Essays on Causal Analysis in Cointegrated Systems: Causality, Exogeneity, and Driving Forces

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Declaration of Original Authorship

Declaration:

I confirm that this is my own work and the use of all material from other sources has been properly and fully acknowledged.

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The writing of this thesis and the conclusions drawn from this research would not have been possible without the help of my supervisors.

Firstly, despite my initial interest in causality in general, it was thanks to their foresight that I approached the problem within the context of cointegrated models. Their suggestion to study the work of Hoover was the seed of this thesis. Without this initial guidance, none of what follows would have existed.

Furthermore, it is thanks to them that I was able to freely explore this topic that had long intrigued my intellectual curiosity. I sincerely want to thank them for guiding me without imposing on the topic I chose, despite the numerous difficulties encountered in producing results. The trust they placed in my work, even in its early stages, forced them to bear a risk that ultimately allowed me to decipher this small piece of econometric literature.

I hope and believe that I have demonstrated to them that this research is a personal commitment of mine, and any future developments will not be a means for my future academic career, but rather, my future career will be a means to further this line of research.

Abstract

The appropriate definition, role and use of exogeneity in economic analysis has been an area of considerable contention. The empirical resolution of these disputes has proven to be challenging due to the incongruity between the econometric tools, which rely on statistical considerations, and the theoretical perspectives that emphasize causal relationships. The findings of the thesis aim to reconcile these two perspectives by investigating whether causal features manifest in model coefficients.

Drawing on the properties of the graphical representation of the latent causal structure, the first chapter introduces a causal definition of exogeneity. Exploiting state-of-the-art tools in the field of cointegration analysis, we investigate the interplay between causal exogeneity and the restrictions it imposes on models, without requiring that all relevant variables are observed. The duality between causal exogeneity and the restrictions on model parameters lays the foundation for the development of testing procedures tailored to facilitate causal logic.

The second chapter identifies an additional duality between model restrictions and causal properties and leverages it to propose an alternative testing technique. The chapter also

evaluates the performance of the two tests using simulated data and demonstrates their practical utility through an empirical illustration on monetary variables.

The third chapter examines the causal interpretation of weak exogeneity and cautions against associating it with causality. The chapter's findings are particularly pertinent in the context of cointegrated systems, where weak exogeneity is associated with constraints on the adjustment matrix. This coupling has led to misinterpretations, since variables unaffected by steady-state violations are associated with non-causality, and the absence of weak exogeneity is incorrectly perceived as a characteristic of causally affected variables. We aim to demonstrate that this duality is not automatic and clarify under what circumstances weak exogeneity can or cannot serve as a proxy for causal inference.

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Y	The set containing every variable affecting a system.
α	Adjustment matrix of the fully observed model.
β	Cointegration matrix of the fully observed model.
ε	Error term of the fully observed model.
Ω	(Diagonal) Covariance matrix of ε.
Y_u	The set containing the unobserved variables.
X_o	The set containing the observed variables explicitly modeled by the user.
T	The set containing the fundamental trends.
X	The set containing the ordinary variables.
X_u	The set containing the unobserved ordinary variables.
$\|A\|$	The cardinality of the set A.

α_o	Adjustment matrix of the partially observed model.
β_o	Cointegration matrix of the partially observed model.
ε_o	Error term of the partially observed model.
Σ_o	Covariance matrix of ε_o.
V	Covariance matrix of Y_u conditioned on X_o.
V_u	Covariance matrix of X_u conditioned on X_o.
V_T	Covariance matrix of T conditioned on X_o.
V_{uT}	Covariance between Y_u and T conditioned on X_o.
$\perp$	Orthogonal Complement.
$\mathcal{P}_Y(x)$	Parents of x in the set Y.
$\rightarrow$	causes.
$\leftarrow$	is caused by.
$\aleph$	The set of weakly causally exogenous variables.
$\beth$	The set of causally endogenous variables.
$\daleth$	The set containing the ancestors of $\aleph$ in X_u.
$\gimel$	The set containing the ancestors of $\beth$ in X_u not included in $\daleth$.
$\aleph_T$	The set containing the driving variables in X_o.

$\daleth$	The set the weakly causally exogenous variables that are not among the driving variables.
Ψ	Impact matrix of the modified model for the identification of weak causal exogeneity.
γ	Adjustment matrix of the modified model for the identification of weak causal exogeneity.
Σ^*	The covariance matrix of the residuals in the VECM modeling $\aleph$ only.
$A_{(i)}$	i^{th} row of matrix A.
$A_{(-i)}$	Matrix resulting after removing the i^{th} row of A.
$A^{(i)}$	i^{th} column of matrix A.
M_o	The matrix relating the observed variables in the state-space representation.
M_{ou}	The matrix relating the observed variables to the unobserved ordinary variables in the state-space representation.
N_o	The matrix relating the observed variables to the fundamental trend in the state-space representation.
M_u	The matrix relating the unobserved ordinary variables in the state-space representation.

M_{uo}	The matrix relating unobserved ordinary variables to the observed variables in the state-space representation.
N_u	The matrix relating unobserved ordinary variables to the fundamental trends in the state-space representation.
$\mathbf{det}(A)$	Determinant of matrix A.

Preface

In this preface, I present a summary of personal reflections and ideas developed during my research journey. These thoughts, shaped by personal impressions rather than rigorous academic scrutiny, represent a flow of thoughts that, at times, may not be well-structured. This overview is intended to offer insight into the underlying thought processes that have guided my work, acknowledging that these reflections may lack the formal structure and depth typical of academic discourse.

Before starting my research career, I envisioned a single, definitive "truth" and saw the role of the researcher as uncovering it. I viewed econometrics as the means to reveal the economic "truths" suggested by the data. However, to my disappointment, I found that the general econometric approach departs from this ideal, at least in certain subtle ways. Although these differences may seem minor at first glance, they profoundly alter both the scientific role and the social mission of econometricians.

I observe a pervasiveness of a practical approach and a preference for efficiency over understanding. The focus is predominantly on modeling—specifically, an approach based on the search for a relatively manageable function that best describes what is observed.

This method has little to do with the idea of "truth". It does not seek to understand the meaning of what happens but merely aims to "dress" what is observed, with the ultimate goal of creating a "map" to navigate smoothly through the intricate "alleys" of economic processes. However, while a map is undeniably effective for finding points of interest, it remains essentially silent regarding the history and significance of those places.

This approach views the econometrician as a passive agent, whose skill lies in reproducing what is seen. It is not far removed from a painter trying to reproduce the landscape in front of them as faithfully as possible, perhaps emphasizing certain key aspects such as colours, lines, or perceptions. Yet, this is merely a process of description that has little to do with he research of the "truth".

By this, I certainly don't mean to imply that a passive approach is wrong or secondary. There is no doubt that it is more effective. The quantitative description of what we observe allows us to make efficient predictions and to describe what happens in a simplified and comprehensible manner. Yet, despite its greater effectiveness, understanding a phenomenon should transcend mere description and focus on its deeper meaning.

A key point lies in understanding how "truth" is articulated. It is indeed true that even a purely model-based description or a rule derived from a mathematical function can be a form of truth. For instance, if the model $\textit{Investments} = \beta \times \textit{Interest Rate}$ is accurate, this representation corresponds to a "true" description. However, the human mind does not find meaningful a set of measured rules. Our eyes perceives the "truth" only when those rules take on meaning.

I find nothing other than causality as the concept that gives meaning to rules. I strongly believe that an analysis aimed at discovering the "truth" must be based on a causal structure. Causality is, in fact, a mental framework that gives shape to a sequence of events that would otherwise be just a simple flow of images. Indeed, observation without causal interpretation would be pointless and meaningless. Observing a sunny day followed by a good harvest is entirely useless until one understands that sunlight causes the plants to grow and develop. Only once a causal interpretation is established does reality make sense, and human action becomes fruitful. In fact, it is only by understanding that sunlight causes plant growth that humans can adjust their agricultural techniques to improve the harvest.

To be honest, it is difficult for me to imagine how sentient beings could observe without attempting to place their perceptions within a causal framework. Almost immediately, as we observe and seek to understand what we see, our brains begin to identify causal connections, allowing our minds to transform mere perception into reality and, ultimately, into "truth"

Causality is foundational to human thought, and the concept of cause appears to be a primitive notion—one that is challenging to define without invoking the idea of cause itself, or related terms such as "generation," "creation," or "production." This suggests that causal reasoning is innate, embedded within the cognitive structure of our minds. It seems less a learned process and more an inherent component of our mental architecture, part of the general settings of our innate cognition.

One indication that suggests the concept of cause is primordial, rather than a notion defined ad hoc following specific guidelines, is the ease with which this term is used despite its extraordinary semantic complexity. This complexity can be observed by noticing that when we say "A causes B," this is often just a simple approximation of the true cause. This can be illustrated by the child's game of "Why?", in which a child repeatedly asks "Why?" in response to any answer given, and the person questioned must continually provide new reasons. The game usually ends either when the child loses interest or when the adult runs out of explanations. Inevitably, one either accepts their own ignorance or resorts to invoking a superhuman entity, namely the will of God.

It should be noted that no matter how much knowledge is refined, there will always remain an unresolved "why." For instance, if one begins with the question "Why does touching fire cause pain?", even if one ends up with a detailed explanation of how thermoreceptors in nociceptors react to heat due to the depolarization of the cell membrane triggered by the opening of ion channels, one might still ask why these ion channels open. At that point, one might have no choice but to attribute it to the will of God, who decreed that ion channels should open in response to thermal stimulation.

Causality is like a chain to which links can always be added by tracing backward. Moreover, as if it is a fractal, each connection contains within it infinite sub-connections. Causality, therefore, has infinite potential, but our minds have finite capacities. As a result, the structure upon which our reasoning is built becomes too complex for our brains to fully comprehend. This complexity necessitates halting the chain at some point, and the only

solution is to invoke the will of some entity, which by definition, would have a divine nature.

Although I believe that causal reasoning is the foundational structure of our minds, I am uncertain whether causality exists independently of human perception—as a mechanism by which nature operates. If this were the case, our way of thinking would align with the workings of nature. Could it be that this very aspect of our thought process is what makes us in the image and likeness of God? I obviously have no suggestions on this matters, and the only recourse is to hope, with faith, that it is so.

However, one thing I am certain of is that any model aiming to capture truth as perceived by humans cannot disregard a causal framework. This applies to economic models as well and aligns with the approach of the founding fathers of structural models. Indeed, non-causal interpretations have been offered retrospectively by more recent authors. Yet, I would challenge anyone to understand how an interpretation of these models based on conditional probabilities could possibly explain the functioning of the economic system without introducing causal intuitions.

The problem with classical structural methods is that the "true model" must be pre-specified. It can then be tested using the rules of statistical inference. However, it is highly unlikely that we can successfully design the true model, and it is very plausible that any specification we can imagine is incorrect. The difficulty in identifying the true model arises from several factors, such as assumptions of linearity, the choice of specific time lags, the measurability of economic variables, and the ability to account for the

multitude of factors influencing the economic system.

I fully agree with those who view with skepticism the possibility of representing the "true" model, and I believe this issue undermines the effectiveness of the classical approach. Consequently, I am not satisfied with the standard strategies, and this is why I believe that econometric techniques should approach the problem with an awareness of this limitation: instead of constructing the model first and then gathering evidence to contradict it, I believe we should focus on determining which "true" model could have generated the characteristics observed in the evidence we have—evidence that is likely to be limited. This approach is somewhat related to the methodologies used in graphical causal inference, where statistical patterns are observed first, and then the "true" causal structure that may have generated them is understood.

This thesis proposes techniques that leverage this perspective. Rather than specifying a model and then testing to determine if it is the true one, it introduces hypothesis tests that, based on their results, correspond to properties of the "true" model.

Naturally, the approach proposed here does not address all the challenges that can arise in formulating the true model. It focuses instead on overcoming what I consider to be the greatest limitation: the need to include all relevant variables in the model (e.g., those with common causes). In a complex system like the economy, this step is particularly challenging, and the idea of being able to account for all these variables seems to me a hopeless task.

In conclusion, I have written this preface to convey that this thesis is not merely the

result of statistical refinements for economic analysis, but that the techniques proposed here stem from questions I have asked myself about the essence of econometric inquiries. Every mathematical argument is the outcome of a personal reflection on the ultimate purpose of an econometric model. That said, I still do not fully grasp what a "true model" is, nor can I yet provide an effective definition of causality. Furthermore, I am uncertain whether it will ever be possible to perfectly refine an econometric technique which follows the aforementioned approach. Despite this, I currently believe that this idea is the right one to pursue, and my future research will be guided by this thought, even though I am aware that this may be nothing more than a tilt at windmills.

Introduction

An examination of economic and macroeconomic ideas reveals how economic reasoning significantly relies on causal language. Both normative and positive analyses necessitate a meticulous identification of causative factors and their corresponding effects to extrapolate information about the functioning of economic systems. This critical aspect becomes apparent when considering the fundamental inquiries posed by economists, such as: what are the determinants of economic fluctuations? What is the optimal policy-making strategy to maintain inflation rates below specified thresholds? How should policy instruments be selected to stimulate GDP growth? Implicitly or explicitly, these inquiries demand an in-depth comprehension of stable directional relationships between economic magnitudes, denoted as causes and effects.

For instance, understanding the determinants of economic fluctuations involves identifying variables that cause changes in economic activity and assessing their impact. Similarly, determining the optimal policy strategy for controlling inflation requires understanding the causal mechanisms through which policy actions influence inflationary pressures. Selecting appropriate policy instruments for GDP growth stimulation entails recognizing the causal

pathways through which these instruments affect economic output.

It is not surprising that philosophers such as Mäki (2001) recognize causality as a central tenet in economic ontology. This acknowledgment underscores the essential duality between causal language and the discipline of economics. It must be emphasised that this interconnection is not confined to philosophical considerations but extends to practitioners and it is probably not a mere coincidence that prominent philosophers of causality also contributed to the economic discipline (Hoover (2006)).

The historical interplay between philosophical thought on causality and economic theory traces back even to ancient Greece. In his renowned treatise "Physics," Aristotle expounded upon his reflections concerning material, formal, final, and efficient causes. Simultaneously, Aristotle's contributions to economics were predominantly articulated in works such as the "Topics", the "Politics", and the "Nicomachean Ethics". However, the preeminent example of an intellectual who unequivocally shaped both the philosophy of causality and economic thought was David Hume. In Hume (2003), the author made seminal contributions to the philosophical understanding of causality, while his insights into economic principles were elaborated in essays such as "On Money," "On Interest," and "Of the Balance of Trade" (see Hume (1907)).

The prominence of the term "cause" in the titles of seminal economic works further underscores the enduring association between causality and economics. Notably, Adam Smith's foundational work, "An Inquiry into the Nature and Causes of the Wealth of Nations," epitomizes this connection (Smith (1776)). This emphasis on causality in economic inquiry

is not merely terminological but reflects a fundamental concern with understanding the determinants of economic phenomena.

The divergence of opinions regarding the causal order of macroeconomic variables has significantly shaped the discourse among various economic theories, becoming a primary source of economic debates. An illustrative case is the longstanding and still relevant controversy surrounding the relationship between supply and demand. Say's law, a renowned proposition articulated by Jean-Baptiste Say, posits that there is no inherent tendency toward economic depression, contending that an increase in supply prompts a corresponding increase in demand (Say (1803)). This conceptualization can be represented by the following graphical sketch: $Y \rightarrow AD$, where Y signifies output and AD represents aggregate demand. The implied causal trajectory in this theory is clear, commencing with supply and concluding with demand.

The conceptual underpinning of Say's law found support and prominence through the advocacy of Ricardo (1817), while encountering strong opposition in the ideology of Malthus (1836). Malthus proposed an alternative perspective, asserting the principle of effective demand, wherein demand influences the quantity of goods supplied. His alternative conceptualization implies a causal structure diametrically opposed to that posited by Say, as exemplified by the following sketch: $Y \leftarrow AD$.[1]

[1]During the relevant period, the prevailing economic orthodoxy regarded Ricardo's and Say's law as triumphant in the ensuing debate. Keynes, employing hyperbolic language, asserted that "Ricardo conquered England as the Holy Inquisition conquered Spain. His theory was accepted by the city, by the statesmen and by academic world. But controversy ceased; The other point of view disappeared, it ceased to be discussed. The great puzzle of Effective Demand with which Malthus had wrestled vanished from the economic literature"Keynes (1936). However, Malthus' ideas came back to life during the great crisis of '29 when they were taken up by Keynes, becoming in those years the orthodoxy. (see for detail Gordon

Divergent perspectives on causal directionality are evident across various key macroeconomic variables. For instance, the Mercantilist school proposed that an abundance of precious metals leads to national prosperity, as exemplified in works such as Malynes (1601). In stark contrast, Hume asserted the opposite viewpoint, by supporting the idea that national prosperity will eventually lead to an abundance of gold and other valuable alloys.

In the 19th century, the English Bullionists and the Currency school advocated a transmission mechanism wherein money influences prices, while the Banking school defended a perspective grounded in the opposite causal structure (Price $\rightarrow$ Money). This debate on the causal relationship between Price and Money persisted through subsequent economic thinkers. For instance Keynes, after initially defending the quantity view ($Y \leftarrow$ Money $\rightarrow$ Prices) (Keynes (1930)), he refuted it in subsequent works, shifting focus to the impact of interest rates and output on money demand (Keynes (1936)).[2] The discourse resurfaced with the influential positions of Friedman (1956), who revitalized the quantity theory of money.

Even within the same theoretical frameworks, economists diverged on the causal pathways. For instance, within the proponents of the quantity theory of money, some argued that an increase in money supply leads to higher prices, thereby stimulating business activity and subsequently boosting output (Money $\rightarrow$ Prices $\rightarrow Y$). Conversely, others emphasized a

(1965)), and later found new popularity after the great financial crisis of 2007.

[2]The revised perspectives of Keynes garnered substantial endorsement from economists associated with the Stockholm School, including prominent figures such as Bertil Ohlin and Gunnar Myrdal. These scholars played a pivotal role in elucidating the causal reasoning underpinning Keynes' ideas. A comprehensive exploration of this emphasis on causal understanding can be found in works such as Myrdal (1957).

pathway where an increase in money first affects output, which in turn influences prices, positing that higher money supply increases demand and output, gradually leading to inflation (Money $\rightarrow Y \rightarrow$ Prices) (see Blanchard (2000)).

Unfortunately, many economists have shown a reluctance to explicitly employ causal language and reasoning. This aversion is particularly evident in the works of Friedman (see Hammond (1996)). Friedman argues that no single cause can be isolated as complete, and each direct cause is itself influenced by other direct causes. Despite his aversion, he often finds it challenging to avoid using synonyms with substantial causal implications. For instance, in Friedman and Schwartz (1963a) and Friedman and Schwartz (1963b), he frequently employs terms like "affect," "influence," "response," and "prime mover," among others. As argued in Hoover (2004), Friedman's avoidance of explicit causal language seems more like a personal quirk rather than a genuine methodological commitment.

A possible contributing factor to the scarcity of explicit causal language in economic discourse originates from the prevailing orthodox thought of the 19th century. This ideological framework not only shaped the economic dynamics of 20th-century works but also influenced contemporaneous economic theories. This orthodoxy, influenced by proponents of general equilibrium theory such as Walras (2013) and Marshall (2009), emphasizes the presence of re-balancing forces where fluctuations in variables are generated by deviations from steady states. This perspective minimized the perceived utility of explicitly delineating clear causal directions, making causal reasoning appear unnecessary at first glance.

However, the presence of simultaneous causality does not negate the possibility of causal interpretations; instead, it signifies a causal structure characterized by cyclical relationships. Furthermore, as extensively elaborated in this thesis, the process of equilibrium adjustment does not imply a mandated bidirectionality of causal path. There are instances where market forces impact only one variable, prompting it to respond and restore equilibrium while allowing the other variable to fluctuate freely. This dynamic exemplifies a non-symmetrical framework that can be effectively conceptualized using the asymmetrical notion of causality. Therefore, the existence of rebalancing forces and dynamics resulting from violations of steady states does not preclude the application of causal reasoning; rather, it necessitates a distinct formulation of the concept of causality. This aspect will be better explained at the end of this introduction and will be largely elucidated throughout this thesis.

We have demonstrated examples from macroeconomic literature to underscore the importance of using causal language and highlighted how disagreement on the causal structure generates the economic debates. When competing economic theories lack logical inconsistencies, empirical evidence becomes the primary means to distinguish the validity of one theory over another. Unfortunately, numerous challenges arise in the pursuit of measuring and inferring causal connections. Indeed, far from granted is the task of reconciling the inherent causal dynamics of macroeconomics with the associational methodologies of statistics. The well-known adage "correlation is not causation" succinctly captures the

pervasive awareness of the considerable gap between statistical tools and the nuanced concept of causality.

As emphasized by various authors, an econometric model must be equipped to model and infer causal relationships. The fundamental difference between statistics applied to economic data and econometrics lies precisely in their treatment of causality. Statistical methods primarily identify and measure associations, whereas econometrics has developed methodologies designed specifically for identifying and measuring causal relationships.

The Nobel laureate James Heckman stated:

> The major contributions of twentieth-century econometrics to knowledge were: the definition of causal parameters . . . the analysis of what is required to recover causal parameters from data . . . and clarification of the role of causal parameters in policy evaluation (Heckman (2000) p.45).[3]

However, the relationship between econometrics and causality has frequently been contentious. The prospect of identifying and quantifying causal relationships through empirical observations has encountered skepticism among economists and, more broadly, philosophers of science. This skepticism echoes the concerns articulated by Hume, who harbored general doubts about our ability to comprehend the essential nature of causation in objects (see for instance Hume (2018) and Hume (2003)).

John Stuart Mill, another economist and philosopher renowned for his contributions to the study of causality, adopted a somewhat less extreme position. He formulated several

[3]Extract taken from the abstract of the paper with emphasis added by Hoover (2004)

principles for inferring causation (Mill (1843)), while admitting his wariness about their application in economics (Mill (1848)). Mill attributed this caution to the imprecise and distinct nature of economics as a discipline, where fundamental principles are largely understood a priori (Hausman (1992)). In fact, in economic inquiry, controlled experiments are often impractical or ethically unsound, making it impossible to maintain the ceteris paribus condition. Mill's skepticism (Mill (1967)) influenced subsequent researchers such as Ludwig Von Mises, who even categorically rejected the notion of economics as an empirical science (von Mises (1966)).

A pivotal moment initiating the reconciliation between economic and statistical languages is attributed to Haavelmo (1944). Often regarded as the inception of modern econometrics, Haavelmo's work introduced the idea of deconstructing an economic process into deterministic and stochastic components. According to Haavelmo, if the researcher successfully identifies the causal structure of the deterministic component, the stochastic element will conform to the law of probabilities. Haavelmo's framework provided a roadmap for integrating the causal discourse of economics with the statistical associations, offering a concept that appears straightforward yet robust that has shaped and continues to underpin the foundation of the econometric model developed since.

