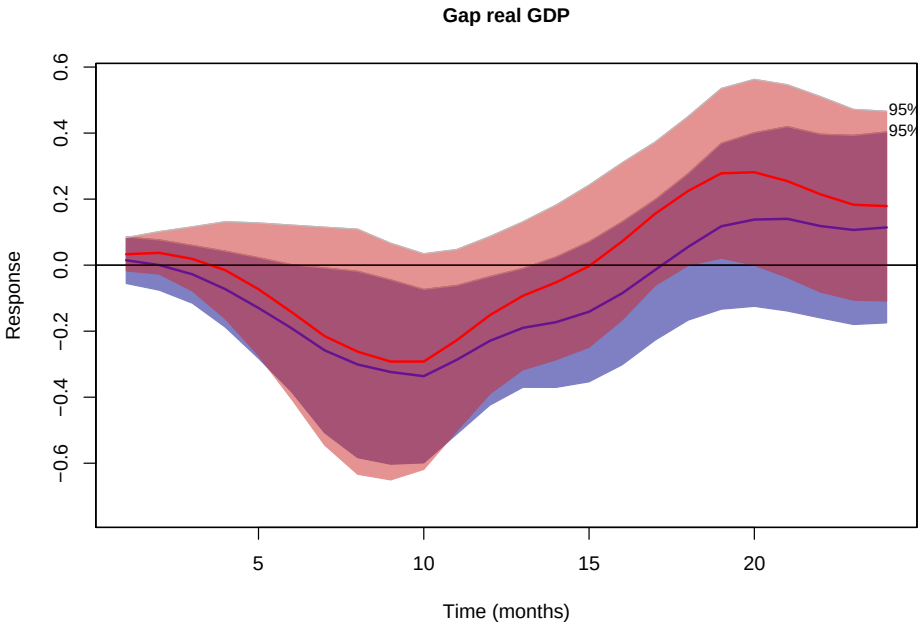

Figure 40 – EGCIRF of real GDP gap for 25 basis points uprise in Selic rate against no variation. Blue results are the ones in Chapter 3 and red results correspond to estimates using data only up to 2014.

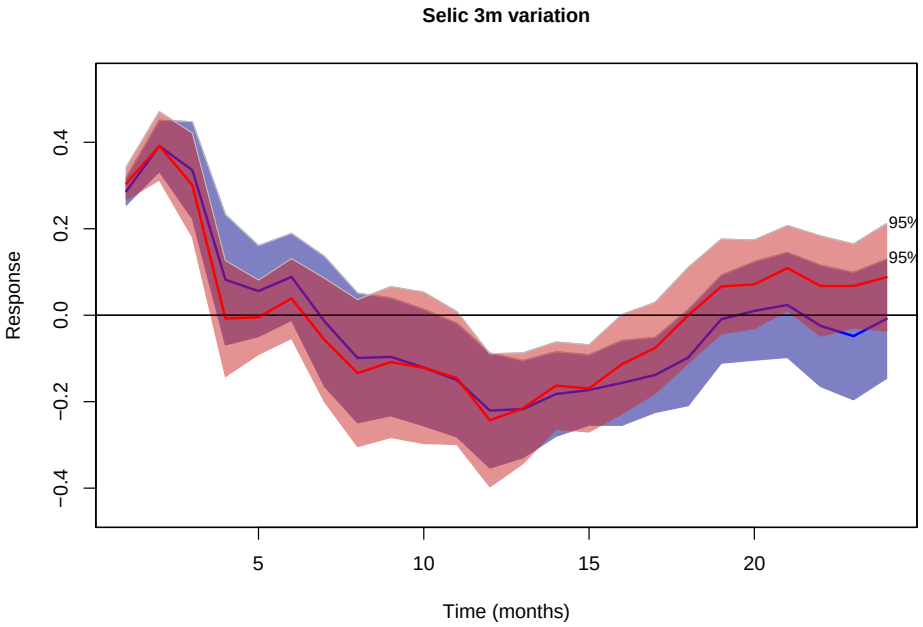

Figure 41 – EGCIRF of 3 months Selic rate variation for 25 basis points uprise in Selic rate against no variation. Blue results are the ones in Chapter 3 and red results correspond to estimates using data only up to 2014.