The ideas and methodologies pioneered by Haavelmo garnered significant support and elaboration within the community of economists associated with the Cowles Commission tradition. Building upon Haavelmo's foundational concepts, these economists conceptualized the economic system as a system of equations, known as Structural Equation Models

(SEM).[4] In SEMs, each equation incorporates the influenced variable on the left-hand side, and the variables that exert causal influence on it along with a stochastic error term on the right-hand side. This framework enables a detailed description and quantification of causal relationships among exogenous and endogenous variables, as well as among endogenous variables themselves (Simon (1953)). The equality sign in the equations should be read as "is caused by", devoid of the symmetry inherent in its mathematical counterpart. Each equation embodies a counterfactual logic, shedding light on hypothetical outcomes in presence or absence of specific actions (such as policy interventions). The strength of the model is that, given a correct identification of the causal order, the causal parameters can be estimated using standard techniques like Ordinary Least Squares or Maximum Likelihood. The SEM founders explicitly articulate a causal reading. For example Haavelmo (1943) links each structural equation to a statement about a hypothetical controlled experiment[5], while scholars like Marschak (1950), Koopmans (1949), and Simon (1953) emphasized that SEM serves the purpose of addressing the hypothetical changes that can be induced by policy interventions.[6]

[4]The initial application of a system of equations to delineate and infer the extent of causal connections occurred beyond the realm of economics; it was in fact pioneered by the geneticist Wright (1921).

[5]See Pearl (2015) for a discussion on the explicit causal meaning implied by Haavelmo's works.

[6]The robust emphasis on causal reasoning by the founders of SEM has been shrunk over time. Numerous scholars have underscored the necessity of self-contained causal interpretation for a structural equation (see, for example, James et al. (1982) Bollen (1989)). In their seminal textbook on SEM, Schumacker and Lomax (2016) explicitly stated, "We often see the terms cause, effect, and causal modeling used in the research literature. We do not endorse this practice and therefore do not use these terms here". Leamer (1985) made the observation, "It is my surprising conclusion that economists know very well what they mean when they use the words "exogenous", "structural", and "causal", yet no textbook author has written adequate definitions". LeRoy (1995) goes as far as denying any causal interpretation for most of the structural parameters that economists and social scientists strive to estimate. The original causal connotation of the parameters in SEM has gradually been supplanted by an algebraic interpretation. However, this does not negate the causal logic behind SEM; instead, it underscores additional challenges in attaining a causal understanding.

The economists affiliated with the Cowles Commission emphasized the crucial task of accurately delineating causal structures to construct meaningful models (e.g., Koopmans (1949), Koopmans et al. (1953)). In tackling this challenge, practitioners relied on their theoretical convictions and core economic principles. As a result, the identifying restrictions varied depending on the researcher's theoretical stance, often imposing preconceived notions onto the data rather than allowing the data to speak independently. This methodological approach introduces arbitrary elements into the model, potentially yielding divergent conclusions among researchers even when analyzing the same dataset. This concern was articulated by Sims (1980), who criticized the multitude of assumptions required for econometric identification, suggesting that these could lead to the generation of "incredible" restrictions. In addressing this issue, Sims advocated treating every variable as endogenous, thereby avoiding interference from prior knowledge. This idea prompted the construction of Vector AutoRegressive model (VAR), emerging as one of the most widely used tools in macroeconometric literature.

The VAR model belongs to the econometric tradition pioneered by Wold (e.g., Wold (1960)) and focused on the process properties, where causality is defined in the sense of Granger (hereafter Granger-causality). Granger (1969) defined causality in terms of incremental predictability and later in terms of conditional probability in Granger (1980) and Granger and Newbold (2014). This conceptual framework does not accommodate a counterfactual interpretation of causality. For example, if a researcher determines that changes in the money supply do not Granger-cause changes in the price level, it does

not necessarily imply that an expansionary monetary policy will not lead to inflation.[7] Granger himself in Granger (1995) underscored the differences between these methodologies, emphasizing that Granger-causality lacks the counterfactual implications found in the structural approach.[8] Granger-causality, upon which VAR models are based, primarily assesses the predictive power of variables, making it especially valuable for forecasting purposes. In contrast, structural causality aligns more closely with the demands of policy analysis.

The limitations of the VAR model in terms of conducting counterfactual analyses prompted Sims et al. (1982) to propose a structural variant known as the structural VAR (S-VAR) (see also Sims et al. (1986)). This model redirects focus from the parameters to the covariance matrix of the system. The premise is that unobservable exogenous forces, not directly observed by the econometrician, cause oscillations in the economic system. As these shocks are deemed primitive (lacking common causes), treating them as approximately uncorrelated becomes a natural choice (Bernanke (1986)). In practical terms, this involves orthogonalizing the covariance matrix, with its decomposition contingent upon the causal structure of contemporaneous observations. The necessity to define contemporaneous causal structures has compelled practitioners to rely once again on their theoretical convictions, reintroducing the problems related to the arbitrary settings of identifying restrictions akin to those encountered in SEM methodologies.

[7] For detailed discussions on scenarios where structural and Granger causality converge, refer to White and Lu (2010) and White and Pettenuzzo (2014)

[8] This limitation has led to significant errors in economic literature, as discussed by Hendry and Mizon (1999)

A widely adopted method for imposing identifying restrictions is to employ a hierarchical causal ordering. This approach is favored for its practicality, both from an economic and mathematical standpoint Enders (2008).[9] In this hierarchical framework, one variable exerts contemporaneous influence on others while remaining unaffected by them. Specifically, the i^{th} variable contemporaneously affects all k^{th} variables where $k \geq i$, and it is in turn contemporaneously influenced solely by the j^{th} variables, where $j \leq i$. This hierarchical arrangement results in a parameterization of contemporaneous relationships structured as a lower triangular matrix. A common technique to achieve this parameterization involves decomposing the covariance matrix using the Cholesky decomposition.

Given a Cholesky decomposition, the alteration of the sequence of variables in S-VAR results in a corresponding adjustment to the estimated structural parameters. In a conference centered on a paper addressing the identification of S-VAR (Hoover et al. (2009)), Hoover illustrated the potential for divergent outcomes based on different choices of variable orders. Such variations can lead to significant errors in the counterfactual interpretation.[10] Thus, the careful selection of the contemporaneous causal structure is of paramount importance. Practitioners are used to address this challenge relying on economic theory, perpetuating the issue associated with the arbitrary selection of identifying restrictions.

[9]The hierarchical structure is preferred as it minimizes the number of required identifying restrictions. However, alternative methodologies have been proposed, such as Bernanke (1986) who used an explicitly structural model to orthogonalize the VAR residuals, or Blanchard (1989) who used Keynesian theory to set identifying restrictions

[10]Hoover illustrated this matter by presenting a scenario in which the impulse response function, derived from a specific variable order, anticipates an increase in Y in response to an increase in X. Subsequently, he demonstrated that by altering the variable order, the prediction could shift to a decrease in Y following an increase in X.

In parallel, beyond the realm of economics, researchers at UCLA (e.g. the contributions of Pearl (2009) and Verma and Pearl (1992)), and Carnegie Mellon University (CMU) (e.g. the contributions of Spirtes and Meek (1995), Spirtes et al. (2000), and Kwon and Bessler (2011)), were engaged in the development of algorithms designed for the inference of causal structures within non-temporal and non-experimental datasets. Their works are centered on the idea that it is possible to translate conditional and unconditional dependence and independence patterns into a directed acyclic graph (DAG), commonly referred to as a Bayesian graph (Pearl (1985)).

The primary aim of these methodologies is to uncover relationships among variables by identifying parents, ancestors, children, and descendants based on measures of conditional and unconditional association. The theoretical foundation lies on the idea that, despite the well-established principle that correlation does not imply causation, an associative measure necessarily conceals an underlying causal rationale (Reichenbach (1971)). The process of inferring causality hinges on the premise that, when conditioning the association measure with additional variables, its value experiences alterations contingent upon the direction of causality.

To illustrate how conditional and unconditional association measures can be used to infer causal graphs, consider three variables X, Y, and Z and the three fundamental causal structures: the chain, the fork, and the collider (depicted in Figure I.1).

A causal chain is a primitive graph where the variables are arranged in a sequential order, namely every variable influences the subsequent one. Conversely, a causal fork contains

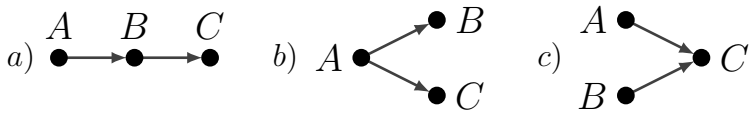

Figure I.1: Fundamental Causal Graphs with Three Variables: *a*) Causal Chain, *b*) Causal Fork, *c*) Collider

a single variable serving as the cause of multiple effects. In both scenarios, every pair of variables exhibits an unconditional association. However, in a causal chain, the association between two variables that are not directly linked disappears when the measure of association is conditioned on a variable mediating their causal path. Conversely, in a causal fork, no association between two child variables is discernible when conditioning on their parent.

The third primitive causal graph is the collider, which is the converse of a fork. In a collider, there is a single child variable influenced by multiple parent variables. In this scenario, only pairs including the child and some parents are unconditionally associated. However, the parents themselves become associated when conditioned on the child. This phenomenon, known as Berkson's paradox (Berkson (1946)), may appear counter-intuitive but can be clarified through a straightforward example. Consider a light bulb that illuminates only if both of two switches are turned on. These switches are operated by two individuals who have no means of coordination. Therefore, the state of one switch is entirely independent of the state of the other. However, if we examine only instances when the light bulb is illuminated (i.e., conditioning on a third variable), there will naturally be perfect correlation between the two switches.

These concepts have been integrated into various inferential algorithms, with the PC algorithm emerging as the most prominent (see Spirtes and Glymour (1991)). The PC algorithm operates through three sequential steps: identifying the skeleton, orienting the colliders, and directing the remaining edges. The algorithm initiates with a fully interconnected undirected graph, where each node is linked to every other node. Subsequently, edges corresponding to variables that are unconditionally or conditionally uncorrelated are removed. The algorithm then evaluates whether a variable can unblock the paths among unconditionally uncorrelated variables. If so, a collider is identified, initiating the process of assigning directionality to the relevant edges. The final phase of the algorithm involves assigning directions to the remaining edges based on logical criteria. This process adheres to the principle that causal relationships cannot form cycles, and any remaining undirected connections cannot lead to colliders. This systematic approach ensures the determination of the directionality of the residual edges.

Despite the PC algorithm has been initially developed for non-time series data, econometricians have adapted these techniques to suit their specific needs. In fact, graphical causal inference methods enable the identification of contemporaneous causal structures, and the resultant estimations are useful in establishing the identifying restrictions of an S-VAR model. The integration of the S-VAR model and the PC algorithm was first explored by Swanson and Granger (1997), who introduced a simplified version of the algorithm tailored for the hierarchical causal order required by the Cholesky decomposition. Demiralp and Hoover (2003) generalized the approach to a larger class of models and provided Monte

Carlo evidence to prove the effectiveness of the method. In a follow-up study, Demiralp et al. (2008) developed a bootstrap method that allows one to assess the reliability of a data-based identification scheme.

The macro-econometric literature provides several empirical applications of the S-VAR model integrated with the PC algorithm. For instance, Perez and Siegler (2006) applied the PC algorithm to investigate causal relationships among agriculture, money, interest rates, prices, and real GDP. The graphical method for causal inference has also found applications in applied microeconomics (e.g. Bessler and Akleman (1998)) and in the financial domain (e.g. Bessler and Yang (2003)).

The integration of the S-VAR model with the PC algorithm marks a significant advancement for counterfactual analysis in economics. This approach enables the estimation of a structural model without the subjective imposition of identifying restrictions. Naturally, certain implicit assumptions persist in this methodology. For instance, the requirement that the underlying structure be a Directed Acyclic Graph (DAG) indirectly imposes identifying restrictions. Additionally, the efficacy of the PC-algorithm depends on causal sufficiency and the assumption that the Data Generating Process (DGP) adheres to stability conditions.[11] Causal sufficiency entails that the graph correctly incorporates all common causes among each pair of variables, while stability condition is met when all probabilistic relationships, both conditional and unconditional, remain unchanged under variations in their numerical values (Pearl and Verma (1995)). In order to maintain the

[11] Referred to as the faithful condition by Spirtes et al. (2000) and DAG-isomorphism by Pearl (1988).

integrity of the causal inference process, it is also assumed that the variables follow specific distributions, such as normal (Pearl (1988)) or binomial (Meek (1995)). Furthermore, instances exist where (un)conditional patterns of (in)dependence generated by a specific causal graph are equivalent to those in another DGP linked to a different causal structure (Verma and Pearl (1991)).

To address these challenges, researchers have developed more versatile algorithms, such as the Cyclic Causal Discovery (CCD) algorithm by Richardson and Spirtes (1996), aimed at relaxing acyclicity constraints. Another notable advancement is the Fast Causal Inference (FCI) algorithm introduced by Spirtes et al. (2000), which relaxes the Markov condition. A thorough examination and comparison of various algorithms for causal structure discovery can be found in the comprehensive review by Sangüesa and Cortés (1997).

In the meantime, econometrics underwent a significant evolution marked by the discovery of the importance of non-stationarity and the emergence of cointegration theory. The term "cointegration" was introduced by Granger (1981), who initially hypothesized the existence of an error correction mechanism where a linear combination of $I(d)$ variables is $I(b)$ with $d > b$ (see Granger and Weiss (1983)). This innovative idea enabled the refinement of models that describe the variables' dynamic based on adjustments of long-term components, integrating cointegration in the error correction mechanism introduced by earlier works such as Sargan (1964), Phillips (1957), and notably by Davidson et al. (1978). The error correction mechanism inherently suggests that deviations from equilibrium in one

period are partially corrected in subsequent periods. This behaviour is typically estimated by regressing the changes in a series on the lagged values of a linear combination of two or more non-stationary series.

The foundational concepts laid out by Granger were subsequently expanded upon by Johansen (1988a) and Yoo (1986), leading up to the seminal contribution of Engle and Granger (1987), which introduced the Engle-Granger test, one of the most widely used tools in the econometric literature.

Johansen (1988b) integrated the concept of cointegration into the framework of Vector Autoregressive (VAR) models, creating the Cointegrated VAR (C-VAR). This advancement rendered the traditional VAR model obsolete, as it can now be seen as a special case of the C-VAR.[12] Further refinements of the model were presented in Johansen's subsequent works, such as Johansen (1991) and Johansen (1992b), and its application to various macroeconomic scenarios is evident in works such as Johansen and Juselius (1990) and Johansen and Juselius (1994).

The C-VAR model is situated within the process tradition and does not find direct application in structural analysis. Additionally, it incorporates two different unidentified causal structures: the short-term and the long-term. The former mirrors the approach taken in the Structural VAR (S-VAR), and can be estimated by employing the techniques previously discussed. In contrast, the long-term structure introduces additional complexities and nuances that are central to this thesis. This dual framework amplifies the complex-

[12]The classical equation of a C-VAR is $\Delta X_t = \Pi X_{t-1} + \sum_{i=1}^{k} \Delta X_{t-i} + \varepsilon_t$. By constraining Π to be 0, we derive the representation of a VAR(k) model.

ity of the identification problem, necessitating specialized methodologies for disentangling causal relationships in both temporal dimensions.

Only in recent years researchers initiated inquiries into the potential expansion of concepts derived from graphical causal inference to address the long-run identification problem. For instance, Malinsky and Spirtes (2019) proposed a methodology based on the Bayesian Information Criterion (BIC) value, while Hoover (2020) investigated the possibility of mapping cointegration and weak exogeneity patterns onto a Directed Acyclic Graph (DAG), proposing foundational rules for a long-run causal discovery algorithm.

Studying long-term causality holds significant importance in economic analysis. In fact, the disparity between long-term and short-term causality transcends mere temporal considerations. The demarcation lies in the stability of the relationship among variables. The former is predicated on an equilibrium concept, whereas the latter denotes the average influence that variations in one variable exert on others. Consequently, given that the vast majority of economic theories associate variables fluctuations to dynamic generated by equilibrium violation, long-term causality appears to be more coherent in describing economic mechanisms.

Long-term causality will be rigorously defined and explained throughout the thesis. Here, we provide a preliminary definition to initially elucidate the behaviour of the variables under study. We say that a variable y is caused in the long-term by a set of variables X if there exists an equilibrium relationship between y and X and violations of this equilibrium

trigger a reaction with either positive or negative adjustments of y leading to changes that tend to restore the equilibrium state over time. The impact on y is contingent upon the condition that all other factors remain constant and the comprehensive observation of all variables affecting the system. This last aspect will be particularly evident in the subsequent chapters of the thesis, where the concept of partially observed systems will be thoroughly addressed.

To illustrate the conceptual divergence between long and short term causality, consider two integrated variables X and Y governed by the subsequent data-generating process:

$$\begin{cases} \Delta Y_t = 0.5\Delta X_t + \varepsilon_t \\ \Delta X_t = \omega_t \end{cases} \tag{1}$$

where $\varepsilon \sim WN(0, \sigma_\varepsilon^2 > 0)$ and $\omega \sim WN(0, \sigma_\omega^2 > 0)$ and WN stands for white noise.

It is evident that the values of ΔY (and consequently Y) are influenced by interventions on X (namely on ΔX) while the converse is not true. This asymmetry is visually represented by the directed graph $X \to Y$.

In the given system of equations, there is no explicit equilibrium relationship specified between X and Y. Therefore, there does not exist any stable relationship to which the system tends; rather, it is just known that, on average, when X increases or decreases by a unit, Y correspondingly increases or decreases by 0.5. To illustrate this concept, we reported simulations of X and Y, where the observations are generated according to the aforementioned system of equations. The resulting series along with their ratio are

displayed in Figure I.2 and Figure I.3.

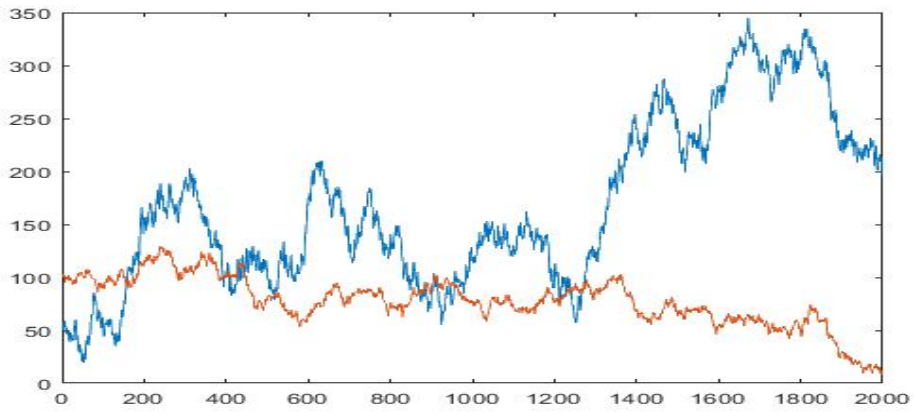

Figure I.2: Plot of Y (Red) and X (Blue) Generated According to 1. $\sigma_\varepsilon^2 = \sigma_\omega^2 = 1$. Number of Obs.=2000. Initial Values: $X_0 = 50$, $Y_0 = 100$.

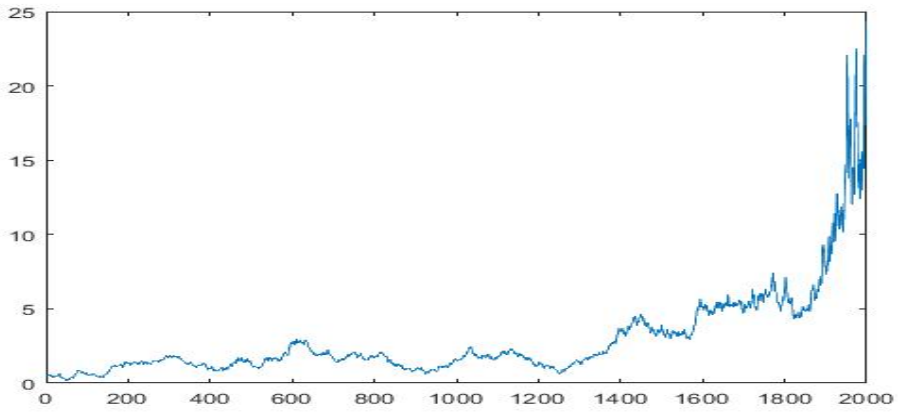

Figure I.3: Graph of the Ratio $\frac{Y}{X}$ for the Series plotted in figure I.2

The variability observed in the ratio between the two variables suggests a lack of persistent equilibrium relationship between X and Y despite the impact of fluctuations in X on those in Y. This form of causal interaction is referred to as short-term causality.

Consider now the scenario where it exists an equilibrium relationship between X and Y. In this context, the presence of equilibrium implies that any deviation from this steady

state prompts a response from at least one of the variables to restore the equilibrium condition.[13] Let us assume that equilibrium is maintained when Y is half of the value of X (i.e., when $Y = 0.5X$). If this equality is disrupted, Y adjusts to return to this initial equilibrium. Specifically, when $Y - 0.5X$ is negative, Y should increase ($\Delta Y > 0$), while if $Y - 0.5X$ is positive, Y should decrease ($\Delta Y < 0$). This dynamic can be expressed by the equation

$$\Delta Y_t = \alpha(Y_{t-1} - 0.5X_{t-1})$$

where α indicates the speed of adjustment. For instance, if $\alpha = -1$, ΔY reacts by adjusting the entire amount of the previous period's disequilibrium.[14]

Now, suppose the speed is -0.6, and that X does not react to the disequilibrium. This situation is characterized by the following system of equations:

$$\begin{cases} \Delta Y_t = -0.6(Y_{t-1} - 0.5X_{t-1}) + \varepsilon_t \\ \Delta X_t = \omega_t \end{cases} \tag{2}$$

where $\varepsilon \sim WN(0, \sigma_\varepsilon^2 > 0)$ and $\omega \sim WN(0, \sigma_\omega^2 > 0)$.

Here, the disequilibrium status does not affect X but triggers a response in Y. This asymmetric relationship can be graphically summarised by the structure $X \to Y$, signifying that X causes Y. To clarify the divergence between these causality concepts, a simulation

[13] A steady state is an economic configuration in which all short-run variations have stabilized, and quantities are either constant or growing at a constant (exponential) rate Hoover (2001). Steady states represent the scenario implied by the equilibrium status

[14] In case $\alpha = -1$ the adjustment process lasts only one period. This actually imply short-term causality. Nevertheless, since it arises from the error correction mechanism we will treat this case as a particular instance of long-term causality.

of X and Y was performed using the data-generating process described in Equation 2. The resulting series and their ratio are depicted in Figure I.4 and Figure I.5.

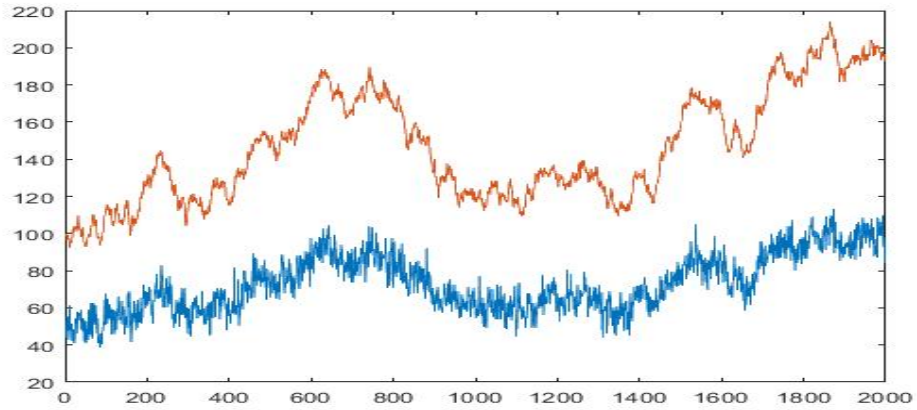

Figure I.4: Plot of Y (Blue) and X (Red) Generated According to 2. $\sigma_\varepsilon^2 = \sigma_\omega^2 = 1$. Number of Obs.=2000

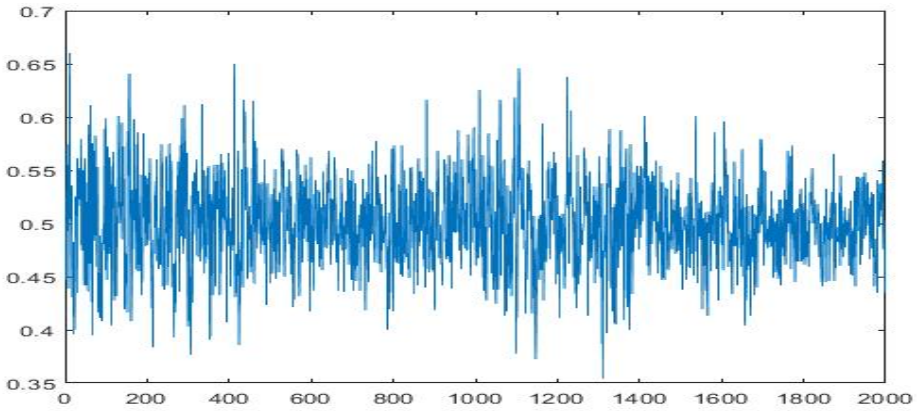

Figure I.5: Graph of the Ratio $\frac{Y}{X}$ for the Series Plotted in Figure 2.4

Notably, the relationship between the two variables remains stable over time. For this reason, we refer to this type of causality as long-run causality.

Long and Short term causality can also coexist. Consider the following system of equations:

$$\begin{cases} \Delta X_t = 0.6(X_{t-1} - 0.5Y_{t-1}) + \varepsilon_t, & \varepsilon \sim WN(0, \sigma_\varepsilon^2) \\ \Delta Y_t = 0.5\Delta X_t + \omega_t, & \omega \sim WN(0, \sigma_\omega^2) \end{cases}$$

This system exhibits both long-run and short-run causal structures. The former is characterized by the graphical representation $Y \to X$, indicating that Y influences X in the long-run. Conversely, the short-run causal structure is represented as $X \to Y$ signifying that X influences Y in the short term.

Up to this point, our examination has focused on simplified cases involving only two variables. Natural extensions of these systems of equations can be expressed in vector auto-regressive forms. For short-run causality, either the simple VAR or the structural VAR models can be employed. Conversely, to capture long-run causality, it is necessary to use the Cointegrated VAR (C-VAR) in its Vector Error Correction Model (VECM) representation. When both short and long-term causality are present, the multivariate extension is represented by the C-VAR(k) model with $k > 1$, indicating a VECM with dynamics spanning both short and long term behaviour.

To clarify, consider a causal structure delineated by the graph in Figure I.6. We begin by considering causal relationships of a short-term nature, such that the DGP is represented by a simple VAR model. For the sake of simplicity, assume that the influences after interventions manifest with a time distance of one period. This temporal separation enables the representation of the DGP using a simple VAR(1) model of the form $\Delta X_t = \Phi \Delta X_{t-1} + \varepsilon_t$.

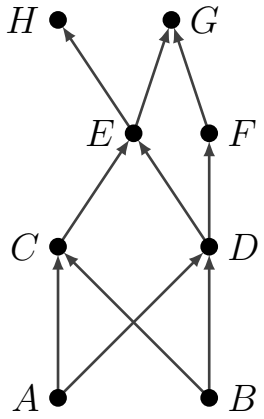

Figure I.6: Causal Graph Representing the Long-Term Causal Structure

Incorporating the causal graph depicted in Figure I.6 into a VAR model is straightforward. It just requires assigning a non-zero value to the coefficients linking the caused and causal variables, while setting to null those coefficients linking variables not directly connected in the graph. Therefore, the graph in figure I.6 is easily translated into a VAR as follows:

$$\Delta \begin{bmatrix} A \\ B \\ C \\ D \\ E \\ F \\ G \\ H \end{bmatrix}_t = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ * & * & 0 & 0 & 0 & 0 & 0 & 0 \\ * & * & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & * & * & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & * & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & * & * & 0 & 0 \\ 0 & 0 & 0 & 0 & * & 0 & 0 & 0 \end{bmatrix} \Delta \begin{bmatrix} A \\ B \\ C \\ D \\ E \\ F \\ G \\ H \end{bmatrix}_{t-1} + \varepsilon_t$$

where ε is an 8-dimensional white noise and $* \in (-\infty, 0) \cup (0, \infty)$.[15]

[15]Note that $*$ represents a generic nonzero number, and the $*$'s values in the matrix need not correspond

The natural multivariate extension of Equation 2 is the VECM(0), represented by the following equation:

$$\Delta X_t = \alpha\beta' X_{t-1} + \varepsilon_t$$

Here, X_t is a $p \times 1$ vector, α is a $p \times r$ matrix containing the speeds of equilibrium adjustment, $\beta' X_{t-1}$ is an $r \times 1$ matrix representing the deviations to the long-run relationship, and r is the cointegration rank.

Incorporating the causal structure in Figure I.6 is slightly more involved. The initial step involves analyzing the equilibrium relationships to determine the entries of the coefficient matrix β'.

The first equilibrium relationship of the graph in figure I.6, namely the one between C, A, and B, is described by the equation $C = \beta_1 A + \beta_2 B$. As for linear regression, the equal sign is not a symmetrical relation of identity. Rather, it is closer to the assignment concept. The equal sign should be read as "is caused by" or "is driven by", and the equality can be viewed as an experiment. For this reason, the coefficient that multiplies C should be set to unity.[16] It follows that the first row of the β' matrix will have two non-zero coefficients in the columns corresponding to A and B, and a unit entry in the column corresponding to C. The same logic applies to the second equilibrium relationship, i.e., the one between D, A, and B. Every successive equilibrium relationship is incorporated similarly.

to the same numerical values: each one can be a distinct nonzero number.

[16]This is what we implicitly do in linear regression. When we identify the dependent variable, we restrict its coefficient to be equal to 1.

The resulting β' matrix takes the following form:

$$\beta' = \begin{bmatrix} * & * & 1 & 0 & 0 & 0 & 0 & 0 \\ * & * & 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & * & * & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & * & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & * & * & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & * & 0 & 1 \end{bmatrix}$$

and the matrix $\beta' X_t$ containing the dis-equilibria at time t is

$$\beta' X_t = \begin{bmatrix} *A + *B + C \\ *A + *B + D \\ *C + *D + E \\ *D + F \\ *E + *F + G \\ *E + H \end{bmatrix}_t$$

Next, it is derived the α matrix containing the speeds to which the variables adjust to achieve the equilibrium. According to the definition of long-run causality, variables that are not directly caused do not adjust when a disequilibrium occurs. Hence, variables A and B remain unaffected by any disequilibrium, resulting in zero vectors for their respective

rows in α[17]. On the other hand, C adjusts when $C \neq *A + *B$, thus the row of α corresponding to C will have a non-zero element in the column corresponding to the first long-run relationship. This principle extends similarly to the adjustment of D and the other directly caused variables.

Thus, the resulting adjustment matrix is structured as follows:

$$\alpha = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ * & 0 & 0 & 0 & 0 & 0 \\ 0 & * & 0 & 0 & 0 & 0 \\ 0 & 0 & * & 0 & 0 & 0 \\ 0 & 0 & 0 & * & 0 & 0 \\ 0 & 0 & 0 & 0 & * & 0 \\ 0 & 0 & 0 & 0 & 0 & * \end{bmatrix}$$

[17]This condition matches weak exogenous. It is said that A and B are weakly exogenous with respect to the cointegration matrix in the C-VAR modeling $Y = \{A, B, C, D, E, F, G, H\}$

The complete VECM is then given by:

$$\Delta \begin{bmatrix} A \\ B \\ C \\ D \\ E \\ F \\ G \\ H \end{bmatrix}_t = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ * & 0 & 0 & 0 & 0 & 0 \\ 0 & * & 0 & 0 & 0 & 0 \\ 0 & 0 & * & 0 & 0 & 0 \\ 0 & 0 & 0 & * & 0 & 0 \\ 0 & 0 & 0 & 0 & * & 0 \\ 0 & 0 & 0 & 0 & 0 & * \end{bmatrix} \begin{bmatrix} * & * & 1 & 0 & 0 & 0 & 0 & 0 \\ * & * & 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & * & * & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & * & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & * & * & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & * & 0 & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \\ D \\ E \\ F \\ G \\ H \end{bmatrix}_{t-1} + \varepsilon_t$$

■

Consider the causal structures illustrated in Figure I.7[18], which combines both long-run and short-run dynamic. Incorporating the techniques from previous examples results in a Data Generating Process that encompasses both short and long-run structures, as the one illustrated in the figures. The resulting model adopts the form of a C-VAR(k) with $k > 1$. Assuming that short-run causality affects variables after one lag, we can adopt the Vector Error Correction Model (VECM) representation of a C-VAR(2). The matrix representing short-term dependencies includes non-zero entries corresponding to the relationships between C and D, A and C, and B and C.

Conversely, the cointegration matrix embodies three equilibrium relationships: $B = *A$,

[18]For the sake of conciseness, the graph related to the short term dependencies is depicted disregarding the time index. Nevertheless, it is implied that the relation $A \to B$ must be considered as $A_t \to B_{t+1}$.

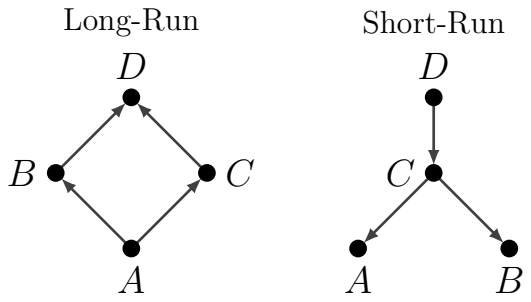

Figure I.7: Causal Graphs Representing the Short and Long-Term Causal Structures

$C = *A$, and $D = *B + *C$. The adjustment matrix consists in an empty first row and a single element in each of the remaining three rows. Specifically, it must delineate the influence that the first, second, and third equilibria exert on B, C, and D, respectively. The final model is expressed as follows:

$$\Delta \begin{bmatrix} A \\ B \\ C \\ D \end{bmatrix}_t = \begin{bmatrix} 0 & 0 & 0 \\ * & 0 & 0 \\ 0 & * & 0 \\ 0 & 0 & * \end{bmatrix} \begin{bmatrix} * & 1 & 0 & 0 \\ * & 0 & 1 & 0 \\ 0 & * & * & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \\ D \end{bmatrix}_{t-1} + \begin{bmatrix} 0 & 0 & * & 0 \\ 0 & 0 & * & 0 \\ 0 & 0 & 0 & * \\ 0 & 0 & 0 & 0 \end{bmatrix} \Delta \begin{bmatrix} A \\ B \\ C \\ D \end{bmatrix}_{t-1} + \varepsilon_t$$

Despite this last case can be of significant importance for specific economic scenario, throughout this thesis we will focus exclusively on long-term dynamics, excluding the possibility of data-generating processes given by cointegrated VAR of order higher than 1.

This thesis contributes to the econometric literature by developing new tools for inferring long-run causality. Although it is inspired by previous work on the topic, particularly by Hoover (2020) and generally by the works on graphical causal inference, the proposed

techniques diverge from the standard approach. Instead of focusing directly on translating statistical patterns into causal graphs, this work aims to define novel causal properties that can be estimated using standard hypothesis testing procedures. Thus, the technique presented here aligns with classical statistical methodologies departing from the iterative algorithmic procedures proposed by earlier literature on this topic.

The statistical concept on which we built our approach and drew inspiration is weak exogeneity, particularly within the cointegration framework. Weak exogeneity has often been associated with concepts pertinent to the causal perspective. For example, it has been linked to variables that remain unaffected by others within the system or to the notion of driving trends, i.e., variables that govern the long-term dynamics. Simultaneously, weak exogeneity manifests as model restrictions, enabling the construction of standard inferential procedures for its identification. This dual nature of weak exogeneity allows hypotheses related to causal assertions to be translated into hypotheses related to model restrictions, which are subject to statistical identification. Consequently, by testing the latter, one can analyse the former.

Nevertheless, weak exogeneity is a concept developed exclusively for statistical purposes, and its causal interpretation has often been hazardous since prior to Hoover (2020) there was little effort to rigorously analyse the relationship between structural causality and weak exogeneity. Moreover, economists have already generally recognized that statistical exogeneity has limited applicability to macroeconomic issues. Despite the central importance of identifying exogenous variables in macroeconomics, this identification cannot

generally be achieved by examining weak exogeneity. As stated in Sieroń (2019) p.332:

> Now, it should be clear that the debate is not about exogeneity in the statistical sense, [...] (in this type of exogeneity), what really matters is the adequacy of the model, not the nature of the variables per se. [...] The confusion stems from the fact that what really matters is causality, not exogeneity.

Inspired by this observation, we recognized the necessity for a refined formulation of the concept of exogeneity—one that provides a rigorous definition while maintaining compatibility with the causal discourse essential in macroeconomic analysis. Consequently, we introduced a novel definition of exogeneity anchored to the latent causal structure governing the data generating process of economic systems.

In recognition of the causal underpinning of this new definition of exogeneity, we have termed it "causal exogeneity." This novel conceptualization underscores the fundamental roots of the terms endogeneity and exogeneity. Etymologically, "endogenous" signifies "internally generated" while "exogenous" means "externally generated". Our approach defines internal and external generation based on the causal graph governing the system. Specifically, given the set of modeled variables, which represent just a portion of the nodes of the causal graph governing the whole system, if one of its elements has all its parents among the observed variables, it is categorized as internally generated. Conversely, if some of its parents are outside this subset, it is considered externally generated. Importantly, this definition also carries a counterfactual implication, since it implies that causally exogenous variables wield full manipulative influence over the nominal values of causally

endogenous variables.

Throughout this thesis, we explored whether causal exogeneity manifests as a model property. As we will prove in the first two chapters, causal exogeneity corresponds to model's constraints, allowing us to translate these constraints into causal hypotheses. Importantly, these model restrictions can be empirically tested using standard statistical techniques. Consequently, we can develop statistical tests that indirectly assess causal properties. This approach parallels that of weak exogeneity, as previously discussed, but with a crucial distinction: the new concept is inherently grounded in the causal structure of the system, thereby avoiding ambiguity stemming from its causal interpretation.

This approach addresses the critique raised in Sieroń (2019). However, the same paper presents another concern regarding the use of statistical exogeneity in the analysis of macroeconomic issues related to exogeneity:

> In econometrics and Statistics, exogeneity means that a variable is independent of other variables, while endogeneity implies a variable is jointly determined with other variables in the system. Of course, in reality all variables are interrelated. Hence, in a statistical sense, whether a variable is exogenous or endogenous can only be determined in the context of a particular model (explicitly constructed) (Sieroń (2019) p.332).

At first glance, our approach only partially addresses this critique. The definition of causal exogeneity hinges on the model, contingent upon the identification and inclusion of specific variables in the analysis. Hence, varying the selection of variables can modify

the properties of causal exogeneity. This implies that causal exogeneity is not an inherent characteristic of a variable itself, but rather a property that can only be defined with respect to other variables within the model.

However, we need to emphasize two key aspects. Firstly, the property of causal exogeneity is not dependent on the model in the sense of the selected structure. This implies that it does not alter based on arbitrary specifications, such as identification restrictions imposed by the model user, but rather depends solely on the variables included in the analysis. Secondly, the critique hinges on the insufficient focus on the complex interrelationships among economic variables. However, our approach is grounded in the properties of the underlying causal graph that defines the causal relations among all variables influencing a system. This includes every causal connection, even those that are mediated by unknown and un-modeled factors. Therefore, while it is accurate to say that the definition of causal exogeneity is model-dependent, it does not imply a lack of consideration for the complexity of real economic systems.

Our approach offers a valuable tool for analyzing macroeconomic issues through inferential procedures that prioritize causality while acknowledging the intricate structures arising from the multitude of social relations inherent in economic systems. Hence, we believe that our new approach takes Sieron's critiques seriously and effectively addresses them. Naturally, we recognize that the method represents a reconciliation between statistical methodologies and causal reasoning, necessitating compromises. However, while certain nuances may remain unaddressed, we posit that these new tools can effectively address

macroeconomic issues by rigorously incorporating causal reasoning.

The thesis is structured into three chapters. The first chapter defines the concept of causal exogeneity, delineating both its weak and strong forms. It proves that these concepts are manifest in the model properties, demonstrating how this duality facilitates the translation of causal hypotheses into model constraints. Additionally, the chapter develops inferential procedures based on standard techniques for indirectly testing causal assertions. Chapter 2 delves deeper into weak causal exogeneity, presenting an alternative technique for its identification. It includes results from simulations and discusses the performance of the proposed tests. Furthermore, the chapter illustrates the practical utility of causal exogeneity through an empirical analysis focusing on monetary exogeneity and endogeneity. Finally, the third chapter analyses the causal insights derived from hypothesis testing on standard weak exogeneity. It investigates the extent to which weak exogeneity captures information pertaining to non-causality and driving trends. In conclusion, an empirical illustration based on theories of business cycles illustrates the correct causal interpretation of weak exogeneity.

Chapter 1

Identification and Inference of Long-Run Causal Exogeneity

1.1 Introduction

Attributing exogeneity to economic variables poses a complex challenge, and disagreements concerning their endogeneity nature have generated extensive debates. A notable example is found in monetary economics, where the characteristics of the money supply have sparked controversy since the 19th century, with conflicting perspectives from the Currency and Banking Schools (see e.g. Knapp (1924) and Menger (1871)). Disagreements have persisted in modern literature, with neoclassical synthesists and post-Keynesian scholars contributing to the discourse (e.g. Kaldor (1970), Moore (1989)). Although there currently is a widely accepted consensus that central banks determine interest rates and permit the quantity of reserves to fluctuate, the ongoing discourse remains both active and pivotal

within the realm of monetary theory, with significant consequences for policy-making. (see Sieroń (2019)).

The dispute over exogeneity extends beyond monetary economics. For instance, economists remain divided on the origins of business cycles, with some advocating for internal generation due to economic dynamics, while others support external origins attributed to shocks and disturbances (see for instance Gordon (1991)).

In parallel, the field of econometrics has recognized the fundamental significance of exogeneity for statistical purposes. Building upon the foundational work of Koopmans (1950), the celebrated paper by Engle et al. (1983) introduces distinct definitions of exogeneity, each tailored to specific econometric analyses.

Weak exogeneity is essential for efficient estimation and hypothesis testing within conditional models and it serves as a prerequisite for strong and super exogeneity. Strong exogeneity primarily relates to forecasting, while super exogeneity ensures the validity of policy simulations. Those concepts of exogeneity have been the subject of extensive study, leading to the development of inferential procedures for their identification. Weak exogeneity evaluation is relatively straightforward within cointegrated systems, as exemplified in studies such as Johansen (1992c). Strong exogeneity can be ascertained through a combination of weak exogeneity and standard Granger-causality tests, while super exogeneity is assessed by evaluating parameter invariance, as illustrated in Engle and Hendry (1993).[1]

[1]For further discourse on this subject, one may refer to the collection of papers in Ericsson and Irons (1994).

It is important to emphasize that while weak, and strong exogeneity are indispensable for empirical research, their identification serves statistical purposes and may not align with the notions of exogeneity underpinning the ongoing macroeconomic debates. In fact, numerous scholars have treated statistical exogeneity as distinct from the economic notion, emphasizing that economic deliberations prioritize causal understanding over statistical aspects.[2]

Super exogeneity is often linked to causality because it is a notion based on structural invariance of parameters. However, as will be demonstrated throughout this thesis, it does not provide information about the underlying causal structure. In fact, causality may even operate in the opposite direction to what is suggested by super exogeneity (see, for example, the discussion in Section 1.3 and other examples throughout the thesis). The primary purpose of super exogeneity is to assess whether the predictions derived from policy simulations based on the estimated model are valid. Ultimately, it is best understood as a statistical concept.

Kaplan (2004) suggests the dichotomy between statistical and causal conceptions of exogeneity has resulted in the formulation of ambiguous and confusing definitions. For instance, Cohen et al. (2013) define exogenous variables as those not influenced by any other variables within the system, except for other exogenous variables. However, this assertion lacks precision as it implies the possibility of one exogenous variable being dependent on

[2]This viewpoint has been articulated both prior to and subsequent to the publication of the seminal paper by Engle, Hendry, and Richard. For a comprehensive discussion, refer to Chick (1973) and Moore (1989).

another exogenous variable, which logically entails endogeneity. An alternative perspective is offered by Bollen (1989), who defines exogeneity based on the specifications imposed by the model user. Nonetheless, this definition suggests that a variable's exogeneity depends on the researcher's specification. As a result, a variable could be endogenous within a model but not truly endogenous in the underlying Data Generating Process (DGP), indicating that the model may not accurately represent the real-world system. Another common definition, proposed by Wonnacott and Wonnacott (1979), characterizes a variable as exogenous if its value is determined external to the system. However, the exact meaning of "determined outside the system" is not properly explained.

Starting from the heuristic notion that a variable is exogenous when it originates from outside the system, we introduce two novel definitions of exogeneity which are grounded in graphical representations of latent causal structures. The decision to anchor the definitions in causal graphs, as opposed to definitions of statistical exogeneity, as in Engle et al. (1983), means that, as well as complementing those of Engle et al. (1983), our definitions are perhaps closer to the layman's notion of "outside the system", while being clear and precise. The notions of Engle et al. (1983) have sustained empirical research over the last 40 years, but have not met with universal approval. We will systematically examine the differences between the notion of weak exogeneity and our proposed concepts of causal exogeneity, highlighting the instances where they diverge.

In this chapter we make a number of contributions. The first is the development of

new exogeneity concepts which complement those of Engle et al. (1983) and in some instances might better reflect macroeconomic notions of 'exogeneity'. The second is to show how these definitions of causal exogeneity translate to restrictions on the parameters of the cointegrated vector autoregressive model. Finally, we propose testing techniques to determine whether these restrictions hold in any given empirical setting.

The chapter is structured as follows. The next section sets out the underlying assumptions we make, and introduces the tools that will be employed throughout the thesis. In the third section, we describe our novel definition of exogeneity, which is grounded in the graphical representation of causal structures. This new definition, termed "causal exogeneity", has both weak and strong forms. Section 1.4 establishes a relation between model characteristics and weak causal exogeneity to derive corresponding restrictions associated with this status. Exploiting this duality, we propose an inferential procedure for its identification. Section 1.5 introduces the model counterpart of strong causal exogeneity and suggests a testing technique. Section 1.6 proposes an illustrative example, and section 1.7 offers concluding remarks and summarises the findings.