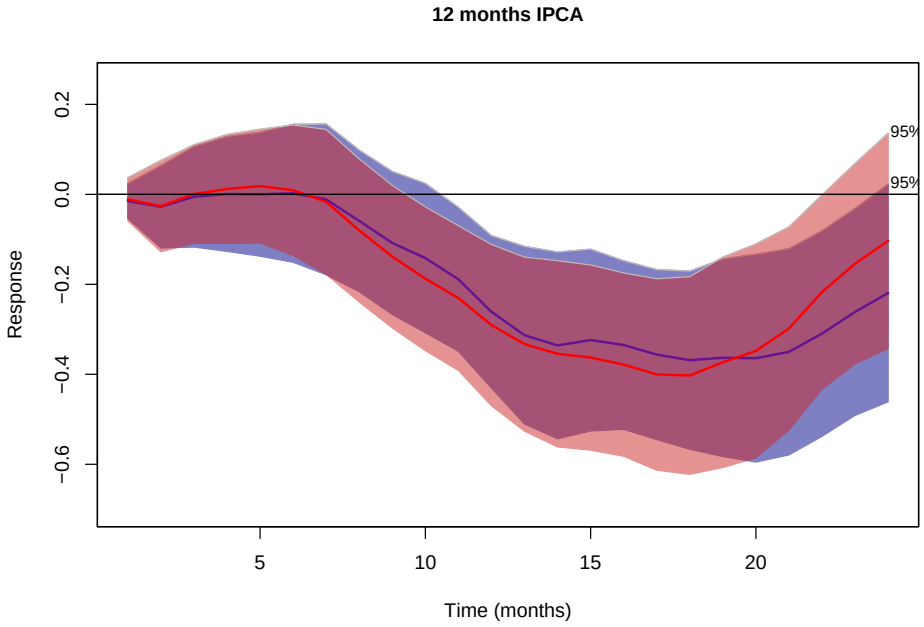

Figure 42 – EGCIRF of 12 months IPCA for 25 basis points uprise in Selic rate against no variation. Blue results are the ones in Chapter 3 and red results correspond to estimates using data only up to 2014.

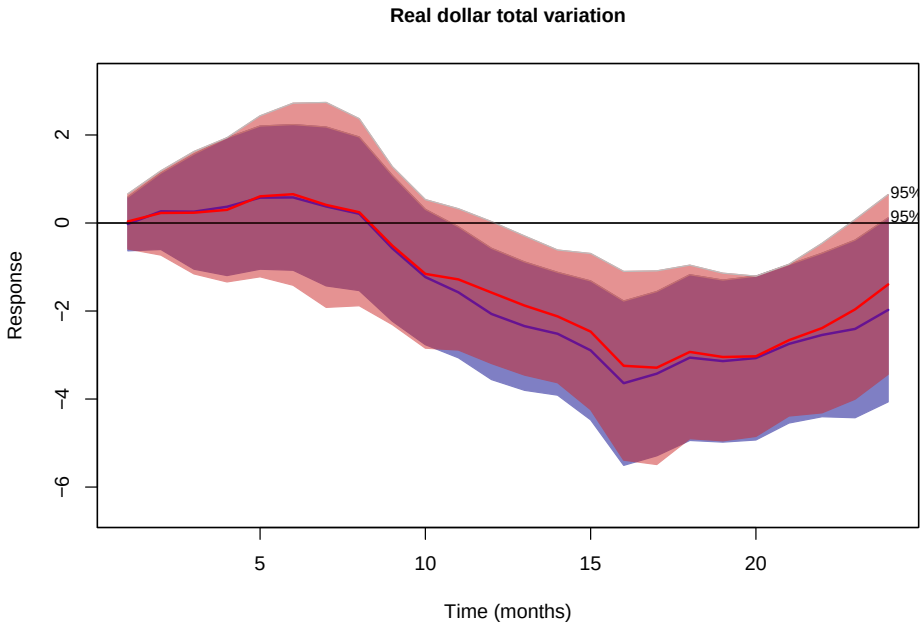

Figure 43 – EGCIRF of real dollar total variation for 25 basis points uprise in Selic rate against no variation. Blue results are the ones in Chapter 3 and red results correspond to estimates using data only up to 2014.

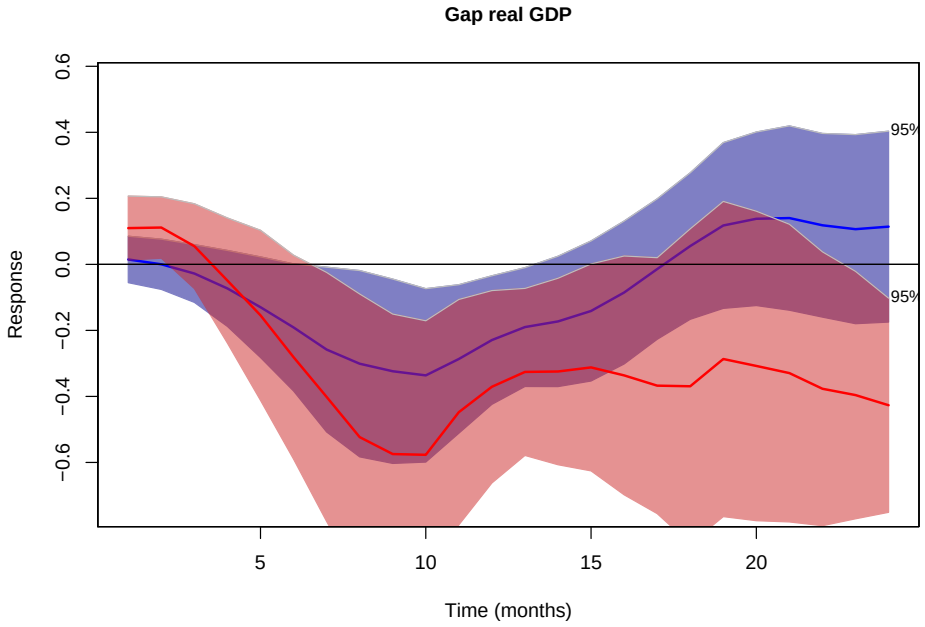

Figure 44 – EGCIRF of real GDP gap for 25 basis points uprise in Selic rate against no variation. Blue results are the ones in Chapter 3 and red results correspond to estimates using data only from mid 2007 on.

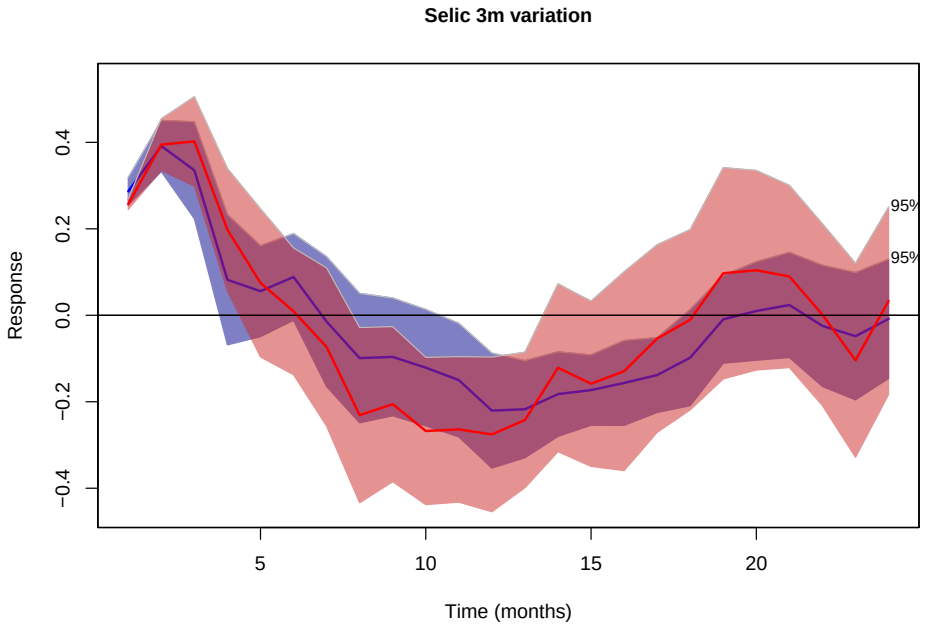

Figure 45 – EGCIRF of 3 months Selic rate variation for 25 basis points uprise in Selic rate against no variation. Blue results are the ones in Chapter 3 and red results correspond to estimates using data only from mid 2007 on.

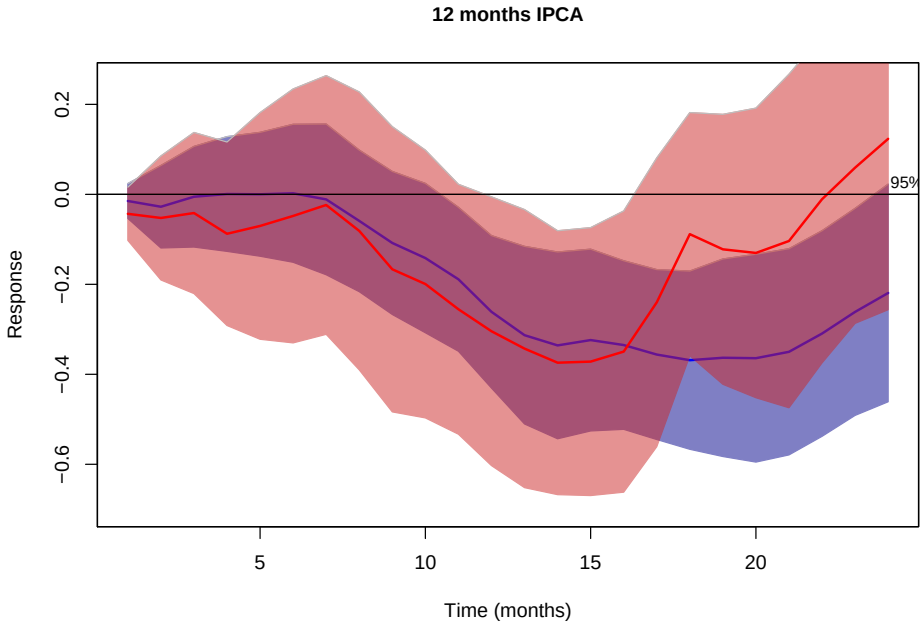

Figure 46 – EGCIRF of 12 months IPCA for 25 basis points uprise in Selic rate against no variation. Blue results are the ones in Chapter 3 and red results correspond to estimates using data only from mid 2007 on.