1.2 Underlying Model and Assumptions

Initially introduced in Engle and Granger (1987), the concept of cointegration has revolutionized econometric analysis involving time series. Their emphasis on the cointegrating relationship as the equilibrium, underscored the importance attributed to the "long run" in seminal contributions by Sargan (1964) and Davidson et al. (1978), closely aligning

statistical models with dynamic adjustment to economic theory relationships.

Subsequent works, notably those by Johansen and Juselius (e.g. Johansen (1988b) and Johansen and Juselius (1990)), model integrated-cointegrated variables in a Vector Autoregressive (VAR) framework. The cointegrated VAR (C-VAR) generalizes other VAR structures, in the sense that simple VAR and related models become special cases of the more comprehensive C-VAR.

The most common representation of a C-VAR is the Vector Error Correction Model (VECM). As our interest lies in conducting a causal analysis in the structural sense, we will work with the VECM in its structural version:

$$\Gamma_0 \Delta Y_t = \alpha \beta' Y_{t-1} + \sum_{i=1}^{k} \Gamma_i \Delta Y_{t-i} + \varepsilon_t \tag{1.1}$$

The variables in Y are I(1) cointegrated processes, consequently ΔY_t is stationary.

The coefficients $\Gamma_{i=0:k}$ describe the short-term behaviour of the system, while the matrices α and β contains the speed of equilibrium adjustment and the steady-state relationships (for a comprehensive explanation, please refer to Johansen (1995) and Juselius (2006)). The term ε represents primitive exogenous forces that are not directly observed by the econometrician but impact the system, leading to oscillations. Because these shocks are primitive, i.e., they do not have common causes, it is natural to treat them as approximately uncorrelated, as in the view of Bernanke (1986). Consequently, the covariance matrix $\Omega = \mathrm{Var}(\varepsilon)$ is, by construction, diagonal.

The coefficients α and β are respectively known as the adjustment and cointegration matrices. They are both $|Y| \times r$ full-rank matrices, where $|Y|$ represents the cardinality of the set Y encompassing the variables within the system, and $0 < r < |Y|$.[3] The value of r is commonly referred to as the cointegration rank. The product $\alpha\beta'$ yields the $|Y| \times |Y|$ matrix of reduced rank r, known as the impact matrix and denoted Π.

Henceforth, we will employ the notation Y to represent the set encompassing all factors influencing a system. Consequently, Equation (1.1) should be considered as a complete model describing the entire system's behaviour, with coefficients embodying relationships akin to natural laws. We will hereafter denote this model as the "fully observed model."

Obviously, the econometrician is not expected to observe and model the entire set Y (as will become evident later). Nevertheless, in pursuit of a substantive causal analysis, it becomes necessary to commence with the authentic natural relationships and discern how they would manifest within a partially observed model.

Throughout the thesis, we will impose the assumption that the structural dynamic of the system is entirely driven by long-term factors. In other words, the fluctuations in Y are solely caused by disequilibrium conditions and violations of steady-state. It is essential to clarify that this hypothesis applies to the fully observed model and does not necessarily hold in the partially observed system estimated by the researcher. This implies that if one empirically encounters short-term dependencies, this does not preclude the application of the methodologies proposed here, as they can be generated by the incompleteness of

[3] If $r = 0$, then the variables in Y do not cointegrate; conversely, if $r = |Y|$, ΔY_t cannot be I(0)

the model. The only implication of this assumption is that all dynamics are triggered by adjustment mechanisms, meaning that the value of a variable at time t is determined solely by an unexpected and unpredictable innovation if at time $t-1$ its value was in equilibrium (with respect to its parents). A further discussion on the implications of this assumption and the necessary extensions for its relaxation can be found in the conclusion.

From a model perspective, the absence of short-term dynamic implies that the coefficients $\Gamma_{i=1:k}$ become zero and the term Γ_o is the identity matrix. Thus, the C-VAR describing the behaviour of Y is of order 1, and Equation (1.1) reduces to

$$\Delta Y_t = \alpha\beta' Y_{t-1} + \varepsilon_t \tag{1.2}$$

It should be further emphasized that these assumptions apply to the fully observed model and do not extend to the partially observed model, as a partially observed C-VAR(1) exhibits short-term dynamics and a non-diagonal covariance matrix. This implies that encountering short-term dynamics in practical applications does not preclude the use of the techniques proposed in the thesis, as it is well understood that the partially observed model generated by the C-VAR(1) inherently includes both contemporaneous causal dynamics and short-term dynamics.

The primary rationale behind this assumption is to conform with the hypotheses established in Johansen (2019). Indeed, these findings will be extensively used in our analysis.

However, it is also plausible to recognize economic rationales for this assumption, as the prevailing body of economic theories is founded upon long-term dynamics and in many cases, modelling these long-term dynamics is adequate to enable a comprehensive analysis of the macroeconomic system. As noted in Hoover (2001) (pages 138-140):

> "Economists provided reasonably persuasive accounts of steady states and real-world economic configurations for which steady states are good approximations. Economic theory rarely provides persuasive accounts of short-run transitional phenomena. [...] Economic theory is largely about steady states. The economic of transitional dynamics has little empirical support and carries little conviction among the economists"

Another assumption addresses the origin of non-stationarity, which can occur in two ways: either from common stochastic non-stationary trends or from specific parameter settings. Throughout this thesis, we adopt the former view. The primary rationale behind this hypothesis can be again attributed to the necessity of aligning with Johansen's framework. Nevertheless, there exists compelling logical reasoning that inclines us towards favouring the notion that non-stationarity is introduced, as opposed to being internally generated by specific parameter settings. In fact, if the latter were the case, even a slight alteration in the coefficient values could lead to the loss of cointegration and the underlying trend behaviour of the ordinary variables. In contrast, the assumption of the existence of fundamental trends is of a generic nature, demonstrating robustness in the face of variations in the values of the structural coefficients. For a more detailed discussion, please refer to section

3 of Hoover (2020).

The subset of Y containing the fundamental trends will be denoted T. Variables within Y that are not part of T are considered ordinary variables and are grouped within the set X, such that $Y = \{T, X\}$. Additionally, $T \subseteq Y = Y \setminus X$, and $X \subset Y = Y \setminus T$, where the operator $\setminus$ denotes set exclusion: for two sets A and B, the expression $A \setminus B$ represents the set of elements in A that are not in B. It is important to note that the fundamental trends can constitute the set Y itself in cases where the system comprises non-cointegrated I(1) variables. However, X cannot coincide with Y as T is its original source of non-stationarity.

As highlighted in the introduction, the type of exogeneity we investigate is rooted in the graphical representation depicting the causal connections. It is evident that the relevant causal structure pertains to the one governing Y rather than any structure artificially generated by modelling only a subset of it. It is thus necessary to understand the connection between causal graphs and the properties of the fully observed VECM. When working with the model in Equation (1.2), the definition of causality revolves around equilibrium adjustments. Specifically, we assert that a variable a is causally influenced in the long-term by a set of variables B if it exists an equilibrium relationship between a and B that exerts an influence on the dynamics of a.

For a deeper comprehension of this concept, we report an example illustrating how to translate a causal network into a VECM. Consider the causal graph depicted in figure

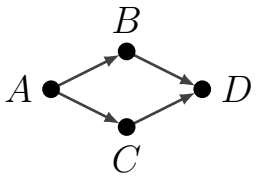

Figure 1.1: Causal Graph Representing the Causal Structure of Equation (1.3)

1.1. This graphical representation comprises three distinct causal relationships, each corresponding to an equilibrium condition: $A \to B \implies B = \beta_1 A$, $A \to C \implies C = \beta_2 A$, and $B \to D \leftarrow C \implies D = \beta_3 B + \beta_4 C$. These steady states are incorporated into the cointegration matrix β so that equilibrium deviations at a given time $t-1$ can be captured by the multiplication $\beta' Y_{t-1}$:

$$\beta' Y_{t-1} = \begin{bmatrix} -\beta_1 & 1 & 0 & 0 \\ -\beta_2 & 0 & 1 & 0 \\ 0 & -\beta_3 & -\beta_4 & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \\ D \end{bmatrix}_{t-1} = \begin{bmatrix} B_{t-1} - \beta_1 A_{t-1} \\ C_{t-1} - \beta_2 A_{t-1} \\ D_{t-1} - \beta_3 B_{t-1} - \beta_4 C_{t-1} \end{bmatrix}$$

where $\beta' Y_{t-1} = 0$ signifies equilibrium.

To induce causal behaviour, the model must be configured so that only the affected variables respond following manipulation of the causal factors, specifically reacting to conditions of disequilibrium. Therefore, for the causal link $A \to B$, the α matrix must be designed such that only B responds to equilibrium violations. This ensures that arbitrary alterations of A will elicit a response in B even if the other variables in the system (i.e., C and D) are kept constant. Moreover, the adjustment matrix must be structured to pre-

vent that manipulations of B affect the level of A. This captures the asymmetric nature of causality depicted in the graph $A \to B$. As a result, the initial steady state exclusively influences B, leading to the first column of the adjustment matrix containing a non-zero entry only in its second row (i.e. the row corresponding to B).

Similarly, the second and third steady states affect C and D respectively, leading to the second and third columns of the adjustment matrix containing non-zero entries solely in the third and fourth rows.

The resultant VEC model is:

$$\Delta \begin{bmatrix} A \\ B \\ C \\ D \end{bmatrix} = \begin{bmatrix} 0 & 0 & 0 \\ \alpha_1 & 0 & 0 \\ 0 & \alpha_2 & 0 \\ 0 & 0 & \alpha_3 \end{bmatrix} \begin{bmatrix} -\beta_1 & 1 & 0 & 0 \\ -\beta_2 & 0 & 1 & 0 \\ 0 & -\beta_3 & -\beta_4 & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \\ D \end{bmatrix}_{t-1} + \varepsilon_t \tag{1.3}$$

For a more comprehensive examination of the process of translating a causal graph into a VECM, please consult section 4 in Hoover (2020).

In the fully observed model, the rows of α corresponding to fundamental trends are null vectors, indicating weak exogeneity for the long-term parameter β. In fact, as proved for instance in Johansen (1992a), a variable is weakly exogenous for β if its corresponding row in the adjustment matrix contains only zero entries. Throughout this thesis, any mention of weak exogeneity will exclusively pertain to weak exogeneity in relation to the cointegration matrix. Thus, for brevity, the complete statement will not be reiterated, and

references to weak exogeneity are implied to be in relation to the long-term parameter.

As previously discussed, it is probably neither practical nor feasible to assume the econometrician observes the entire set of variables Y, given that this set may include a multitude of factors that are challenging or even impossible to gather or measure. Consequently, the set Y will be partitioned into two subsets: the set of observed variables, denoted as X_o and the set of latent factors, denoted as Y_u.

It is supposed that the fundamental trends are in the set of unobserved variables. This assumption is motivated by two distinct considerations. The first rationale pertains to the nature of stochastic trends. Indeed, the components within T consist of unpredictable and independent shocks that can originate from various disparate sources, such as climate disasters, political instabilities, or shifts in social behaviour. However, these factors are typically not simultaneously collected, and their potentially non-economic nature poses challenges in terms of their incorporation into economic models.

Secondly, when examining the issue of exogeneity in models containing T or a subset of it, the analysis becomes straightforward. This is because the elements within T represent the exogenous components *par excellence*, and their identification can be readily achieved through conventional weak exogeneity tests.

Consequently, the set of latent variables Y_u encompasses both the fundamental trends T and the unobserved ordinary variables denoted as X_u. The interrelations among these sets can be readily established: $Y = \{T, X_o, X_u\}$, $X = \{X_u, X_o\}$ and $Y_u = \{T, X_u\}$.

The set X_o encloses the variables that are explicitly modelled. Consequently, the estimated model will be:

$$\Delta X_{o,t} = \alpha_o \beta_o' X_{o,t-1} + \sum_{i=1}^{\infty} \Gamma_{o,i} \Delta X_{o,t-i} + \varepsilon_{o,t} \tag{1.4}$$

where the subscript o is added to make a distinction between the coefficients of the fully and the partially observed model.

Clearly, the assumptions imposed on model (1.2) do not need to hold in the partially-observed system, Equation (1.4). Consequently, short-term dynamics arise[4], as well as the possibility that the covariance matrix $\Sigma_o = \text{Var}(\varepsilon_o)$ is not diagonal.

The analysis will naturally focus on α_o and β_o given that these matrices represent the only estimable components accessible to the model user. Indeed, even the existence of Y_u might be completely ignored by the researcher.

The values of α_o and β_o will be analysed employing the innovative methodologies introduced by Johansen (2019). Commencing with the fully observed model arranged in a state-space framework, Johansen demonstrated that the β_o' can be reformulated as follows:

$$\beta_o' = \left(N_o - M_{ou} M_u^{-1} N_u\right)_\perp' \left(M_o - M_{ou} M_u^{-1} M_{uo}\right) \tag{1.5}$$

where the coefficients are taken from the state-space representation below:

$$\Delta X_{o,t} = M_o X_{o,t-1} + M_{ou} X_{u,t-1} + N_o T_{t-1} + \eta_{o,t}$$

[4] As proved in Johansen and Juselius (2014), a partially observed C-VAR(k) is of order ∞

$$\Delta X_{u,t} = M_{uo} X_{o,t-1} + M_u X_{u,t-1} + N_u T_{t-1} + \eta_{u,t} \tag{1.6}$$

$$\Delta T_t = \eta_{T,t}$$

Similarly, the adjustment matrix of the partially observed VECM is obtained as:

$$\alpha_o = \Sigma_o \Big(M_{ou} V_{uT} + N_o V_T \Big)_\perp \tag{1.7}$$

Here, $\Sigma_o = \mathrm{Var}(\varepsilon_o)$ is calculated as:

$$\Sigma_o = \mathrm{Var}(\varepsilon_o) = \begin{bmatrix} M_{ou} & N_o \end{bmatrix} V \begin{bmatrix} M'_{ou} \\ N'_o \end{bmatrix} + \Omega_o \tag{1.8}$$

The matrix Ω_o is the square diagonal matrix constructed by selecting the entries of $\Omega = \mathrm{Var}(\varepsilon)$ corresponding to X_o.

The matrix V is defined as:

$$V = \mathrm{Var} \begin{bmatrix} X_{u,t} \\ T_t \end{bmatrix} \Big| X_{o,t} \Big] = \begin{bmatrix} V_u & V_{u,T} \\ V_{T,u} & V_T \end{bmatrix}$$

where $\mathrm{Var}(A|B)$ indicates the covariance matrix of A conditioned on B.

The matrix V can be found by solving the algebraic Riccati equation below:

$$V = Q^* V Q^{*'} + \Omega^* - Q^* V C^{*'} (C^* V C^{*'} + \Omega_o)^{-1} C^* V Q^{*'} \tag{1.9}$$

where the coefficient Q^* relates $Y_{u,t}$ to $Y_{u,t-1}$ and C^* relates $X_{o,t}$ to $Y_{u,t-1}$. The matrix Ω^* is constructed considering the rows and columns of Ω corresponding to Y_u. Therefore

$$Q^* = \begin{bmatrix} I + M_u & N_u \\ 0 & I_T \end{bmatrix} \qquad C^* = \begin{bmatrix} M_{ou} & N_o \end{bmatrix}$$

where I_T indicates an identity matrix of dimension $|T|$.

The orthogonal complement $A_\perp$ of a matrix A with dimensions $a \times b$ and rank r is an $a \times (a - r)$ matrix that fulfils the condition $A'A_\perp = 0$. It is important to emphasize that $A_\perp$ does not possess a unique representation and can assume an infinite number of distinct specifications.[5]

Equations (1.5), (1.7), and (1.8) will be extensively employed to explore potential correspondences between the coefficients of the partially observed model and specific characteristics of the causal graph $\mathcal{G}$. In other words, we aim to establish links between the parameter set $\{\alpha_o, \beta_o, \Sigma_o\}$ and the causal relationships depicted by the graph incorporating the causal connections implied by the data generating process (DGP) governing Y.

[5]This attribute is closely linked to the notion of the cointegration space. Indeed, there is not a unique matrix that generates stationary linear combinations of X_o; instead, there exists a potentially infinite number of matrices that can be found by selecting different specification of the orthogonal complement $(N_o - M_{ou}M_u^{-1}N_u)_\perp$

1.3 Causal Exogeneity

To define causal exogeneity, we will start from the heuristic idea that the nominal value of an exogenous variable is generated outside the system, while the level of an endogenous variable is determined within the system.

The partition of Y into T, X_o and X_u provides an initial framework for understanding exogeneity in these terms. Specifically, if one of the factors contributing to the long-term fluctuations of an observed variable $x_i \in X_o$ is found within the set encompassing latent components, we can classify the variable x_i as being generated external to the system, thereby exogenous. Conversely, $x_i \in X_o$ is endogenous if all of the factors contributing to its long-term fluctuations are included in X_o.

In terms of the causal graph $\mathcal{G}$, we can ascertain the endogeneity of x_i by examining the set of adjacent vertices that possess directed edges pointing towards the node corresponding to x_i. If this set comprises only variables included in X_o, then x_i can be considered endogenous.

The definition can be reformulated using terminology commonly employed in the field of causal inference.[6] With reference to the causal graph $\mathcal{G}$, a variable x_i is deemed exogenous if, within the parent set of x_i (henceforth denoted as $\mathcal{P}_Y(x_i)$[7]), at least one element belongs

[6] In the context of causal inference, causal connections are described using structures that parallel family relations. Parents refer to variables that directly influence a given variable of interest, denoted as child. If we consider also indirect causal connections, we refer to ancestors and descendants. For a comprehensive overview of causal inference terminology, refer to Pearl (2009) among others

[7] The subscript Y in $\mathcal{P}_Y(x_i)$ is added to specify that the parent set encompasses all parents of x_i within the set Y

Figure 1.2: Causal Influence Transmitted through Latent Variables

to $Y_u = \{T, X_u\}$. Mathematically, x_i is considered exogenous if $\exists\ u \in \mathcal{P}_Y(x_i)$ such that $u \in Y_u$.

However, this definition fails to fully capture the complexities of exogeneity, and requires further refinement, namely, a more nuanced notion of endogeneity. We assume the ultimate objective of differentiating between exogenous and endogenous variables is to discern which variables possess the capacity to exert full influence over others. We say that a set of variables Z has full influence over x_i if, for a given target value of x_i, denoted as $x_i = x_i^*$, there exists a combination of values $Z = Z^*$ such that setting Z to Z^* causes x_i to take on the value x_i^*. Hence, the differentiation between exogenous and endogenous variables should fundamentally hinge upon the capability of exogenous variables to control the nominal values of the endogenous.

The definition above fails to account for the possibility that the influence from exogenous to endogenous variables passes through latent components.

A deeper understanding of this concept can be facilitated through the use of a simple example. Consider the causal structure illustrated in Figure 1.2. The nodes highlighted in red and the red dashed edges signify, respectively, the unobserved variables and the latent causal connections. The set $Y = \{A, B, C, D\}$, can be partitioned into the following sets: $T = \{A\}$, $X_o = \{B, D\}$, and $X_u = \{C\}$.

In accordance with the initial definition, within the partially observed model, both variables B and D are classified as exogenous. This classification arises from the fact that $\mathcal{P}_Y(B) = A \in Y_u$ and $\mathcal{P}_Y(D) = C \in Y_u$. However, it should be noted that the variable D is fully manipulable through interventions directed at B.

To better understand the necessity of formulating a definition based on the concept of control, it is instructive to consider a policy-oriented perspective. Let us assume that the variable B represents the interest rate i, while D denotes the inflation rate π. According to the causal graph, policymakers possess the capability to exert influence over the inflation rate (π) through interventions targeted at the interest rate (i). Consequently, from a causal perspective, it may seem intuitive to classify the inflation rate as endogenous and the interest rate as exogenous.

However, this causal relationship is inter-mediated by an additional aspect. For instance, it is plausible to posit that higher inflation rates result in reduced consumer spending, subsequently leading to a decline in prices. Therefore, the presence of the node C representing expenditures (denoted as Ex), introduces a nuanced dimension whereby inflation rate is generated outside the system, despite being entirely governable through manipulations of i.

The potential multitude of intermediary phenomena is a challenge to the definition of exogeneity. To illustrate, even if we were to incorporate expenditures into the observed components, there might still be additional variables interposed between i and Ex. Specifically, i exerts its influence on expenditures by impacting borrowing levels. Furthermore,

the interest rate affects borrowing through its influence over the retail rates established by financial institutions which, in turn, are influenced by the inter-bank rates. This chain of interrelated factors can persist *ad infinitum*, posing challenges in identifying a primary, irreducible causal link, which may, in fact, prove elusive.

The example underscores the necessity for a definition of causal exogeneity based on the ability to control rather than the source of generation. However, before proposing a broader definition, it is essential to clarify the concept of forward paths. In graph theory and especially in the context of directed graphs, a forward path refers to a sequence of vertices connected by directed edges that follow the direction of the arrows. It typically describes a path that moves from one vertex to another without backtracking or reversing the direction of the edges. Consider a simple directed graph $\mathcal{G} = (V, E)$, where $V = \{A, B, C, D\}$ is the set of vertices and E represents the set of directed edges between nodes. Define the edges as follows: $A \to B$, $A \to C$, $B \to D$, and $C \to D$. In this graph, there is a forward path from A to D via B: $A \to B \to D$. Similarly, there also is a forward path from A to D via C: $A \to C \to D$. These paths are forward paths because they follow the direction of the edges from the starting node A to the ending node D.

Moving on to the concept of blocked forward paths, a forward path between nodes x and y is deemed blocked by an intermediate node z if z intervenes in the causal path between x and y. For example, the forward causal path $A \to B \to D$ is blocked by B, whereas the forward causal path $A \to C \to D$ is blocked by C.

We can now establish the foundation for a novel definition of exogeneity. When analysed

within the framework of graphical models, a set of variables denoted as $Z \in X_o$ possesses the capacity to exercise control over a variable $x_i \in X_o$ if i) the forward paths originating in T and ending in x_i are blocked by Z and ii) if the set of variables W transmitting causality from Z to x_i does not cause in turn Z.

Causally speaking, requirement i) is satisfied if the causal paths originating from the primary unobserved source of long-run fluctuations (e.g., fundamental trends) and terminating in x_i pass through Z. This ensures that the dynamics of x_i are fully influenced by Z. Specifically, under condition i), holding Z constant will keep x_i constant, except for unexpected shocks captured by the error term.

Requirement ii) is necessary to ensure that control over x_i is not nullified by uncontrollable components. If the set W in turn causes Z, the desired effect on x_i becomes unstable and this instability cannot be managed, as W is not identified and, thus, not controllable by the modeler.

To present a more rigorous definition, it is necessary to establish a novel partition of the set X_o:

Definition 1 (***Set*** $\beth$)**.** *A generic variable $x_j \in X_o$ is incorporated into the set $\beth \subset X_o$ if i) every directed path starting from the fundamental trends and concluding at x_j is blocked by elements in $Z \subseteq X_o \setminus x_j$ and ii) the ancestors of x_j belonging to X_u whose paths with T are blocked by Z are not in turn ancestors of Z*

Symmetrically, its complementary set $\aleph$ is defined as follows:

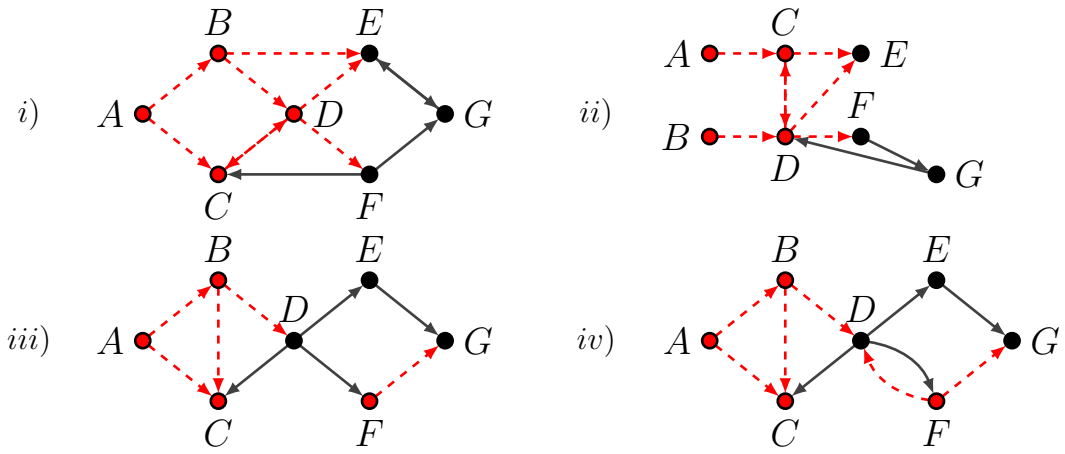

Figure 1.3: Causal Graphs and Causal Sets. Red nodes depict latent variables

Definition 2 (***Set*** ℵ)**.** *The set* ℵ *is the complementary set of* ב *with respect of* X_o*, i.e.* $\aleph = X_o \setminus$ ב

The set of ordinary unobserved variables X_u is instead partitioned as follows:

Definition 3 (***Set*** ך)**.** *A generic variable* $x_p \in X_u$ *is included in the set* $ך \subseteq X_u$ *if it is an ancestor of at least one element in* ℵ

Definition 4 (***Set*** ל)**.** *A generic variable* $x_q \in X_u$ *is included in the set* $ל \subseteq X_u$ *if it is an ancestor of at least one element in* ב *and it does not belong to* ך

It should be noted that the union of these sets does not necessarily result in the set Y as there may be elements in Y_u that belong neither to ך nor to ל. However, this implies that they do not exert any effect on the observed variables, either directly or indirectly, and therefore, they can be excluded from the analysis.

To clarify the set decomposition, consider the graphs in Figure 1.3. In graph *i*), the original source of long-term fluctuations is A, i.e. the set containing the fundamental trends is

$T = A$. The observed and unobserved variables are $X_o = \{E, F, G\}$ and $X_u = \{B, C, D\}$, respectively. Among X_o, the only variable whose forward paths with T are blocked by other elements in X_o is G. In fact, both E and F lie between A and G. Conversely, between A and E as well as between A and F, only variables in X_u stand in the path. Since causality from $\{E, F\}$ and G is direct, G satisfies both requirements *i*) and *ii*) of Definition 1, making $\beth = \{G\}$ and $\aleph = \{E, F\}$. Given that every element of X_u is an ancestor of $\aleph$, we can conclude that $\daleth = \{B, C, D\}$.

In graph *ii*), the original sources of long-term fluctuations are A and B. The observed and latent variables are $X_o = \{E, F, G\}$ and $X_u = \{C, D\}$, respectively. The forward paths between T and G are blocked by F, and causality from F to G does not pass through unobserved components. Consequently, $G \in \beth$ as per Definition 1. Conversely, there exist paths between T and $\{E, F\}$ not blocked by X_o, thus $\aleph = \{E, F\}$. Given that every element of X_u is an ancestor of $\aleph$, we can conclude that $\daleth = \{C, D\}$.

In graphs *iii*) and *iv*), the original source of long-term fluctuations is A, and in both cases, D blocks the paths between A and $\{E, G\}$. E is directly and exclusively affected by D, therefore it belongs to $\beth$. The nature of G changes between graphs *iii*) and *iv*). In both cases, causality between D and G passes through F, but only in *iii*) does F not, in turn, affect E. Consequently, $G \in \beth$ in graph *iii*), while $G \in \aleph$ in graph *iv*). In graph *iv*), F is an ancestor of D, so it belongs to $\daleth$. Conversely, in graph *iii*), F is a parent of G but not of D, thus it is a component of $\gimel$. The causal sets are thus $T = \{A\}$, $\daleth = \{B, C\}$, $\aleph = \{D\}$, $\gimel = \{F\}$, and $\beth = \{E, G\}$ in *iii*); and $T = \{A\}$, $\daleth = \{B, C, F\}$, $\aleph = \{D, G\}$,

$\mathcal{L} = \emptyset$, and $\mathcal{Z} = \{E\}$ in *iv*).

By examining the definitions, we can draw conclusions regarding the causal relations among the sets. From Definitions 3 and 4, it is evident that any causal relation of the type $\mathcal{L} \to \mathcal{K}$ is excluded. According to Definition 3, the components of $\mathcal{K}$ are ancestors of $\mathcal{X}$. It follows that if $\mathcal{L}_i$ causes $\mathcal{K}_j$, $\mathcal{L}_i$ must necessarily be in turn an ancestor of $\mathcal{X}$. Consequently, for definition 3 $\mathcal{L}_i$ belongs to $\mathcal{K}$, contradicting the initial assumption $\mathcal{L}_i \in \mathcal{L}$, given that Definition 4 excludes mutual belonging between $\mathcal{L}$ and $\mathcal{K}$.

Similarly, we can exclude the causal relations of the type $\mathcal{L} \to \mathcal{X}$. If $\mathcal{L}_i$ causes $\mathcal{X}_j$, it follows that the former is an ancestor of the latter, thus a component of $\mathcal{K}$.

By examining Definitions 1 and 3, we can exclude the causal connection $\mathcal{K} \to \mathcal{Z}$. Consider $\mathcal{K}_i$ as an ancestor of $\mathcal{X}$ whose forward paths with T are not blocked by X_o. If $\mathcal{K}_i$ directly causes $\mathcal{Z}_j$, the forward paths between the latter and the original source of causality can only be blocked by subsets of $\mathcal{X}$ if $\mathcal{X}$ blocks the forward paths between T and $\mathcal{K}_i$, contradicting the initial hypothesis. If instead the forward paths between T and $\mathcal{K}_i$ are blocked by X_o, it follows from requirement *ii*) of Definition 1 that if $\mathcal{K}_i \to \mathcal{Z}_j$, it cannot be that $\mathcal{K}_i \to \mathcal{X}_k$, both in direct and indirect way. Nevertheless, this contradicts the Definition 3, since $\mathcal{K}$ contains the ancestors of $\mathcal{X}$.

We can also exclude direct causality between $\mathcal{K}$ and $\mathcal{L}$. Consider $\mathcal{K}_i$ as an ancestor of $\mathcal{X}$ whose paths with T are not blocked by X_o. If $\mathcal{K}_i$ directly causes $\mathcal{L}_j$, the forward paths between the latter and $\mathcal{Z}$ can be blocked if $\mathcal{X}$ stands between the causal path from $\mathcal{L}$ to $\mathcal{Z}$. This proposition contradicts the previous findings since it implies $\mathcal{L} \to \mathcal{X}$. If the

		T	ך	א	ל	ב
		↑	↑	↑	↑	↑
T	→	*	*	*	0	0
ך	→	0	*	*	0	0
א	→	0	*	*	*	*
ל	→	0	0	0	*	*
ב	→	0	*	*	*	*

Table 1.1: Causal Relations Among Sets. The right arrow → should be read as "it causes" while the Up arrow ↑ should be read as "it is caused by"

paths between T and ך_i are blocked by X_o, it follows from $\text{ך}_i \to \text{ל}$ that ך_i is among the ancestors of ב whose paths with T are blocked by א. Nevertheless, this is in contrast with requirement *ii*) of Definition 1.

Following requirement *i*) in Definition 1, we can immediately exclude any direct causal relation between T and ב. Moreover, if $T \to \text{ל}$, the forward paths between ל and ב can be blocked if א stands between their causal path, contradicting $\text{א} \not\to \text{ל}$.

The causal relationships among the sets are summarised in Table 1.1

The partition of X_o into א and ב lays the foundations for a precise definition of causal endogeneity:

Definition 5 (***Causal Endogeneity***)**.** *A variable is causally endogenous if it is in the set* ב*.*

Definition 5 suggests that variables in א should be labeled as causally exogenous. However, ambiguities arise in situations characterized by cyclic behaviour, as this definition does not account for potential feedback effects. This issue can perhaps best be explained with

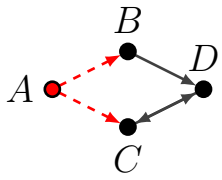

Figure 1.4: Causal Graph with Cyclic Behaviour Affecting Exogeneity

an example.

Consider the causal graph in Figure 1.4, where the red node represents the latent variable, namely $Y_u = \{A\}$. In this scenario, the only unobserved element is the fundamental trend A, while all the ordinary variables are observed. Specifically, we have: $Y = \{A, B, C, D\}$, $T = \{A\}$ and $X_o = \{B, C, D\}$.

By following the principles outlined in Definitions 1 and 2, we can construct the sets $\aleph$ and $\beth$ as follows: $\aleph = \{B, C\}$ and $\beth = \{D\}$ since the directed paths between A and D are blocked by $\{B, C\} \in X_o$ while no observed variable blocks the directed paths between A and the two components of $\aleph$. Nevertheless, classifying the elements of $\aleph$ as exogenous variables may create some perplexities. On one hand, B aligns with the heuristic notions of exogeneity and does not pose any challenges to such categorization. On the other hand, the exogenous nature of C is questioned by the feedback that D has on C.

These considerations underscore the necessity for a twofold definition:

Definition 6 (***Weak Causal Exogeneity***)**.** *A variable is weakly causally exogenous if it is in the set* $\aleph$*.*

Definition 7 (***Strong Causal Exogeneity***)**.** *A variable* $x_i \in X_o$ *is strongly causally*

exogenous if it is part of the set $\aleph$ *and none of its ancestors are included in* $\beth$.

Based on these definitions, both B and C are weakly causally exogenous, while only B satisfies strong causal exogeneity.

Both definitions are logically coherent, and *a priori* neither holds precedence over the other. They serve distinct purposes, and address different inquiries.

For instance, within the realm of political economy, researchers may seek to identify variables that remain unaffected by other macroeconomic factors, in order to find proper instruments whose nominal values can be freely manipulated. In this context, researchers are primarily interested in identifying strongly exogenous variables.

Conversely, there are scenarios in which weak causal exogeneity may be more pertinent. Consider situations where certain policy makers respond to specific policy functions. This dynamic can give rise to cyclic causal interactions between policy instruments and target variables. Nonetheless, the policy function can be intentionally altered, and the research objective may be confined to defining as exogenous those variables that can be manipulated within a counterfactual scenario.

For instance, consider the case where C and D in graph 1.4 are substituted by the interest rate i and the inflation rate π respectively. Suppose that when π fluctuates, policy makers respond by intervening on i. Such behaviour generates a cyclic causal pattern that is artificially induced by the policy function of monetary authorities. However, if the analysis centers on the behaviour that policy makers should adopt, this causal relationship loses its

significance since the policy function itself becomes the focal point of examination, and the analysis should focus on whether modifying the policy function is beneficial. Naturally, by modifying the policy function, we do not mean altering a parameter in the reaction function, but rather considering changes that affect the causal graph. Specifically, this involves either stopping the policy rule or changing it by focusing on a different variable that might be more effective for the policy purposes.

In terms of governability, the distinction between weak and strong exogeneity hinges on the type of intervention necessary on א to control the nominal value of ב. Consider the graphs in Figures 1.1 and 1.4. In both cases, the variable D can be expressed as $D = \beta_1 B + \beta_2 C$. However, in the second instance, C is, in turn, a function of D. This causal feedback is not problematic as long as there is a strong margin of control over E and F, meaning that it is possible to intervene on these two variables to keep them fixed at specific values E_0 and F_0. Conversely, if the control over א is weaker, meaning that it is only possible to induce a transitory shock on א, the effect on ב is unstable and can be nullified by the causal feedback from ב to א.

1.3.1 Causal Exogeneity and Weak Exogeneity

To underscore the distinction between the two causal ideas of exogeneity and the statistical concept of standard weak exogeneity, we assess the latter within the context of the model delineated by the causal graph in Figure 1.4. As explained in the previous section, the

graph can be translated into the VECM:

$$\Delta \begin{bmatrix} A \\ B \\ C \\ D \end{bmatrix}_t = \begin{bmatrix} 0 & 0 & 0 \\ a & 0 & 0 \\ 0 & b & 0 \\ 0 & 0 & c \end{bmatrix} \begin{bmatrix} d & 1 & 0 & 0 \\ e & 0 & 1 & f \\ 0 & g & h & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \\ D \end{bmatrix}_{t-1} + \varepsilon_t$$

The evaluation of weak exogeneity entails an examination of the constraints imposed on the adjustment matrix α_o. Since in our illustration the only unobserved component is the fundamental trend, equation (1.7) can be reduced to[8]:

$$\alpha_o = \Omega_o N_{o\perp} \tag{1.10}$$

Without loss of generality we set $\Omega_o = I$, where I indicates the identity matrix, so that $\alpha_o = N_{o\perp}$.

The matrix N_o establishes the relationship between X_o and T within the state-space representation of the fully observed model. Consequently, it is a 3×1 matrix with non-zero entries in the rows corresponding to variables B and C, specifically:

$$N_o = \begin{bmatrix} ad \\ be \\ 0 \end{bmatrix}$$

[8]See Johansen (2019) for proof

The orthogonal complement $N_{o\perp}$, is a 3×2 matrix. An admissible $N_{o\perp}$ matrix is:

$$\alpha_o = N_{o\perp} = \begin{bmatrix} ad \\ be \\ 0 \end{bmatrix}_{\perp} = \begin{bmatrix} -be & 0 \\ ad & 0 \\ 0 & 1 \end{bmatrix}$$

This shows that neither B nor C satisfy weak exogeneity: the corresponding rows of α_o do not contain only zeros.

The example proves that the weakest statistical concept of exogeneity may impose stricter requirements than the strongest causal definition of exogeneity. This serves to emphasize the distinction between causal exogeneity and the other exogeneity concepts outlined in Engle et al. (1983). Specifically, since weak exogeneity is necessary for both strong and super exogeneity, in the example above, neither B nor C can be considered strong or super exogenous. This contrast highlights the fundamental difference between exogeneity as applied in statistical contexts and its role in causal analysis.

In the particular instance involving the causal structure illustrated in Figure 1.4, the resulting exogeneity patterns are summarised below:

- Weakly Exogenous variables within the set X_o: $\emptyset$
- Strongly causally exogenous variables within the set X_o: $\{B\}$
- Weakly causally exogenous variables within the set X_o: $\{B, C\}$
- Causally endogenous variable within the set X_o: $\{D\}$

1.4 Weak Causal Exogeneity

This section discusses a method for identifying weakly causally exogenous variables. The first subsection explores the correspondence between properties of the partially observed model and weak causal exogeneity. We establish the relationship that holds between the Vector Error Correction Model estimable by the researcher, and the attributes of the underlying latent causal graph. Subsequently, subsection 1.4.2 leverages the findings from subsection 1.4.1 to develop an inferential approach for testing hypotheses related to weak causal exogeneity. Finally, subsection 1.4.3 examines the connection between weak causal exogeneity and its statistical counterpart, i.e. weak exogeneity.

1.4.1 Model Manifestations of Weak Causal Exogeneity

The analysis investigates the impact of weak causal exogeneity on the coefficients of a partially observed VECM. Specifically, the analysis focuses on the properties of a transformation of the adjustment matrix α_o.

The investigation starts with an examination of the properties of the orthogonal complement $(M_{ou}V_{uT} + N_oV_T)_\perp$. This analysis is conducted based on the definitions outlined in section 1.3 and their implications for the causal relationships among the sets א, ב, ד, ל and T. We first prove some intermediate results, while the main result is presented in Theorem 1.

The coefficient M_{ou} in the state-space representation (Equation (1.6)) connects $X_o =$

$\{\aleph, \beth\}$ to $X_u = \{\daleth, \lamed\}$. Given that elements in $\beth$ cannot be influenced by components of $\daleth$, as well as the elements in $\aleph$ cannot be influenced by components of $\lamed$, the sub-matrix of M_{ou} formed by the rows corresponding to $\beth$ and the columns corresponding to $\daleth$ and the sub-matrix of M_{ou} formed by the rows corresponding to $\aleph$ and the columns corresponding to $\lamed$ comprises only null entries. Similarly, the parameter N_o links $X_o = \{\aleph, \beth\}$ to T. Since, by definition, $\beth$ cannot be directly affected by T, the sub-matrix of N_o comprised of rows corresponding to $\beth$ will also consist solely of null entries. Thus, the representations of M_{ou} and N_o are as follows:

$$M_{ou} = \begin{array}{c} \\ |\aleph| \\ |\beth| \end{array} \begin{array}{c} \begin{array}{cc} |\daleth| & |\lamed| \end{array} \\ \begin{bmatrix} \boldsymbol{\mathcal{A}} & \mathbf{0} \\ \mathbf{0} & \boldsymbol{\mathcal{C}} \end{bmatrix} \end{array} \qquad N_o = \begin{array}{c} \\ |\aleph| \\ |\beth| \end{array} \begin{array}{c} \begin{array}{c} |T| \end{array} \\ \begin{bmatrix} \boldsymbol{\mathcal{D}} \\ \mathbf{0} \end{bmatrix} \end{array}$$

where the symbol $|A|$ indicates the cardinality of the set A and $\mathbf{0}$ denotes a null matrix.

To comprehend the properties of the conditional covariance matrix V we need to find the solutions of the Riccati problem in Equation (1.9). The following Lemma provides an analysis of V which will be crucial for deriving subsequent conclusions:

Lemma 1 (***Conditional Linear Independence Between*** $T \cup \daleth$ ***and*** $\lamed$)**.** *The covariance between the components of* $\lamed$ *and the variables in* $T \cup \daleth$ *is zero when conditioned on the elements in* X_o*.*

Proof of Lemma 1*.* To prove this lemma, we must show that the sub-matrix of $\boldsymbol{V}$ relating $\lamed$ to $T \cup \daleth$ contains null entries. Specifically, we should demonstrate that the

following structure of $\boldsymbol{V}$ is an admissible solution:

$$\boldsymbol{V} = \begin{array}{c} \\ |ד \cup T| \\ |ל| \end{array} \begin{array}{c} \begin{array}{cc} |ד \cup T| & |ל| \end{array} \\ \begin{bmatrix} \boldsymbol{V_1} & \boldsymbol{0} \\ \boldsymbol{0} & \boldsymbol{V_2} \end{bmatrix} \end{array}$$

We begin by analyzing the covariance of $T \cup ד$ conditioned on the elements of $X_o \cup ל$. This involves treating $ל$ as observed and considering only $T \cup ד$ as latent. Under this scenario, the coefficients of Equation (1.9) transform as follows:

$$Q_1^* = \begin{array}{c} \\ |ד \cup T| \end{array} \begin{array}{c} \begin{array}{c} |ד \cup T| \end{array} \\ \begin{bmatrix} \boldsymbol{\mathcal{A}} \end{bmatrix} \end{array} \qquad \Omega_1^* = \begin{array}{c} \\ |ד \cup T| \end{array} \begin{array}{c} \begin{array}{c} |ד \cup T| \end{array} \\ \begin{bmatrix} \boldsymbol{\mathcal{E}} \end{bmatrix} \end{array}$$

$$C_1^* = \begin{array}{c} \\ |א| \\ |ב| \\ |ל| \end{array} \begin{array}{c} \begin{array}{c} |ד \cup T| \end{array} \\ \begin{bmatrix} \boldsymbol{\mathcal{C}} \\ \boldsymbol{0} \\ \boldsymbol{0} \end{bmatrix} \end{array} \qquad \Omega_{o1} = \begin{array}{c} \\ |א| \\ |ב| \\ |ל| \end{array} \begin{array}{c} \begin{array}{ccc} |א| & |ב| & |ל| \end{array} \\ \begin{bmatrix} \boldsymbol{\mathcal{G}} & \boldsymbol{0} & \boldsymbol{0} \\ \boldsymbol{0} & \boldsymbol{\mathcal{H}} & \boldsymbol{0} \\ \boldsymbol{0} & \boldsymbol{0} & \boldsymbol{\mathcal{I}} \end{bmatrix} \end{array}$$

where the subscript 1 is used to distinguish these coefficients from those for $\boldsymbol{V} = \mathrm{Cov}(Y_u|X_o)$.

According to Equation (1.9), the covariance matrix $\boldsymbol{W_1} = \mathrm{Cov}(T \cup ד|X_o \cup ל)$ satisfies the following Riccati equation:

$$\boldsymbol{W_1} = Q_1^* \boldsymbol{W_1} Q_1^{*'} + \Omega_1^* - Q_1^* \boldsymbol{W_1} C_1^{*'} (C_1^* \boldsymbol{W_1} C_1^{*'} + \Omega_{o1})^{-1} C_1^* \boldsymbol{W_1} Q_1^{*'} \tag{1.11}$$

Given that $(C_1^* \boldsymbol{W_1} C_1^{*\prime} + \Omega_{o1})^{-1}$ is

$$\left(\begin{bmatrix} \boldsymbol{\mathcal{C}} \\ 0 \\ 0 \end{bmatrix} \boldsymbol{W_1} \begin{bmatrix} \boldsymbol{\mathcal{C}}' & 0 & 0 \end{bmatrix} + \begin{bmatrix} \boldsymbol{\mathcal{G}} & 0 & 0 \\ 0 & \boldsymbol{\mathcal{H}} & 0 \\ 0 & 0 & \boldsymbol{\mathcal{I}} \end{bmatrix} \right)^{-1} = \begin{bmatrix} (\boldsymbol{\mathcal{C}} \boldsymbol{W_1} \boldsymbol{\mathcal{C}}' + \boldsymbol{\mathcal{G}})^{-1} & 0 & 0 \\ 0 & \boldsymbol{\mathcal{H}}^{-1} & 0 \\ 0 & 0 & \boldsymbol{\mathcal{I}}^{-1} \end{bmatrix}$$

Equation (1.11) can be simplified as follows:

$$\boldsymbol{W_1} = \boldsymbol{\mathcal{A}} \boldsymbol{W_1} \boldsymbol{\mathcal{A}}' + \boldsymbol{\mathcal{E}} - \boldsymbol{\mathcal{A}} \boldsymbol{W_1} \boldsymbol{\mathcal{C}}' (\boldsymbol{\mathcal{C}} \boldsymbol{W_1} \boldsymbol{\mathcal{C}}' + \boldsymbol{\mathcal{G}})^{-1} \boldsymbol{\mathcal{C}} \boldsymbol{W_1} \boldsymbol{\mathcal{A}}'$$

Similarly, we can retrieve the covariance matrix of ל conditioned on $X_o \cup T \cup$ ך. This implies treating $X_o \cup T \cup$ ך as observed, with ל as the only set containing latent components.[9] Under this scenario, the coefficients of the Riccati equation become:

$$Q_2^* = \begin{matrix} \\ |\text{ל}| \end{matrix} \begin{matrix} |\text{ל}| \\ \end{matrix} \begin{bmatrix} \boldsymbol{\mathcal{B}} \end{bmatrix} \qquad \Omega_2^* = \begin{matrix} \\ |\text{ל}| \end{matrix} \begin{matrix} |\text{ל}| \\ \end{matrix} \begin{bmatrix} \boldsymbol{\mathcal{F}} \end{bmatrix}$$

$$C_2^* = \begin{matrix} \\ |\text{א}| \\ |\text{ב}| \\ |\text{ך} \cup T| \end{matrix} \begin{matrix} |\text{ל}| \\ \end{matrix} \begin{bmatrix} \boldsymbol{0} \\ \boldsymbol{\mathcal{D}} \\ \boldsymbol{0} \end{bmatrix} \qquad \Omega_{o2} = \begin{matrix} \\ |\text{א}| \\ |\text{ב}| \\ |\text{ך} \cup T| \end{matrix} \begin{matrix} |\text{א}| & |\text{ב}| & |\text{ך} \cup T| \\ \end{matrix} \begin{bmatrix} \boldsymbol{\mathcal{G}} & \boldsymbol{0} & \boldsymbol{0} \\ \boldsymbol{0} & \boldsymbol{\mathcal{H}} & \boldsymbol{0} \\ \boldsymbol{0} & \boldsymbol{0} & \boldsymbol{\mathcal{J}} \end{bmatrix}$$

Note that the subscript 2 denotes the coefficients in this context, differentiating them from the ones employed for $\boldsymbol{V} = \text{Cov}(Y_u | X_o)$ and $\boldsymbol{W_1} = \text{Cov}(T \cup \text{ך} | X_o \cup \text{ל})$.