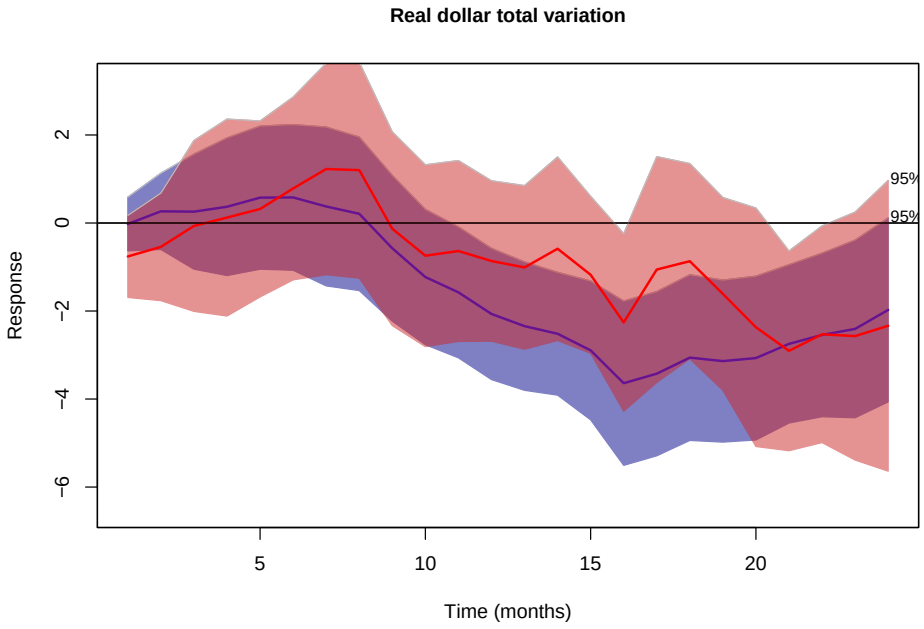

Figure 47 – EGCIRF of real dollar total variation for 25 basis points uprise in Selic rate against no variation. Blue results are the ones in Chapter 3 and red results correspond to estimates using data only from mid 2007 on.

C.3 Regressions diagnostics

Since each EGCIRF is comprised of h regressions, where h is the horizon of the response, we may analyze the properties of these regressions for diagnostics. First of all, lets test the premise of stationarity of regressors. Figure 48 shows a Venn diagram summarizing our tests.

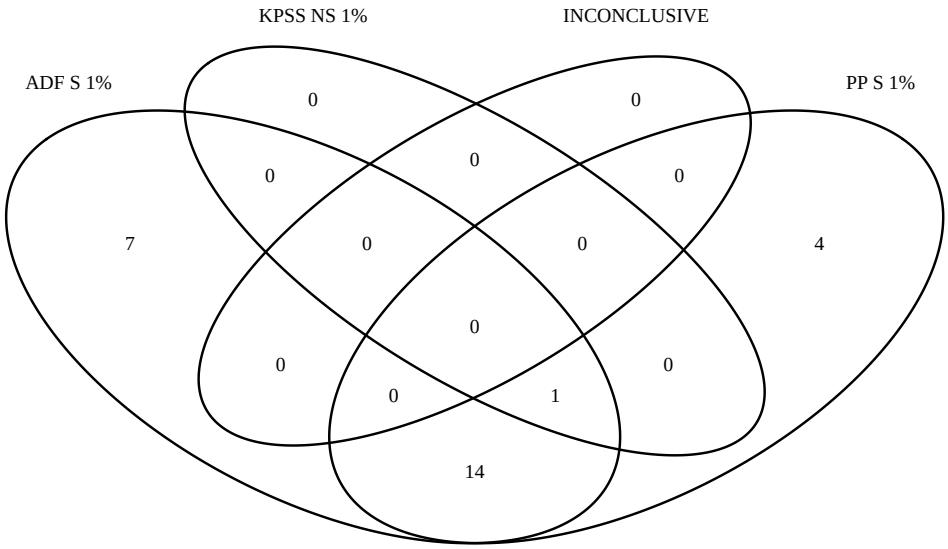

Figure 48 – Venn diagram representing ADF, KPSS and PP stationarity tests results at 1% level.