[9] It should be noted that, despite conditioning on all other causal sets, the variables in ל retain their stochastic behaviour. This follows from the fact that every variable has its own stochastic error term.

According to (1.9), the covariance matrix $\boldsymbol{W_2} = \text{Cov}(\text{ל}|X_o \cup T \cup \text{ד})$ satisfies the Riccati equation given by

$$\boldsymbol{W_2} = Q_2^* \boldsymbol{W_2} Q_2^{*'} + \Omega_2^* - Q_2^* \boldsymbol{W_2} C_2^{*'} (C_2^* \boldsymbol{W_2} C_2^{*'} + \Omega_{o2})^{-1} C_2^* \boldsymbol{W_2} Q_2^{*'} \tag{1.12}$$

Given that $(C_2^* \boldsymbol{W_2} C_2^{*'} + \Omega_{o2})^{-1}$ is

$$\left(\begin{bmatrix} 0 \\ \boldsymbol{\mathcal{D}} \\ 0 \end{bmatrix} \boldsymbol{W_2} \begin{bmatrix} 0 & \boldsymbol{\mathcal{D}}' & 0 \end{bmatrix} + \begin{bmatrix} \boldsymbol{\mathcal{G}} & 0 & 0 \\ 0 & \boldsymbol{\mathcal{H}} & 0 \\ 0 & 0 & \boldsymbol{\mathcal{J}} \end{bmatrix} \right)^{-1} = \begin{bmatrix} \boldsymbol{\mathcal{G}} & 0 & 0 \\ 0 & (\boldsymbol{\mathcal{D}} \boldsymbol{W_2} \boldsymbol{\mathcal{D}}' + \boldsymbol{\mathcal{H}})^{-1} & 0 \\ 0 & 0 & \boldsymbol{\mathcal{I}}^{-1} \end{bmatrix}$$

Equation (1.12) can be simplified as follows:

$$\boldsymbol{W_2} = \boldsymbol{\mathcal{B}} \boldsymbol{W_2} \boldsymbol{\mathcal{B}}' + \boldsymbol{\mathcal{F}} - \boldsymbol{\mathcal{B}} \boldsymbol{W_2} \boldsymbol{\mathcal{D}}' (\boldsymbol{\mathcal{D}} \boldsymbol{W_2} \boldsymbol{\mathcal{D}}' + \boldsymbol{\mathcal{H}})^{-1} \boldsymbol{\mathcal{D}} \boldsymbol{W_2} \boldsymbol{\mathcal{B}}'$$

We now analyse the covariance matrix $\boldsymbol{V} = \text{Cov}(Y_u|X_o)$, which represents the covariance of the unobserved variables conditioned on the observed components. The coefficients to be inserted into the Riccati equation (1.9) are:

$$Q^* = \begin{array}{c} \\ |\text{ד} \cup T| \\ |\text{ל}| \end{array} \begin{array}{c} \begin{array}{cc} |\text{ד} \cup T| & |\text{ל}| \end{array} \\ \begin{bmatrix} \boldsymbol{\mathcal{A}} & \mathbf{0} \\ \mathbf{0} & \boldsymbol{\mathcal{B}} \end{bmatrix} \end{array} \qquad C^* = \begin{array}{c} \\ |\text{א}| \\ |\text{ב}| \end{array} \begin{array}{c} \begin{array}{cc} |\text{ד} \cup T| & |\text{ל}| \end{array} \\ \begin{bmatrix} \boldsymbol{\mathcal{C}} & \mathbf{0} \\ \mathbf{0} & \boldsymbol{\mathcal{D}} \end{bmatrix} \end{array}$$

$$\Omega^* = \begin{array}{c} \\ |ד \cup T| \\ |ל| \end{array} \begin{array}{c} \begin{array}{cc} |ד \cup T| & |ל| \end{array} \\ \begin{bmatrix} \boldsymbol{\mathcal{E}} & \mathbf{0} \\ \mathbf{0} & \boldsymbol{\mathcal{F}} \end{bmatrix} \end{array} \qquad \Omega_o = \begin{array}{c} \\ |א| \\ |ב| \end{array} \begin{array}{c} \begin{array}{cc} |א| & |ב| \end{array} \\ \begin{bmatrix} \boldsymbol{\mathcal{G}} & \mathbf{0} \\ \mathbf{0} & \boldsymbol{\mathcal{H}} \end{bmatrix} \end{array}$$

The final representation for $\boldsymbol{V}$ is:

$$\boldsymbol{V} = Q^* \boldsymbol{V} Q^{*'} + \Omega^* - Q^* \boldsymbol{V} C^{*'} (C^* \boldsymbol{V} C^{*'} + \Omega_o)^{-1} C^* \boldsymbol{V} Q^{*'} \tag{1.13}$$

To prove the lemma, we demonstrate that selecting $\boldsymbol{V}$ as follows

$$\boldsymbol{V} = \begin{array}{c} \\ |ד \cup T| \\ |ל| \end{array} \begin{array}{c} \begin{array}{cc} |ד \cup T| & |ל| \end{array} \\ \begin{bmatrix} \boldsymbol{V_1} & \mathbf{0} \\ \mathbf{0} & \boldsymbol{V_2} \end{bmatrix} \end{array}$$

the equation finds an admissible solution. In this case, Equation 1.13 becomes:

$$\begin{bmatrix} \boldsymbol{V_1} & 0 \\ 0 & \boldsymbol{V_2} \end{bmatrix} = \begin{bmatrix} \boldsymbol{\mathcal{A} V_1 \mathcal{A}'} & 0 \\ 0 & \boldsymbol{\mathcal{B} V_2 \mathcal{B}'} \end{bmatrix} + \begin{bmatrix} \boldsymbol{\mathcal{E}} & 0 \\ 0 & \boldsymbol{\mathcal{F}} \end{bmatrix} - \begin{bmatrix} \boldsymbol{\mathcal{A} V_1 \mathcal{C}'} & 0 \\ 0 & \boldsymbol{\mathcal{B} V_2 \mathcal{D}'} \end{bmatrix} \times$$

$$\times \begin{bmatrix} \boldsymbol{\mathcal{C} V_1 \mathcal{C}' + \mathcal{G}} & 0 \\ 0 & \boldsymbol{\mathcal{D} V_2 \mathcal{D}' + \mathcal{H}} \end{bmatrix}^{-1} \begin{bmatrix} \boldsymbol{\mathcal{C} V_1 \mathcal{A}'} & 0 \\ 0 & \boldsymbol{\mathcal{D} V_2 \mathcal{B}'} \end{bmatrix}$$

which can be clearly solved by setting $\boldsymbol{V_1} = \boldsymbol{W_1}$ and $\boldsymbol{V_2} = \boldsymbol{W_2}$. The admissibility of the solution

$$\boldsymbol{V} = \begin{bmatrix} \boldsymbol{W_1} & 0 \\ 0 & \boldsymbol{W_2} \end{bmatrix}$$

follows from the positive semi-definiteness of covariance matrices $\boldsymbol{W_1}$ and $\boldsymbol{W_2}$. This means that for any non-zero vectors $x \in \mathbb{R}^{|T \cup \text{ך}|}$ and $y \in \mathbb{R}$ to the power of ל, it holds $x' \boldsymbol{W_1} x \geq 0$ and $y' \boldsymbol{W_2} y \geq 0$. Consequently,

$$\begin{bmatrix} x' & y' \end{bmatrix} \begin{bmatrix} \boldsymbol{W_1} & 0 \\ 0 & \boldsymbol{W_2} \end{bmatrix} \begin{bmatrix} x \\ y \end{bmatrix} \geq 0$$

Since x imposes no constraints on the entries of y, the vector $w = \begin{bmatrix} x & y \end{bmatrix}$ represents any non-zero vector in $\mathbb{R}^{|Y_u|}$.

This proves the Lemma

□

The conclusions of Lemma 1 can be intuitively understood through the analysis of blocked and unblocked covariance paths. The paths between ך and T are either not blocked by X_o or unblocked by א. The latter scenario occurs because ך includes the ancestors of א, which are in turn directly or indirectly caused by T. This situation is analogous to the paths among the parents of a collider being unblocked when conditioning on the child. It follows that the entries of V_{uT} corresponding to the elements in ך are non-zero vectors. Conversely, X_o blocks the forward paths between T and ל, and ל does not exert direct or indirect influence over א. Therefore, the entries of V_{uT} corresponding to elements in ל are zeros.

The resulting structure of the matrix $M_{ou}V_{uT} + N_oV_T$ is:

$$(M_{ou}V_{uT} + N_oV_T)_\perp = \left(\begin{array}{cc} & \begin{array}{cc} |ר| & |ל| \end{array} \\ \begin{array}{c} |א| \\ |ב| \end{array} & \begin{bmatrix} \boldsymbol{\mathcal{A}} & \mathbf{0} \\ \mathbf{0} & \boldsymbol{\mathcal{C}} \end{bmatrix} \end{array} \begin{array}{c} |T| \\ \begin{array}{c} |ר| \\ |ל| \end{array} \begin{bmatrix} \boldsymbol{\mathcal{E}} \\ \mathbf{0} \end{bmatrix} \end{array} + \begin{array}{c} |T| \\ \begin{array}{c} |א| \\ |ב| \end{array} \begin{bmatrix} \boldsymbol{\mathcal{D}} \\ \mathbf{0} \end{bmatrix} \end{array} \begin{array}{c} |T| \\ |T| \begin{bmatrix} \boldsymbol{\mathcal{F}} \end{bmatrix} \end{array} \right)_\perp =$$

$$= \begin{array}{c} \\ \begin{array}{c} |א| \\ |ב| \end{array} \end{array} \begin{array}{c} \begin{array}{cc} r - |ב| & |ב| \end{array} \\ \begin{bmatrix} \left(\boldsymbol{\mathcal{A}}\boldsymbol{\mathcal{E}} + \boldsymbol{\mathcal{D}}\boldsymbol{\mathcal{F}}\right)_\perp & \mathbf{0} \\ \mathbf{0} & \boldsymbol{I} \end{bmatrix} \end{array} \tag{1.14}$$

In this expression, r denotes the rank of $M_{ou}V_{uT} + N_oV_T$, and I represents the identity matrix.

The following lemma derives conclusions concerning specific properties of the orthogonal complement, laying the foundation for subsequent deductions:

Lemma 2 (***Linear Dependence of the Rows of an Orthogonal Complement***)**.** *Consider a rectangular matrix A of dimensions $m \times n$ and rank r, where $m > n$, and let B be an $m \times (m - r)$ matrix of full rank, such that $A'B = 0$. If every row of A contains at least one non-zero entry, then all the rows of B are linearly dependent.*

Proof of Lemma 2. Without loss of generality, we establish the linear dependency of the first row of B. In other words, we aim to demonstrate that $B_{(1)}$ can be expressed as a linear combination of the other rows:

$$B_{(1)} = \lambda_2 B_{(2)} + \lambda_3 B_{(3)} + \cdots + \lambda_m B_{(m)} \tag{1.15}$$

where $\lambda_k \neq 0$ for at least one k between 2 and m

By construction, $A'B$ is equal to 0. Consequently, the product of every row of A' with the matrix B must result in a zero vector. Let $A'_{(j)}$ represent the j^{th} row of A'. We have:

$$A'_{(j)}B = \begin{bmatrix} A_{j1} & A_{j2} & \cdots & A_{jm} \end{bmatrix} \begin{bmatrix} B_{11} & B_{12} & \cdots & B_{1(m-r)} \\ B_{21} & B_{22} & \cdots & B_{2(m-r)} \\ \vdots & \vdots & \ddots & \vdots \\ B_{m1} & B_{m2} & \cdots & B_{m(m-r)} \end{bmatrix} = 0$$

By assumption, every row of A contains at least one non-null entry. Therefore, there exists a value of j such that $A_{j1} \neq 0$. In fact, if $A_{j1} = 0$ for all $j = 1 : n$, the j^{th} row of A is a zero vector.

If $A_{j1} \neq 0$ it must hold that either $A_{jk\neq 1} \neq 0$, or $B_{1i} = 0$ for all i. In fact, we know that

$$\sum_{i=1}^{m} A_{ji}B_{ik} = 0 \tag{1.16}$$

must hold $\forall$ admissible k. If $A_{ji} \neq 0$ only for $i = 1$, equation (1.16) reduces to

$$A_{j1}B_{1k} = 0$$

Since $A_{j1} \neq 0$, it follows that $B_{1k} = 0$ for all k, indicating that the first row of B is the zero vector, and thus linearly dependent.

Conversely, if both $A_{j1} \neq 0$ and $A_{jq} \neq 0$ for some $q \neq 1$ it must hold:

$$\begin{bmatrix} 1 & \lambda_2 & \cdots & \lambda_m \end{bmatrix} \begin{bmatrix} B_{11} & B_{12} & \cdots & B_{1(m-n)} \\ B_{21} & B_{22} & \cdots & B_{2(m-n)} \\ \vdots & \vdots & \ddots & \vdots \\ B_{m1} & B_{m2} & \cdots & B_{m(m-n)} \end{bmatrix} = 0$$

where the vector $\boldsymbol{\lambda}$ is obtained after a proper normalization of $A'_{(j)}$ and contains at least two non-zero entries.

This verifies equation (1.15) and establishes the validity of the lemma. □

The result of Lemma 2 can be extended to appraise the characteristics of the partially observed model, setting the stage for the more comprehensive proposition articulated in the following theorem:

Theorem 1 (***Weak Causal Exogeneity and Linear Dependence of the Rows of*** Ψ)**.** *The rows of the matrix* Ψ*, corresponding to elements within the set* $\aleph$*, are linearly dependent. Here,* $\Psi = \Sigma_o^{-1}\Pi_o$*, and* Π_o *is the impact matrix of the partially observed model, defined as* $\Pi_o = \alpha_o\beta'_o$*.*

Proof of Theorem 1. From equation (1.7), we know that the adjustment matrix is equal to $\alpha_o = \Sigma_o(M_{ou}V_{uT}+N_oV_T)_\perp$. Substituting, we have $\Psi = \Sigma_o^{-1}\alpha_o\beta'_o = \Sigma_o^{-1}\Sigma_o(M_{ou}V_{uT}+N_oV_T)_\perp\beta'_o$, which in matrix form is

$$\Psi = \begin{bmatrix} \left(\boldsymbol{\mathcal{A}}\boldsymbol{\mathcal{E}} + \boldsymbol{\mathcal{D}}\boldsymbol{\mathcal{F}}\right)_{\perp} & \mathbf{0} \\ \mathbf{0} & \boldsymbol{I} \end{bmatrix} \beta_o' \tag{1.17}$$

It follows from Lemma 2 that there exists a vector $\boldsymbol{\lambda}$ containing only non-zero elements such that $\boldsymbol{\lambda}\left(\boldsymbol{\mathcal{A}}\boldsymbol{\mathcal{E}} + \boldsymbol{\mathcal{D}}\boldsymbol{\mathcal{F}}\right)_{\perp} = 0$, indicating that the rows of $\Sigma_o^{-1}\alpha_o$ corresponding to the variables in $\aleph$ are linearly dependent.

To conclude the proof of the theorem, it is observed that the rows of Ψ corresponding to $\aleph$ (denoted as $\Psi_{(\aleph)}$) are given by:

$$\Psi_{(\aleph)} = \begin{bmatrix} \left(\boldsymbol{\mathcal{A}}\boldsymbol{\mathcal{E}} + \boldsymbol{\mathcal{D}}\boldsymbol{\mathcal{F}}\right)_{\perp} & \mathbf{0} \end{bmatrix} \beta_o'$$

It is evident that the vector $\boldsymbol{\lambda}$ possesses the property:

$$\boldsymbol{\lambda}\Psi_{(\aleph)} = \boldsymbol{\lambda} \begin{bmatrix} \left(\boldsymbol{\mathcal{A}}\boldsymbol{\mathcal{E}} + \boldsymbol{\mathcal{D}}\boldsymbol{\mathcal{F}}\right)_{\perp} & \mathbf{0} \end{bmatrix} \beta_o' = 0$$

This completes the proof of the theorem. □

From Theorem 1, we can readily deduce the following corollary:

Corollary 1 (***First Corollary to Theorem 1***)**.** *The rows of the matrix* Ψ *corresponding to the variables included in* $\beth$ *are linearly independent.*

Proof of Corollary 1*.* From equation (1.17), we can notice that the rows of Ψ corresponding to $\beth$ (denoted by $\Psi_{(\beth)}$) are equal to $\beta'_{o(\beth)}$, where $\beta'_{o(\beth)}$ represents the last $\beth$ rows

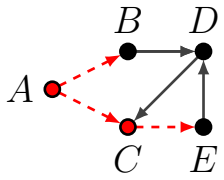

Figure 1.5: Example to Illustrate the Findings of Theorem 1 and its First Corollary

of β'_o.[10]

Considering that β'_o has a greater number of columns than rows, any linear dependence among the rows of $\beta'_{o(\beth)}$ would imply rank deficiency in β'_o. However, such a scenario contradicts the fundamental characteristic of the cointegration matrix, which is inherently constructed as a full-rank rectangular matrix. □

To clarify the concept of Theorem 1 and its Corollary, consider the graph in figure 1.5 and the corresponding VECM shown below:

$$\Delta \begin{bmatrix} A \\ B \\ C \\ D \\ E \end{bmatrix}_t = \begin{bmatrix} 0 & 0 & 0 & 0 \\ a & 0 & 0 & 0 \\ 0 & b & 0 & 0 \\ 0 & 0 & c & 0 \\ 0 & 0 & 0 & d \end{bmatrix} \begin{bmatrix} e & 1 & 0 & 0 & 0 \\ f & 0 & 1 & g & 0 \\ 0 & h & 0 & 1 & i \\ 0 & 0 & j & 0 & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \\ D \\ E \end{bmatrix}_{t-1} + \varepsilon_t$$

Suppose the model user observes only B, D, and E, so that $Y = \{A, B, C, D, E\}$, $X_o = \{B, D, E\}$, $T = \{A\}$, and $X_u = \{C\}$. As observed, the forward path between T and

[10] It is important to mention that $\beta'_{o(\beth)}$ refers to the last $\beth$ rows of β'_o within the order utilized thus far, and this order can be easily obtained through a trivial rearrangement of the variables

B is direct and thus not blocked by any other variable within X_o. On the other hand, the forward path between T and E is indirect as it passes through C. Since $C \in X_u$, no observed variable blocks the path between the fundamental trend and C. Therefore, both B and E belong to $\aleph$. Conversely, the forward paths between A and D are all blocked by X_o. Specifically, B blocks $A \to B \to D$ while E blocks $A \to C \to E \to D$. Additionally, causality from X_o to D is direct, thus satisfying condition ii) of Definition 1. C is a parent of $E \in \aleph$, thus it belongs to $\daleth$ according to Definition 3. In summary, the causal sets are $\aleph = \{B, E\}$, $\beth = \{D\}$, $\daleth = \{C\}$, and $T = \{A\}$. It is noteworthy that $D \in \beth$ indirectly causes $E \in \aleph$ through its effect on C. This renders E exclusively weakly causally exogenous, whereas only B is also strongly causally exogenous.

The coefficients of the state-space representation in Equation 1.6 can be derived from the fully observed VECM. Specifically, in this example, they are[11]:

$$M_o = \begin{bmatrix} a & 0 & 0 \\ 0 & d & 0 \\ ch & ci & c \end{bmatrix} \qquad M_{ou} = \begin{bmatrix} 0 \\ dj \\ 0 \end{bmatrix} \qquad N_o = \begin{bmatrix} ae \\ 0 \\ 0 \end{bmatrix}$$

$$M_u = \begin{bmatrix} b \end{bmatrix} \qquad M_{uo} = \begin{bmatrix} 0 & 0 & bg \end{bmatrix} \qquad N_u = \begin{bmatrix} bf \end{bmatrix}$$

[11]For illustrative purposes, the order chosen for the observed variables is $\{B, E, D\}$.

By substituting the above matrices into Equation 1.5, we can determine that the cointegration matrix of the partially observed system modeling B, D, and E only is a 3×2 matrix with non-zero elements in all of its entries:

$$\beta_o = \begin{bmatrix} \beta_{o1} & \beta_{o2} \\ \beta_{o3} & \beta_{o4} \\ \beta_{o5} & \beta_{o6} \end{bmatrix}$$

To derive the matrix $\gamma = (M_{ou}V_{uT} + N_oV_T)_\perp$, we need to understand the characteristics of the conditional covariance matrix V. Since A and C are directly connected, $\text{Cov}(A, C|X_o) \neq 0$. Similarly, the same holds for the conditional variance of A. Consequently,

$$\gamma = \left(\begin{bmatrix} 0 \\ djV_{uT} \\ 0 \end{bmatrix} + \begin{bmatrix} aeV_T \\ 0 \\ 0 \end{bmatrix} \right)_\perp = \begin{bmatrix} \gamma_1 & 0 \\ \gamma_2 & 0 \\ 0 & \gamma_3 \end{bmatrix}$$

The resulting Ψ matrix is therefore:

$$\Psi = \gamma\beta_o' = \begin{bmatrix} \gamma_1\beta_{o1} & \gamma_1\beta_{o3} & \gamma_1\beta_{o5} \\ \gamma_2\beta_{o1} & \gamma_2\beta_{o3} & \gamma_2\beta_{o5} \\ \gamma_3\beta_{o2} & \gamma_3\beta_{o4} & \gamma_3\beta_{o6} \end{bmatrix} \tag{1.18}$$

As observed, the first two rows are linearly dependent. Specifically, $\Psi_{(1)}$ can be expressed as $\delta\Psi_{(2)}$, where $\delta = \frac{\gamma_1}{\gamma_2}$. Similarly, $\Psi_{(2)}$ can be written as $\lambda\Psi_{(1)}$, where $\lambda = \frac{\gamma_2}{\gamma_1}$. This

observation aligns with Theorem 1, as in the chosen order, the variables corresponding to the first two rows are B and E, which are elements of $\aleph$.

Conversely, the third row of Ψ is linearly independent. This results from the fact that by construction $\begin{bmatrix} \beta_{o2} & \beta_{o4} & \beta_{o6} \end{bmatrix}$ is linearly independent with respect to $\begin{bmatrix} \beta_{o1} & \beta_{o3} & \beta_{o5} \end{bmatrix}$. To suggest otherwise would imply that β_o has rank 1, which contradicts the construction of the orthogonal complement of $C_o - M_{ou}M^{-1}uC_u$. This result corroborates Corollary 1, as $\Psi_{(3)}$ corresponds to $D \in \beth$.

1.4.2 Testing Hypotheses on Causal Endogeneity

Theorem 1 and its corollary proved a duality between the properties of matrix Ψ and weak causal exogeneity, and consequently, causal endogeneity. This result implies that hypotheses concerning causal exogeneity can be reformulated as hypotheses on the model's parameters.

To illustrate, consider the following null and alternative hypotheses:

$$\mathbf{H_{0c}}: \textit{The elements of } \Xi \textit{ are causally endogenous}$$

$$\mathbf{H_{1c}}: \textit{The set } \Xi \textit{ contains at least one weakly causally exogenous variable}$$

Where $\Xi \subseteq X_o$ represents a collection of arbitrarily selected variables, subject to investigation regarding their causal properties.

Based on the findings from the previous subsection, the causal hypotheses $\mathbf{H_{0c}}$ and $\mathbf{H_{1c}}$ can be restated in terms of constraints on the model as follows:

$\mathbf{H_{0m}}$ **:** *The rows in* Ψ *corresponding to the variables in* Ξ *are all linearly independent*

$\mathbf{H_{1m}}$ **:** *Some of the rows in* Ψ *corresponding to the variables in* Ξ *are linearly dependent*

Up to this point, we have translated the causal hypotheses in such a way as to distinguish ℵ and ⊐ by examining the linear dependencies within the rows of the matrix Ψ. The challenge is to understand how to exploit this feature for inferential purposes. The first difficulty is the estimation of Ψ. Consider the model adjusted for the short-term dependencies[12]:

$$R_{0,t} = \alpha_o \beta_o' R_{1,t} + \varepsilon_{o,t}$$

where $R_{0,t}$ and $R_{1,t}$ are the residuals from regressing (respectively) $\Delta X_{o,t}$ and $X_{o,t-1}$ on $\{\Delta X_{o,t-1}, \Delta X_{o,t-2}, \dots\}$. Upon pre-multiplying by Σ_o^{-1}, we derive[13]:

$$\Sigma_o^{-1} R_{0,t} = Q_t = \Sigma_o^{-1} \alpha_o \beta_o' R_{1,t} + \Sigma_o^{-1} \varepsilon_{o,t} = \Psi R_{1,t} + \eta_t \tag{1.19}$$

where $\text{Var}(\eta) = \Sigma_o^{-1} \Sigma_o \Sigma_o'^{-1} = \Sigma_o^{-1}$. Since multiplication by a full rank square matrix preserves rank, it follows that $\text{rank}(\alpha_o) = \text{rank}(\Sigma_o^{-1} \alpha_o)$. Consequently, we have $\text{rank}(\Psi) = \text{rank}(\alpha_o \beta_o')$. Thus, the model in Equation (1.19) constitutes a reduced rank regression, suitable for estimation using the technique introduced by Anderson (1951).

Notably, $\Sigma_o = \text{Var}(\varepsilon_o)$ is not directly observable, but it can be estimated as it represents the covariance matrix of the residuals in the partially observed VECM. Therefore, in practical applications, Q_t cannot be directly used. Instead, $\hat{Q}_t = \hat{\Sigma}_o^{-1} R_{0,t}$ is employed,

[12] Short-term dependencies can be wiped out by using the Frisch-Waugh theorem, see Johansen (1995) and Juselius (2006)

[13] Σ_o is a covariance matrix, thus it is square and has full rank. Consequently its inverse exists

where $\hat{\Sigma}_o$ is the estimate of Σ_o. This substitution can modify the distribution under the null as well as the critical values. However, $\hat{\Sigma}_o$ converges to Σ_o, such that $\hat{Q}_t$ converges to Q_t as the sample size increases. It follows that the critical values converge to the standard ones as $n \to \infty$.

Linear dependency can be detected by examining the influence that each row of Ψ exerts on the rank r of the regression in (1.19). In fact, the removal of a linearly independent row causes a reduction in the rank of Ψ. Conversely, the drop of a linearly dependent row leaves the rank unchanged.

This mechanism can be easily adapted to our objectives: if, by excluding the i^{th} row of the matrix Ψ (denoted by $\Psi_{(i)}$) the cointegration rank diminishes, we can infer that $\Psi_{(i)}$ is linearly independent, and consequently, the i^{th} variable in X_o belongs to the set $\beth$. Conversely, if the elimination of the i^{th} row of Ψ does not affect the regression rank, we can conclude that $\Psi_{(i)}$ is linearly dependent, indicating that the i^{th} variable in X_o belongs to the set $\aleph$.

To illustrate, consider the matrix Ψ in Equation (1.18) from the example proposed in the previous subsection. As previously analysed, the first two rows are linearly dependent, while the third row is linearly independent. Given that the rank of Ψ is 2, we expect to obtain rectangular matrices of rank 2 when deleting either of the first two rows. This can be observed by examining $\Psi_{(-B)}$ and $\Psi_{(-E)}$, where $\Psi_{(-B)}$ is Ψ with the first row removed, and $\Psi_{(-E)}$ is Ψ with the second row removed:

$$\Psi_{(-B)} = \begin{bmatrix} \gamma_2\beta_{o1} & \gamma_2\beta_{o3} & \gamma_2\beta_{o5} \\ \gamma_3\beta_{o2} & \gamma_3\beta_{o4} & \gamma_3\beta_{o6} \end{bmatrix}$$

$$\Psi_{(-E)} = \gamma\beta_o' = \begin{bmatrix} \gamma_1\beta_{o1} & \gamma_1\beta_{o3} & \gamma_1\beta_{o5} \\ \gamma_3\beta_{o2} & \gamma_3\beta_{o4} & \gamma_3\beta_{o6} \end{bmatrix}$$

Since $\begin{bmatrix} \beta_{o1} & \beta_{o3} & \beta_{o5} \end{bmatrix}$ is linearly independent with respect to $\begin{bmatrix} \beta_{o2} & \beta_{o4} & \beta_{o6} \end{bmatrix}$ and γ_1 and γ_3 are two scalars, it follows that the two matrices have rank 2, as anticipated. Conversely, the matrix $\Psi_{(-D)}$ obtained after removing from Ψ its third row, is

$$\Psi = \gamma\beta_o' = \begin{bmatrix} \gamma_1\beta_{o1} & \gamma_1\beta_{o3} & \gamma_1\beta_{o5} \\ \gamma_2\beta_{o1} & \gamma_2\beta_{o3} & \gamma_2\beta_{o5} \end{bmatrix}$$

Here, the resulting matrix is of reduced rank 1, given that the second row lies in the space spanned by the first (and vice-versa).

$\Psi_{(i)}$ can be removed by a manipulation of Q_t. The most intuitive approach involves directly eliminating the variable $Q_{(i),t}$. However, this method necessitates an extension of the rank test to account for cases where the dimensions of the left-hand side do not align with those of the right-hand side. To circumvent this step, an alternative strategy is proposed: substituting $Q_{(i),t}$ with a series of independently and identically distributed randomly

generated numbers. Specifically, we utilise a Gaussian white noise process denoted as:

$$W_t = \omega_t \qquad \omega \sim N(0, \sigma_\omega^2)$$

with $0 < \sigma_\omega^2 < \infty$.

Since W is generated independently of $\hat{Q}$ and R_1, its theoretical correlation with all other model's components is zero. Consequently, it is uncorrelated with the equilibria in $\beta_o' R_{1,t-1}$. This transformation renders $Q_{(i)}$ weakly exogenous and reduces $\Psi_{(i)}$ to a zero vector.

Setting $\Psi_{(i)} = \mathbf{0}$ has the same effect as dropping $\Psi_{(i)}$, as the zero vector does not span any space, and thus, it cannot increase the rank of the whole matrix.

Consequently, the model transforms into:

$$\tilde{Q}_t = \tilde{\Psi}_o R_{1,t} + \tilde{\eta}_{o,t} \tag{1.20}$$

where $\tilde{Q}_t$ denotes the manipulated left-hand side, obtained by replacing the columns of R_0 corresponding to the variables of interest.

Whether the estimated model changes when the modification is applied has not been proven and requires further analysis. However, what matters is not whether the estimates themselves change but whether the rank of β_o changes in accordance with the conclusions of Theorem 1 and its corollary. We do not have a proof, but we can make the following

reasoning: the left-hand side of the VECM remains unmodified, ensuring that $\beta_o' X_{o,t-1}$ remains stationary regardless of the substitution. Suppose that ΔX_i is correlated with the first column of $X_o\beta_o$. If $X_i \in \aleph$, then there exists an $X_j \in X_o$ such that its first difference is also correlated with the first column of $X_o\beta_o$. Consequently, by removing (or substituting with white noise) X_i, there will still be a variable in X_o whose first difference is correlated with the first column of $X_o\beta_o$, implying that β_o' will not change. Conversely, if $X_i \in \beth$, it is the sole variable whose first difference is correlated with that column of β_o'. Thus, removing ΔX_i from the left-hand side would mean that the first column of β_o could no longer be estimated, leading to a reduction in the rank of β_o'.

Based on the conclusions reached thus far, we propose an alternative formulation of hypotheses $\mathbf{H_{0m}}$ and $\mathbf{H_{1m}}$ based on regression rank rather than model constraints:

$\mathbf{H_{0r}}$ **:** *The cointegration rank of the model in Equation* (1.20) *is less or equal to* $r - |\Xi|$

$\mathbf{H_{1r}}$ **:** *The cointegration rank of the model in Equation* (1.20) *is greater than* $r - |\Xi|$

Here, r denotes the cointegration rank of the original model, specifically of the partially observed model or the regression in Equation (1.19), and $|\Xi|$ represents the cardinality of Ξ.

The isomorphism between $\mathbf{H_{0c}}$, $\mathbf{H_{1c}}$ and $\mathbf{H_{0r}}$, $\mathbf{H_{1r}}$ is crucial, as the latter pair aligns with the null and alternative hypotheses of the trace test or maximum eigenvalue test illustrated for instance in Johansen (1995) and Juselius (2006). Consequently, the causal hypotheses can be examined by applying Johansen's tests to model (1.20) to determine if the rank has decreased by $|\Xi|$ units.

Presented below are key points summarizing the steps for conducting the test on causal endogeneity:

- Determine Ξ and denote ι the set including the indexes representing the positions of its elements in X_o
- Estimate the cointegration rank r through standard procedures such as the Johansen trace test
- Obtain $R_{0,t}$ and $R_{1,t}$ by cleaning $\Delta X_{o,t}$ and $X_{o,t-1}$ for the short term dynamics.
- Estimate Σ_o from the residuals of the standard VEC model.
- Generate $\hat{Q}_t$ by multiplying $\hat{\Sigma}_o^{-1}$ and $R_{0,t}$
- Generate $\tilde{Q}_t$ by substituting the ι^{th} rows of $\hat{Q}_{0,t}$ with randomly generated white noises.
- Compute $S_{11} = N^{-1}\sum_{t=1}^{N} R_{1,t}R_{1,t}'$, $\tilde{S}_{00} = N^{-1}\sum_{t=1}^{N} \tilde{Q}_t\tilde{Q}_t'$, $\tilde{S}_{10} = N^{-1}\sum_{t=1}^{N} R_{1,t}\tilde{Q}_t'$, and $\tilde{S}_{01} = N^{-1}\sum_{t=1}^{N} \tilde{Q}_t R_{1,t}'$
- Solve the eigenvalue problem $\det\Big(\lambda S_{11} - \tilde{S}_{10}\tilde{S}_{00}^{-1}\tilde{S}_{01}\Big) = 0$ and sort the eigenvalues in descending order
- Compute the log-likelihood ratio statistic by using the formula $LRT = -N\ln\prod_{i=q}^{|X_o|}(1-\lambda_i)$, where $q = \hat{r} + 1 - |\Xi|$
- Compare the result with the non-standard distribution tabulated in Juselius (2006).

1.4.3 Relationship with Standard Weak Exogeneity

The analysis of the connections between weak causal exogeneity and conventional weak exogeneity reveals valuable insights, as detailed in the ensuing corollaries:

Corollary 2 (***Second Corollary to Theorem 1***)**.** *Weak exogeneity is a sufficient condition for weak causal exogeneity.*

Proof of Corollary 2. The corollary can be proven from the matrix representation of Equation (1.7):

$$\alpha_o = \begin{array}{c} \\ |\aleph| \\ |\beth| \end{array} \begin{array}{c} \begin{array}{cc} |\aleph| & |\beth| \end{array} \\ \begin{bmatrix} \mathcal{G} & \mathcal{H} \\ \mathcal{H}' & \mathcal{J} \end{bmatrix} \end{array} \begin{array}{c} \\ |\aleph| \\ |\beth| \end{array} \begin{array}{c} \begin{array}{cc} r-|\beth| & |\beth| \end{array} \\ \begin{bmatrix} (\mathcal{A}\mathcal{E}+\mathcal{D}\mathcal{F})_{\perp} & \mathbf{0} \\ \mathbf{0} & \boldsymbol{I} \end{bmatrix} \end{array} = \begin{bmatrix} \mathcal{G}(\mathcal{A}\mathcal{E}+\mathcal{D}\mathcal{F})_{\perp} & \mathcal{H} \\ \mathcal{H}'(\mathcal{A}\mathcal{E}+\mathcal{D}\mathcal{F})_{\perp} & \mathcal{J} \end{bmatrix}$$

The $|\beth| \times |\beth|$ sub-matrix $\mathcal{J}$ cannot have null vectors as rows. It indeed possess in the main diagonal the variances of the variables in ε_o corresponding to the elements in $\beth$. Since we deal with stochastic variables, the variance cannot be null. This verifies that only the rows of α_o corresponding to $\aleph$ can be null vector.

This completes the proof of the corollary.

□

Corollary 3 (***Third Corollary to Theorem 1***)**.** *Weak exogeneity is not a necessary condition for weak causal exogeneity.*

The necessity of providing a formal proof for Corollary 1.3 is obviated, as the specific case presented in section 1.3 suffices to demonstrate that weak causal exogeneity does not imply weak exogeneity.

In summary, weak exogeneity implies weak causal exogeneity. However, rejecting weak exogeneity does not necessarily imply causal endogeneity.

1.5 Strong Causal Exogeneity

This section investigates strong causal exogeneity and the restrictions it implies on the model coefficients. We explore the possibility of identifying causal information by modeling weakly causally exogenous elements separately and comparing the resulting residuals with the error term of the original model.

Subsection 1.5.1 explores the impact of strong causal exogeneity on estimable coefficients. Subsection 1.5.2 leverages these insights to devise an inferential procedure. Finally, Subsection 1.5.3 delineates the differences between strong causal exogeneity and the statistical notion of weak exogeneity.

1.5.1 Model Manifestations of Strong Causal Exogeneity

To detect strong causal exogeneity, we will focus on the covariance matrix Σ_o, retrieved as per Equation (1.8). If the elements in $\aleph$ are strongly causally exogenous, it holds that both $\aleph$ and $\daleth$ are not caused by $\beth$. In light of these considerations, under strong causal

exogeneity the matrices M_{ou} and N_o assume the structures depicted below:

$$M_{ou} = \begin{array}{c} \\ |\text{א}| \\ |\text{ב}| \end{array} \begin{array}{c} \begin{array}{cc} |\text{ד}| & |\text{ל}| \end{array} \\ \begin{bmatrix} \boldsymbol{\mathcal{A}} & \mathbf{0} \\ \mathbf{0} & \boldsymbol{\mathcal{C}} \end{bmatrix} \end{array} \qquad N_o = \begin{array}{c} \\ |\text{א}| \\ |\text{ב}| \end{array} \begin{array}{c} \begin{array}{c} |T| \end{array} \\ \begin{bmatrix} \boldsymbol{\mathcal{D}} \\ \mathbf{0} \end{bmatrix} \end{array} \tag{1.21}$$

In the conditional covariance matrix V, the sub-matrix relating ד to ל is null, as ל cannot cause ד and א, and ד cannot cause ב, therefore exclusing the possibility of collider type structure with elements of ד and ל serving as parents. As detailed in the preceding section, the sub-matrix of V connecting ל and T consists of null entries. Thus, the overall structure of V is as follows:

$$V = \begin{array}{c} \\ |\text{ד}| \\ |\text{ל}| \\ |T| \end{array} \begin{array}{c} \begin{array}{ccc} |\text{ד}| & |\text{ל}| & |T| \end{array} \\ \begin{bmatrix} \boldsymbol{\mathcal{K}} & \mathbf{0} & \boldsymbol{\mathcal{M}} \\ \mathbf{0} & \boldsymbol{\mathcal{N}} & \mathbf{0} \\ \boldsymbol{\mathcal{M}}' & \mathbf{0} & \boldsymbol{\mathcal{O}} \end{bmatrix} \end{array} \tag{1.22}$$

The following Theorem identifies a duality between model features and the condition of strong causal exogeneity. Specifically, it compares the covariance matrices of residuals from two models: one fitted to X_o and the other modeling א only. We denote $\varepsilon_{\text{א}}$ as the residuals corresponding to variables in א for the former model, and ε^* as the residuals of the latter model.

Theorem 2 (***Strong Causal Exogeneity and Equivalence between Models' Covariance Matrices***)**.** *If the variables in* א *are strongly causally exogenous, the covariance matrix* $\Sigma_{\text{א}} = Cov(\varepsilon_{\text{א}})$ *equals the covariance matrix* $\Sigma^* = Cov(\varepsilon^*)$

Proof of Theorem 2. To examine the covariance matrix of the residuals relative to א, we need to consider the |א|-dimensional full rank square sub-matrix constructed by selecting the rows and columns of Σ_o corresponding to א.

The residual covariance matrix of model (1.4) can be obtained by inserting Equations (1.21) and (1.22) into Equation (1.8). Implementing the matrix calculations yields the following representation for $\Sigma_{\aleph}$:

$$\Sigma_{\aleph} = \Big(\boldsymbol{\mathcal{A}}\boldsymbol{\mathcal{K}} + \boldsymbol{\mathcal{D}}\boldsymbol{\mathcal{M}}'\Big)\boldsymbol{\mathcal{A}}' + \Big(\boldsymbol{\mathcal{A}}\boldsymbol{\mathcal{M}} + \boldsymbol{\mathcal{D}}\boldsymbol{\mathcal{O}}\Big)\boldsymbol{\mathcal{D}}' + \Omega_{\aleph} \tag{1.23}$$

where $\Omega_{\aleph}$ is the diagonal sub-matrix of Ω_o relative to the residuals of א.

If only the variables in א are modeled, ב becomes unobserved, leading to modifications in the matrices of the state-space model. We use the superscript $*$ to denote the matrices relative to the model including the variables in א only, thus the covariance matrix of the model fitted to א will be denoted Σ^*.

According to the definition of strong causal exogeneity, ב cannot cause א. Consequently the sub-matrix M^*_{ou} connecting ב to א will contain zero entries. The resulting state-space coefficients are

$$M^*_{ou} = \begin{matrix} |\text{א}| \end{matrix}\begin{bmatrix} \begin{matrix} |\text{ד}| & |\text{ל}| & |\text{ב}| \end{matrix} \\ \boldsymbol{\mathcal{A}} \quad \mathbf{0} \quad \mathbf{0} \end{bmatrix} \qquad N^*_o = \begin{matrix} |\text{א}| \end{matrix}\begin{bmatrix} |T| \\ \boldsymbol{\mathcal{D}} \end{bmatrix} \tag{1.24}$$

The variables in א continues to block the forward paths between T and ל, as well as

the ones between $\{T, \text{ך}\}$ and ב. Moreover, since ב does not either directly or indirectly cause א, the sets $\{T, \text{ך}\}$ and ב do not have conditional shared ancestors. Therefore, the sub-matrices of $V^* = \text{Cov}\left[\{X_{ut}, T_t, \text{ב}_t\} \mid \text{א}_t\right]$ relating T to $\{\text{ל}, \text{ב}\}$ are null. The same goes for the sub-matrices of V^* relating ך and $\{\text{ל}, \text{ב}\}$. Consequently, the structure for the conditional covariance matrix V^* is as follows:

$$V^* = \begin{array}{c} \\ |\text{ך}| \\ |\text{ל}| \\ |\text{ב}| \\ |T| \end{array} \begin{array}{c} \begin{array}{cccc} |\text{ך}| & |\text{ל}| & |\text{ב}| & |T| \end{array} \\ \begin{bmatrix} \boldsymbol{\mathcal{K}}^* & \mathbf{0} & \mathbf{0} & \boldsymbol{\mathcal{M}}^* \\ \mathbf{0} & \boldsymbol{\mathcal{N}}^* & \boldsymbol{\mathcal{R}}^* & \mathbf{0} \\ \mathbf{0} & \boldsymbol{\mathcal{R}}^{*\prime} & \boldsymbol{\mathcal{S}}^* & \mathbf{0} \\ \boldsymbol{\mathcal{M}}^{*\prime} & \mathbf{0} & \mathbf{0} & \boldsymbol{\mathcal{O}}^* \end{bmatrix} \end{array} \tag{1.25}$$

By inserting (1.24) and (1.25) into (1.8) we obtain the following representation for Σ_s^*:

$$\Sigma^* = \left(\boldsymbol{\mathcal{A}}\boldsymbol{\mathcal{K}}^* + \boldsymbol{\mathcal{D}}\boldsymbol{\mathcal{M}}^{*\prime}\right)\boldsymbol{\mathcal{A}}' + \left(\boldsymbol{\mathcal{A}}\boldsymbol{\mathcal{M}}^* + \boldsymbol{\mathcal{D}}\boldsymbol{\mathcal{O}}^*\right)\boldsymbol{\mathcal{D}}' + \Omega_{\text{א}} \tag{1.26}$$

where $\boldsymbol{\mathcal{K}}^* = \text{Var}(\text{ך} \mid \text{א})$, $\boldsymbol{\mathcal{M}}^* = \text{Cov}(\text{ך}, T \mid \text{א})$, and $\boldsymbol{\mathcal{O}}^* = \text{Var}(T \mid \text{א})$.