ADF is the Augmented Dickey–Fuller test (SAID; DICKEY, 1984; BANERJEE et al., 1993; GREENE, 2003) whose null hypothesis is the presence of unit root, KPSS is the Kwiatkowski-Phillips-Schmidt-Shin test (KWIATKOWSKI et al., 1992) whose null hypothesis is the stationarity of the series and PP is the Phillips–Perron unit root test (PERRON, 1988; BANERJEE et al., 1993) whose null hypothesis is the presence of unit root. The diagram reads as the number of series decided by each test, that is, the sets ADF and PP contains series whose presence of unit root was rejected at 1% level, the KPSS contains series whose stationarity was rejected at 1% level and the INCONCLUSIVE set contains series whose no conclusion is reached. As can be seen, only one series is decided non-stationary by KPSS test, but ADF and PP together decided for its stationarity, so we suppose it stationary.

For the response variables we have:

1. Real GDP gap with unit root rejected at 5% by ADF and stationarity not rejected by any standard[2] level by KPSS;

2. 3 months Selic variation with unit root rejected at 1% by ADF and PP and stationarity not rejected by any standard level by KPSS;

3. 12 months IPCA with unit root rejected at 1% by ADF and stationarity not rejected at 1% by KPSS;

4. Real dollar total variation with unit root rejected at 1% by ADF and PP and stationarity not rejected at 1% by KPSS.

[2] Standard levels are 1%, 5% and 10%.

So, all variables are relatively well behaved regarding their stationarity.

Now, consider residual tests for each horizon of each EGCIRF. Figures 49, 50, 51 and 52 shows p-values for Shapiro-Wilk normality test, KPSS[3], ADF and PP unit root tests, Durbin-Watson autocorrelation test and the Variance Inflation Factor (VIF) for the policy variable coefficient (SHEATHER, 2009). The red dashed line represents 5% significance level. In general, results point to stationary residuals, with some more severe deviations from normality by real GDP gap and real dollar responses. This deviation from normality is not so severe if we consider the wider set of covariates of Section C.1, and since results are close, this does not seem too much important.

Another important feature are the VIF values, between 5 and 10 (close to 7.5 for all estimates), showing some concern about correlations among policy variable and covariates. According with Neter, Wasserman and Kutner (1989), VIF > 10 is reason to concern, so we are fine. But, Sheather (2009) points to a cutoff of VIF > 5. One strong source of these correlations is the presence of lags of the policy variables, which we added by reasons presented in Chapter 3. Without these lags, all VIFs became closer to 5, in more acceptable levels. Since, with the exception of 1 month EGCIRF for 3 months Selic rate variation, all results are pretty close with or without the lags added, we conclude that VIF is not a concern for our estimations.

Finally, consider the Durbin-Watson p-values for all estimations (DURBIN; WATSON, 1971; CHATTERJEE; SIMONOFF, 2013). It points to the presence of autocorrelation in residuals, and this is unavoidable as explained in Chapter 3. For this reason we compute all confidence intervals using Newey-West HAC estimators.

[3] P-values above 10% for KPSS are not computed and so are represented as 10%.

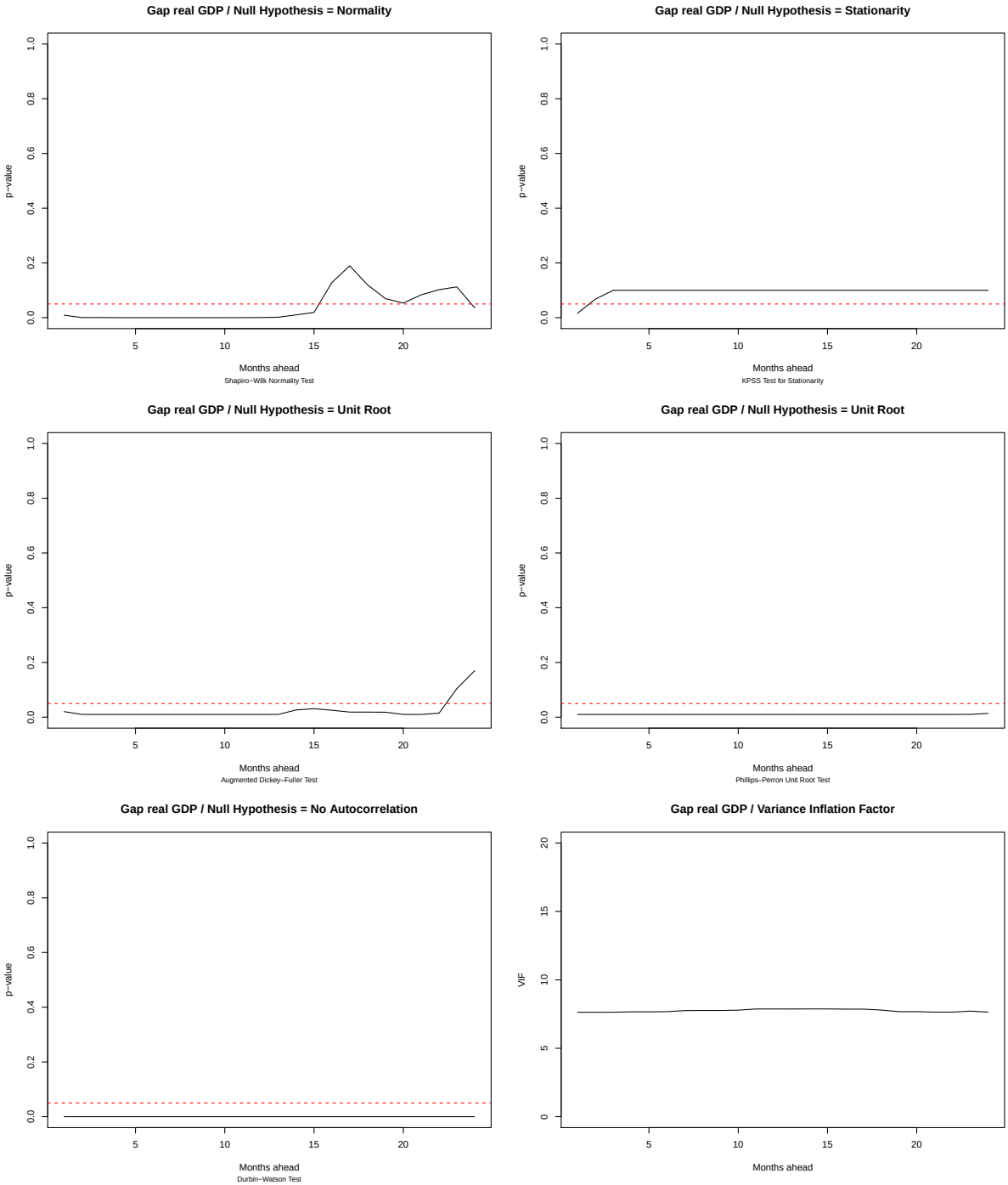

Figure 49 – Residuals diagnostics and VIF for real GDP gap.

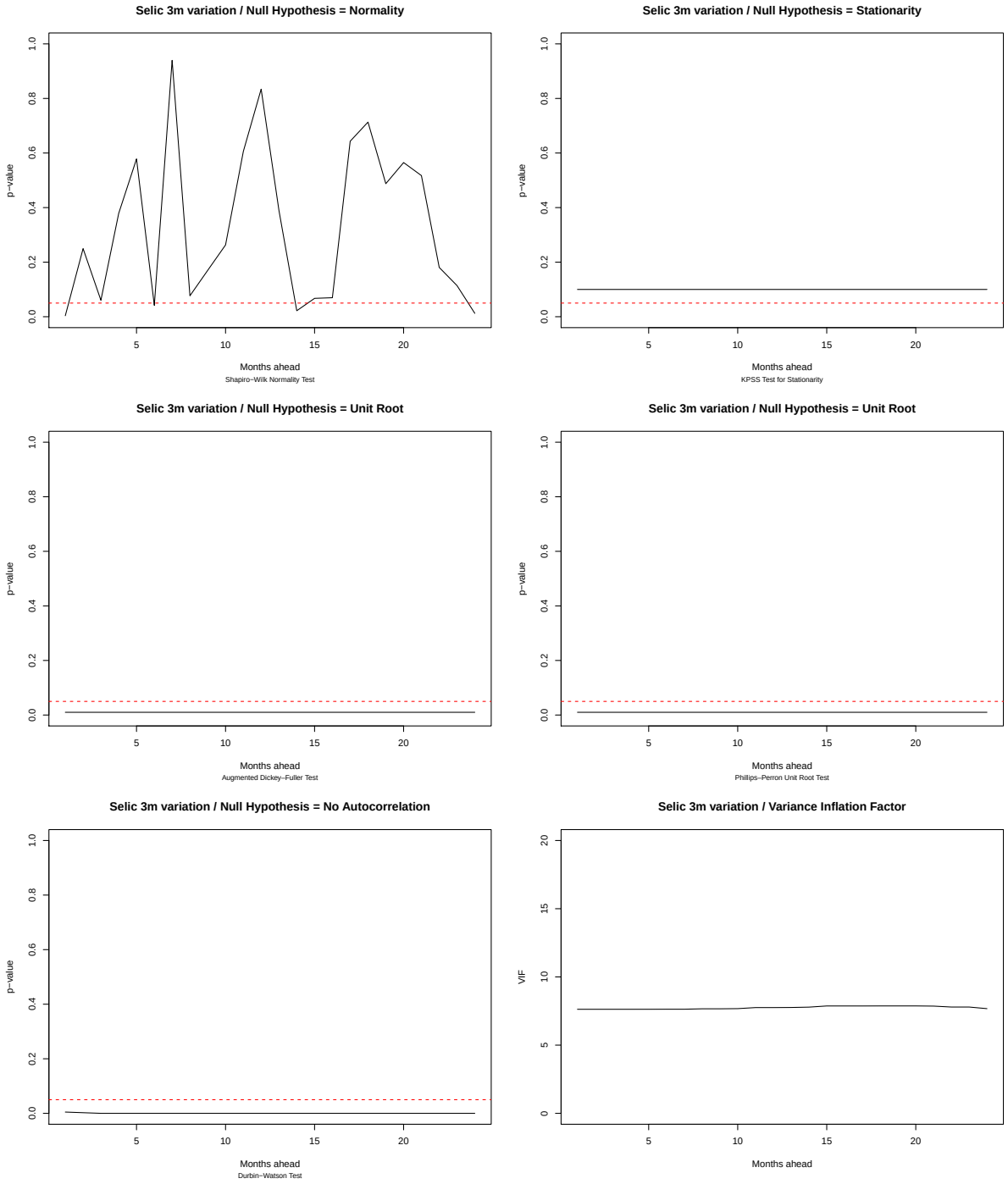

Figure 50 – Residuals diagnostics and VIF for 3 months Selic variation.

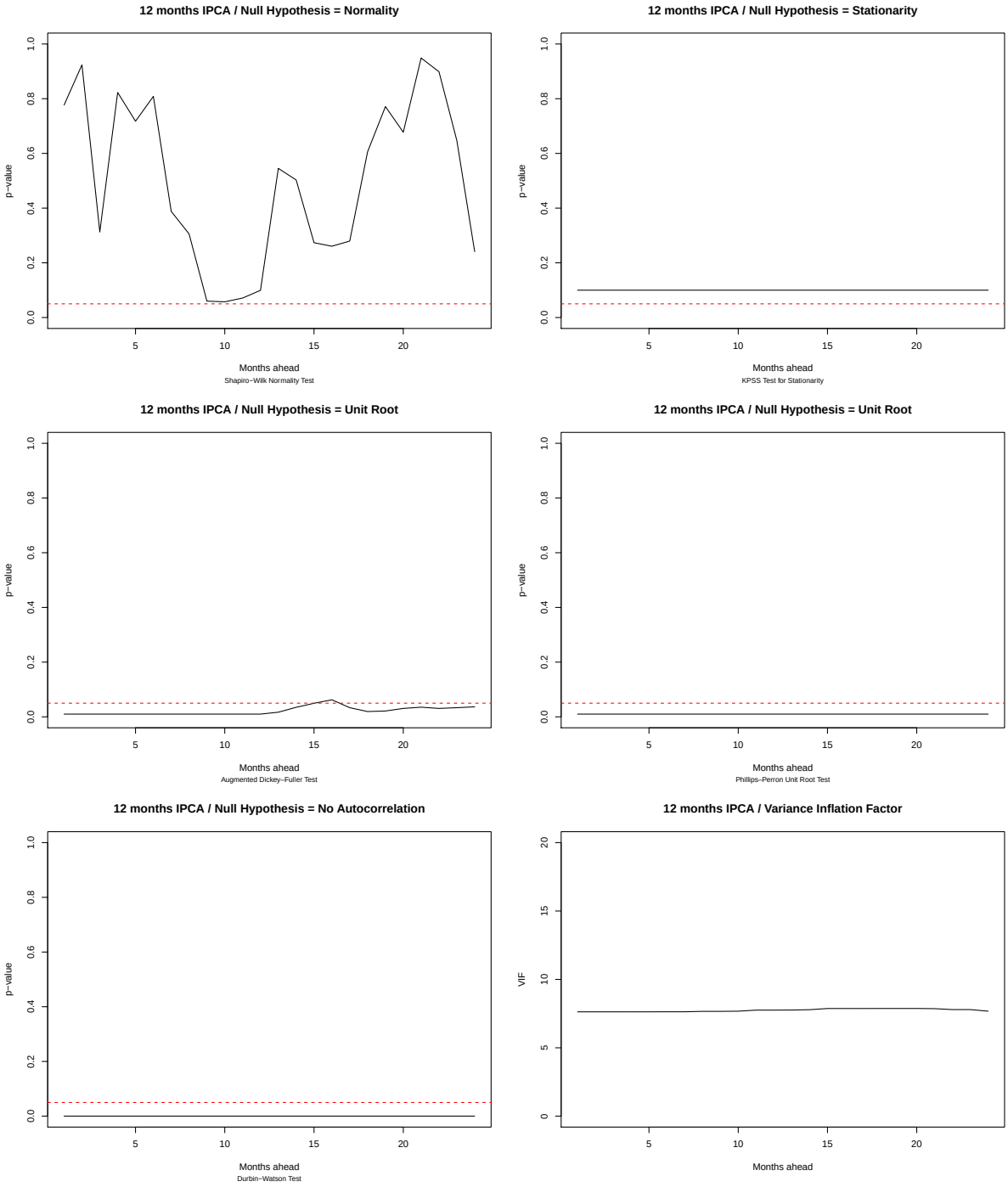

Figure 51 – Residuals diagnostics and VIF for 12 months IPCA.

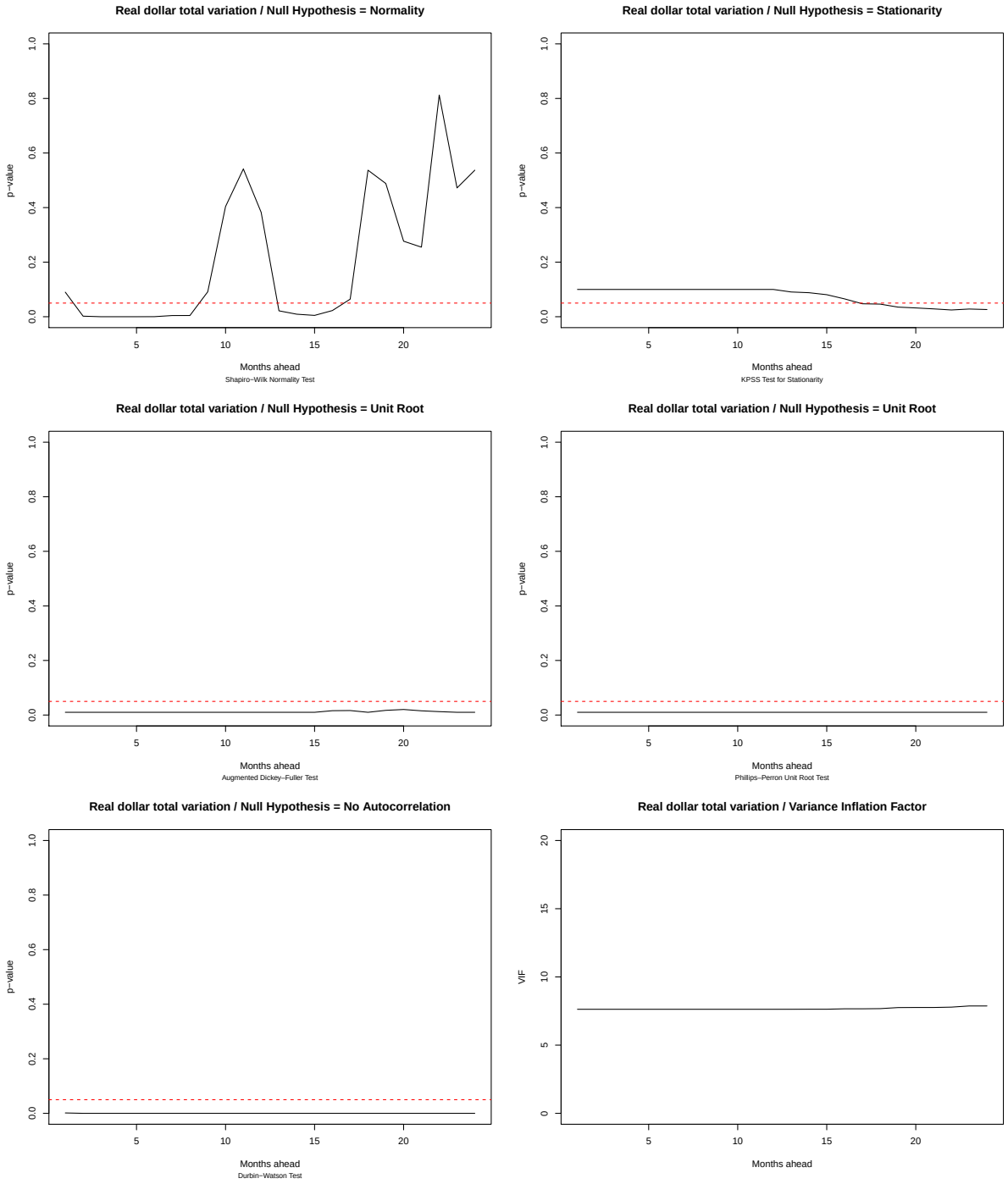

Figure 52 – Residuals diagnostics and VIF for real dollar total variation.

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