Since the elements in ב do not interfere in the connections between the components of the set $\{T, \text{ך}\}$, i.e. it does not exist any indirect path between ב and $\{T, \text{ך}\}$ when conditioning on א, the covariances $\boldsymbol{\mathcal{K}}^*$, $\boldsymbol{\mathcal{M}}^*$, and $\boldsymbol{\mathcal{O}}^*$ equate $\boldsymbol{\mathcal{K}}$, $\boldsymbol{\mathcal{M}}$, and $\boldsymbol{\mathcal{O}}$ respectively. This can be formally proven by tracking the steps of the proof of Lemma 1, as it requires demonstrating that $\text{Cov}(\text{ך} \cup T|X_o) = \text{Cov}(\text{ך} \cup T|\text{א})$ under the hypothesis of strong causal exogeneity. To retrieve $\text{Cov}(\text{ך} \cup T|\text{א})$, the variables in ב are treated as unobserved, while only the variables in א are included in the observed set.

We begin by analyzing the covariance matrix $V = \mathrm{Cov}(Y_u \mid X_o)$. Under this scenario, the coefficients of Equation (1.9) assume the following structures:

$$Q^* = \begin{array}{c} \\ |ד \cup T| \\ |ל| \end{array} \begin{array}{c} \begin{array}{cc} |ד \cup T| & |ל| \end{array} \\ \begin{bmatrix} \boldsymbol{\mathcal{A}} & \mathbf{0} \\ \mathbf{0} & \boldsymbol{\mathcal{B}} \end{bmatrix} \end{array} \qquad C^* = \begin{array}{c} \\ |א| \\ |ב| \end{array} \begin{array}{c} \begin{array}{cc} |ד \cup T| & |ל| \end{array} \\ \begin{bmatrix} \boldsymbol{\mathcal{C}} & \mathbf{0} \\ \mathbf{0} & \boldsymbol{\mathcal{D}} \end{bmatrix} \end{array}$$

$$\Omega^* = \begin{array}{c} \\ |ד \cup T| \\ |ל| \end{array} \begin{array}{c} \begin{array}{cc} |ד \cup T| & |ל| \end{array} \\ \begin{bmatrix} \boldsymbol{\mathcal{E}} & \mathbf{0} \\ \mathbf{0} & \boldsymbol{\mathcal{F}} \end{bmatrix} \end{array} \qquad \Omega_o = \begin{array}{c} \\ |א| \\ |ב| \end{array} \begin{array}{c} \begin{array}{cc} |א| & |ב| \end{array} \\ \begin{bmatrix} \boldsymbol{\mathcal{G}} & \mathbf{0} \\ \mathbf{0} & \boldsymbol{\mathcal{H}} \end{bmatrix} \end{array}$$

It should be noted that these structures hold even in the absence of strong causal exogeneity, as causality from $T \cup$ ד to ל and vice-versa is excluded by construction. The same exclusion applies to causality from ל to א and from $T \cup$ ד to ב.

By substituting the coefficients above into Equation (1.9), and recognizing that the conditional covariance between ד $\cup T$ and ל is zero as per Lemma 1, we obtain the following representation for $\boldsymbol{W_1} = \mathrm{Cov}(T \cup$ ד$|X_o)$, i.e., for the $|T \cup$ ד$| \times |T \cup$ ד$|$ square sub-matrix constructed by considering the rows and columns of V corresponding to $T \cup$ ד:

$$\boldsymbol{W_1} = \boldsymbol{\mathcal{A}}\boldsymbol{W_1}\boldsymbol{\mathcal{A}}' + \boldsymbol{\mathcal{E}} - \boldsymbol{\mathcal{A}}\boldsymbol{W_1}\boldsymbol{\mathcal{C}}'(\boldsymbol{\mathcal{C}}\boldsymbol{W_1}\boldsymbol{\mathcal{C}}' + \boldsymbol{\mathcal{G}})^{-1}\boldsymbol{\mathcal{C}}\boldsymbol{W_1}\boldsymbol{\mathcal{A}}' \tag{1.27}$$

We now consider the covariance matrix of ד $\cup T$ conditioned on א only. In this case, ב is treated as latent, and א is the only observed variable. Without loss of generality, we combine ב and ל together, so that the two sets forming the latent variables are $T \cup$ ד and

$\beth \cup \lambda$. In the presence of strong causal exogeneity, causal feedback from $\beth$ to $\daleth$ is excluded, as well as feedback from $\beth$ to $\aleph$. Therefore, the coefficients of Equation (1.9) become:

$$Q_1^* = \begin{array}{c} \\ |\daleth \cup T| \\ |\lambda \cup \beth| \end{array} \begin{array}{c} \begin{array}{cc} |\daleth \cup T| & |\lambda \cup \beth| \end{array} \\ \begin{bmatrix} \boldsymbol{\mathcal{A}} & \mathbf{0} \\ \mathbf{0} & \boldsymbol{\mathcal{I}} \end{bmatrix} \end{array} \qquad \Omega_1^* = \begin{array}{c} \\ |\daleth \cup T| \\ |\lambda \cup \beth| \end{array} \begin{array}{c} \begin{array}{cc} |\daleth \cup T| & |\lambda \cup \beth| \end{array} \\ \begin{bmatrix} \boldsymbol{\mathcal{E}} & \mathbf{0} \\ \mathbf{0} & \boldsymbol{\mathcal{J}} \end{bmatrix} \end{array}$$

$$C_1^* = \begin{array}{c} \\ |\aleph| \end{array} \begin{array}{c} \begin{array}{cc} |\daleth \cup T| & |\lambda \cup \beth| \end{array} \\ \begin{bmatrix} \boldsymbol{\mathcal{C}} & \mathbf{0} \end{bmatrix} \end{array} \qquad \Omega_{o1} = \begin{array}{c} \\ |\lambda \cup \beth| \end{array} \begin{array}{c} \begin{array}{c} |\daleth \cup T| \end{array} \\ \begin{bmatrix} \boldsymbol{\mathcal{G}} \end{bmatrix} \end{array}$$

The subscript "1" is added to distinguish the above coefficients with the parameters of the equation related to $V = \mathrm{Cov}(Y_u|X_o)$. It should be emphasized that these representations are not valid in the absence of strong causal exogeneity. Specifically, in such cases, the submatrices in positions $\{1, 2\}$ in both Q_1^* and C_1^* would not be zero.

By selecting $V_1 = \mathrm{Cov}(Y_u \cup \beth|\aleph)$ as follows

$$V_1 = \begin{array}{c} \\ |\daleth \cup T| \\ |\lambda \cup \beth| \end{array} \begin{array}{c} \begin{array}{cc} |\daleth \cup T| & |\lambda \cup \beth| \end{array} \\ \begin{bmatrix} \boldsymbol{W_2} & \mathbf{0} \\ \mathbf{0} & \boldsymbol{W_3} \end{bmatrix} \end{array}$$

Equation (1.9) splits into two independent parts:

$$\begin{cases} \boldsymbol{W_2} = \boldsymbol{\mathcal{A}} \boldsymbol{W_2} \boldsymbol{\mathcal{A}}' + \boldsymbol{\mathcal{E}} - \boldsymbol{\mathcal{A}} \boldsymbol{W_2} \boldsymbol{\mathcal{C}}' (\boldsymbol{\mathcal{C}} \boldsymbol{W_2} \boldsymbol{\mathcal{C}}' + \boldsymbol{\mathcal{G}})^{-1} \boldsymbol{\mathcal{C}} \boldsymbol{W_2} \boldsymbol{\mathcal{A}}' \\ \boldsymbol{W_3} = \boldsymbol{\mathcal{I}} \boldsymbol{W_3} \boldsymbol{\mathcal{I}}' + \boldsymbol{\mathcal{F}} \end{cases}$$

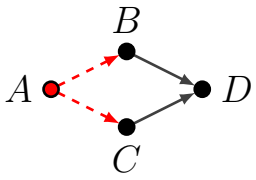

Figure 1.6: Example of Causal Graph Generating Equivalence Among the Covariance Matrices of $\varepsilon_{\aleph}$ and ε^*

Since $\boldsymbol{W_3}$ is positive semi-definite, and $\boldsymbol{\mathcal{F}}$ is a square matrix with positive entries in its main diagonal and zero otherwise, it follows that the above representation for $\boldsymbol{W_3}$ is suitable, as it results in a positive semi-definite matrix. Moreover, from (1.27), we can immediately understand that selecting $\boldsymbol{W_2} = \boldsymbol{W_1}$ provides an admissible result, given that $\boldsymbol{W_1}$ is positive semi-definite by construction. It follows that $\text{Cov}(T \cup \gimel | X_o) = \text{Cov}(T \cup \gimel | \aleph)$ under strong causal exogeneity. Consequently, Equations (1.23) and (1.26) are equal, and the covariance matrix of the residuals relative to $\aleph$ of the smaller model is the same as the covariance matrix of the residuals relative to $\aleph$ in the larger model.

This is sufficient to conclude the proof.

□

To illustrate the concepts in Theorem 2 and its corollary, consider the causal graph in Figure 1.6, where the observed variables are B, C, and D, while the only unobserved variable is the fundamental trend A. In this context, X_u is empty. The forward paths between A and D are blocked by B and C, while the fundamental trend directly influences B and C. Consequently, $\aleph = \{B, C\}$ and $\beth = \{D\}$. Naturally, $T = \{A\}$, $\gimel = \emptyset$, and $\daleth = \emptyset$. As we can see, D neither directly nor indirectly causes B and C, thus all the

elements in $\aleph$ are strongly causally exogenous.

The conditional variance $V = \mathrm{Var}(A|\{B, C, D\})$ is equal to

$$\mathrm{Var}(A|\{B, C\}) - \mathrm{Cov}(A, D|\{B, C\})\mathrm{Var}(D|\{B, C\})^{-1}\mathrm{Cov}(D, A|\{B, C\})$$

This follows from the identity $\mathrm{Cov}(A|B) = \mathrm{Cov}(A) - \mathrm{Cov}(A, B)\mathrm{Cov}(B)^{-1}\mathrm{Cov}(B, A)$. Since $\aleph$ blocks all the paths from A to D and D does not have feedback on $\aleph$ in such a way to form a collider-type structure where A and D serve as parents, it results that $\mathrm{Cov}(A, D|B, C) = 0$, thus $\mathrm{Var}(A|B, C, D) = \mathrm{Var}(A|B, C)$.

Since X_u is empty, the matrix M_{ou} does not exist. The matrix N_o, relating X_o to T, has two non-zero elements in its first and second rows, corresponding to B and C, given that A directly influences B and C. Conversely, there is no direct edge between A and D, so the third row of N_o contains a null entry. Hence, the structure of N_o is

$$N_o = \begin{bmatrix} a \\ b \\ 0 \end{bmatrix}$$

Substituting N_o and V into Equation (1.8), we obtain

$$\Sigma_o = \begin{bmatrix} a^2\text{Var}(A|\{B,C,D\}) + \Omega_B & ab\text{Var}(A|\{B,C,D\}) & 0 \\ ba\text{Var}(A|\{B,C,D\}) & b^2\text{Var}(A|\{B,C,D\}) + \Omega_C & 0 \\ 0 & 0 & \Omega_D \end{bmatrix} \tag{1.28}$$

where Ω_B, Ω_C, and Ω_D are the elements in the diagonal of Ω_o.

By modeling $\aleph$ only, the variable D is treated as unobserved. In this case, the set of ordinary latent variables is no longer empty, so that M^*_{ou} exists. However, since D has no causal feedback to $\aleph$, M^*_{ou} contains only zero entries. Therefore, the matrix Σ^* is

$$\Sigma^* = \begin{bmatrix} a^2\text{Var}(A|\{B,C\}) + \Omega_B & ab\text{Var}(A|\{B,C\}) \\ ba\text{Var}(A|\{B,C\}) & b^2\text{Var}(A|\{B,C\}) + \Omega_C \end{bmatrix}$$

Since $\text{Var}(A|B,C) = \text{Var}(A|B,C,D)$, the entries of Σ_o corresponding to A and B are equal to the matrix Σ^*. This aligns with Theorem 2, as it implies that the covariance matrix of the residuals corresponding to $\aleph$ in the VECM modeling X_o is equal to the residual's covariance matrix of the VECM modeling $\aleph$ only.

Corollary 4 (***First Corollary to Theorem 2***)**.** *If $\aleph$ contains non strongly causally exogenous elements, the covariance matrix $\Sigma_\aleph = Cov(\varepsilon_\aleph)$ differs from the covariance matrix $\Sigma^* = Cov(\varepsilon^*)$.*

Proof of Corollary 4. For brevity, we consider the set $Y_u = \{T, \beth, \gimel\}$ instead of ana-

lyzing the individual effects of T, ך, and ל.

Let $\boldsymbol{\mathcal{T}}$ denote the coefficient of the state space model relating א to Y_u, and $\boldsymbol{\mathcal{U}}$ denote the covariance of Y_u conditioned on X_o. The matrix $\Sigma_{\aleph}$ is obtained by inserting $\boldsymbol{\mathcal{T}}$ and $\boldsymbol{\mathcal{U}}$ into Equation (1.8), yielding

$$\Sigma_{\aleph} = \boldsymbol{\mathcal{T}}\boldsymbol{\mathcal{U}}\boldsymbol{\mathcal{T}}' + \Omega_{\aleph} \tag{1.29}$$

where $\Omega_{\aleph}$ is the square diagonal matrix containing the variances of the errors corresponding to א in the fully observed model.

Now, consider the model fitted to א only, treating ב as unobserved. Let $\boldsymbol{\mathcal{V}}$ represent the coefficient of the state space model relating א to ב, $\boldsymbol{\mathcal{U}}^*$ denote the covariance of Y_u conditioned on א, $\boldsymbol{\mathcal{W}}^*$ denote the covariance between Y_u and ב conditioned on א, and $\boldsymbol{\mathcal{X}}^*$ represents the covariance of ב conditioned on א. The matrix Σ^* obtained from Equation (1.8) is given by

$$\Sigma^*_{\Upsilon} = \boldsymbol{\mathcal{T}}\boldsymbol{\mathcal{U}}^*\boldsymbol{\mathcal{T}}' + \boldsymbol{\mathcal{V}}\boldsymbol{\mathcal{X}}^*\boldsymbol{\mathcal{V}}' + \boldsymbol{\mathcal{V}}\boldsymbol{\mathcal{W}}^{*\prime}\boldsymbol{\mathcal{T}}' + \boldsymbol{\mathcal{T}}\boldsymbol{\mathcal{W}}^*\boldsymbol{\mathcal{V}}' + \Omega_{\Upsilon} \tag{1.30}$$

To prove the inequality between the two covariance matrices, we rewrite $\boldsymbol{\mathcal{U}}$ in terms of $\boldsymbol{\mathcal{U}}^*$. Utilizing the relation $\text{Cov}(A|B) = \text{Cov}(A) - \text{Cov}(A,B)\text{Cov}(B)^{-1}\text{Cov}(B,A)$, we express $\boldsymbol{\mathcal{U}}$ as

$$\boldsymbol{\mathcal{U}} = \boldsymbol{\mathcal{U}}^* - \boldsymbol{\mathcal{W}}^*\boldsymbol{\mathcal{X}}^{*-1}\boldsymbol{\mathcal{W}}^{*\prime} \tag{1.31}$$

since $\text{Cov}(Y_u|\{\text{א},\text{ב}\}) = \text{Cov}(Y_u|\text{א}) - \text{Cov}(Y_u,\text{ב}|\text{א})\text{Cov}(\text{ב}|\text{א})^{-1}\text{Cov}(\text{ב},Y_u|\text{א})$. Inserting (1.31)

into (1.29), we observe that the equality between (1.29) and (1.30) holds only if

$$\boldsymbol{\mathcal{V}}\boldsymbol{\mathcal{X}}^{*}\boldsymbol{\mathcal{V}}' + \boldsymbol{\mathcal{V}}\boldsymbol{\mathcal{W}}^{*\prime}\boldsymbol{\mathcal{T}}' + \boldsymbol{\mathcal{T}}\boldsymbol{\mathcal{W}}^{*}\boldsymbol{\mathcal{V}}' - \boldsymbol{\mathcal{T}}\boldsymbol{\mathcal{W}}^{*}\boldsymbol{\mathcal{X}}^{*-1}\boldsymbol{\mathcal{W}}^{*\prime}\boldsymbol{\mathcal{T}}' = 0$$

By assumption, the variables in ℵ are not strongly causally exogenous, indicating a causal feedback between ⊐ and ℵ. Direct feedback is captured in $\boldsymbol{\mathcal{V}}$, while indirect feedback manifests in $\boldsymbol{\mathcal{W}}^{*}$. Given that ⊐ aither directly or indirectly causes ℵ, at least one between $\boldsymbol{\mathcal{V}}$ and $\boldsymbol{\mathcal{W}}^{*}$ must be non-null. If $\boldsymbol{\mathcal{V}} \neq 0$ and $\boldsymbol{\mathcal{W}}^{*} = 0$ (indicating exclusively direct feedback), the equality between (1.29) and (1.30) holds only if $\boldsymbol{\mathcal{V}}\boldsymbol{\mathcal{X}}^{*}\boldsymbol{\mathcal{V}}' = 0$, which is not possible since $\boldsymbol{\mathcal{X}}^{*}$ is a non-null full rank matrix. Conversely, if $\boldsymbol{\mathcal{V}} = 0$ and $\boldsymbol{\mathcal{W}}^{*} \neq 0$ (indicating exclusively indirect feedback), the equality between (1.29) and (1.30) holds only if $\boldsymbol{\mathcal{T}}\boldsymbol{\mathcal{W}}^{*}\boldsymbol{\mathcal{X}}^{*-1}\boldsymbol{\mathcal{W}}^{*\prime}\boldsymbol{\mathcal{T}}' = 0$, which is also not possible for the same reason.

If both $\boldsymbol{\mathcal{V}}$ and $\boldsymbol{\mathcal{W}}^{*}$ are non-null, the equality holds only under fine-tuned parameter values, as the entries of $\boldsymbol{\mathcal{X}}$ depend on $\Omega_{\beth}$, which is not dependent on the values of $\boldsymbol{\mathcal{V}}$ and $\boldsymbol{\mathcal{W}}^{*}$.

This concludes the proof of the corollary.

□

To clarify the findings of Corollary 4, consider the graph in Figure 1.6 with the addition of the causal connection $D \rightarrow C$, resulting in the graph depicted in Figure 1.4. This modification introduces a causal feedback from ⊐ to ℵ, implying that ℵ is no longer strongly causally exogenous.

In this modified graph, the fundamental trend and D form a collider-type structure on C. Consequently, their covariance is not zero when conditioning on C. Therefore, $\text{Cov}(A, D|B, C) \neq 0$, making the second term of

$$\text{Var}(A|B, C) - \text{Cov}(A, D|B, C)\text{Var}(D|B, C)^{-1}\text{Cov}(D, A|B, C)$$

non-zero, leading to the inequality $\text{Var}(A|B, C) \neq \text{Var}(A|B, C, D)$.

The structure of N_o remains unchanged with respect to the previous example, so the resulting Σ_o matrix is equivalent to the one in Equation (1.28), as shown below:

$$\Sigma_o = \begin{bmatrix} a^2\text{Var}(A|B, C, D) + \Omega_B & ab\text{Var}(A|B, C, D) & 0 \\ ba\text{Var}(A|B, C, D) & b^2\text{Var}(A|B, C, D) + \Omega_C & 0 \\ 0 & 0 & \Omega_D \end{bmatrix}$$

When modeling $\aleph$ only, the matrix M^*_{ou} exists and contains a non-zero entry in the row corresponding to C:

$$M^*_{ou} = \begin{bmatrix} 0 \\ \\ c \end{bmatrix}$$

This reflects the causal feedback that D has on C.

Substituting into Equation (1.8), we obtain:

$$\Sigma^* = \begin{bmatrix} a^2V_T^* + \Omega_B & acV_{uT}^* + abV_T^* \\ acV_{uT}^* + abV_T^* & c^2V_u^* + 2bcV_{uT}^* + b^2V_T^* + \Omega_C \end{bmatrix}$$

where $V_T^* = \text{Var}(A|\{B,C\})$, $V_u^* = \text{Var}(D|\{B,C\})$, and $V_{uT}^* = \text{Var}(A,B|\{B,C\})$.

In accordance with Corollary 4, the covariance matrix of the residuals corresponding to $\aleph$ in the VECM modeling X_o differs from the residual's covariance matrix of the VECM modeling $\aleph$ only. For example, this can be observed by comparing their first elements, given that $\text{Var}(A|B,C) \neq \text{Var}(A|B,C,D)$.

1.5.2 Testing Hypotheses on Strong Causal Exogeneity

The findings presented in section 1.5.1 can serve as a basis for translating causal assertions into hypotheses concerning the equality of covariance matrices. This lays the foundation for the construction of an inferential methodology aimed at identifying strongly causally exogenous variables. In fact, the null and alternative hypotheses

$\mathbf{H_{0c}}$ **:** *The set* $\Upsilon = \hat{\aleph}$ *contains only strongly causally exogenous variables*

$\mathbf{H_{1c}}$ **:** *Not every element of* $\Upsilon = \hat{\aleph}$ *is strongly causally exogenous*

find an isomorphism in the hypotheses

$\mathbf{H_{0m}}$ **:** *The covariance matrix of the residuals relative to* Υ *in the VECM fitted to* X_o *is equal to the covariance matrix of the residuals relative to* Υ *in the VECM modeling* Υ

only

$\mathbf{H_{1m}}$ **:** *The covariance matrix of the residuals relative to* Υ *in the VECM fitted to* X_o *is NOT equal to the covariance matrix of the residuals relative to* Υ *in the VECM modeling* Υ *only*

The equivalence between $\{\mathbf{H_{0c}}, \mathbf{H_{1c}}\}$ and $\{\mathbf{H_{0m}}, \mathbf{H_{1m}}\}$ results immediately from the findings of Theorem 2 and its first Corollary.

To implement a testing technique, it is necessary to estimate two models: one including X_o and the other including $\aleph$ only. The identification of the set $\aleph$ can be undertaken by employing the procedure elaborated in section 1.4.

Several methods can be employed to test hypotheses related to equality of variances, such as an F-test or a Bartlett's Test. In case the comparison is among covariance matrices it can be used the multivariate version of the Bartlett's Test, i.e. the Box's M Test. It must be emphasized that the implementation of these tests necessitates the independence of the series. However, this condition is likely violated for the residuals of the two models. To address this issue, it is suggested to partition the sample into two parts and use different sub-samples for the two series of residuals. It is important to note that this approach needs further investigation, and an assessment of its effectiveness is reserved for future research.

Below, we outline the key steps for the practical implementation of the strong causal exogeneity test employed in this paper:

- Set Υ as the estimated set of weakly causally exogenous elements, i.e. $\Upsilon = \hat{\aleph}$
- Determine the set Υ for which the hypothesis of strong causal exogeneity should be tested and denote ι as the set including the indexes representing the positions of its elements in X_o
- Estimate the VECM fitted to X_o and store ε_Υ, where ε_Υ represents the residuals in positions ι.
- Estimate the VECM fitted to the variables in Υ and store ε^*_Υ, where ε^*_Υ represents the residuals in positions ι^*.
- Take the first $N/2$ observations of ε_Υ as well as the last $N/2$ observations of ε^*_Υ and denote them respectively $\varepsilon_{\Upsilon,1:N/2}$ and $\varepsilon^*_{\Upsilon,N/2+1:N}$. Here N denotes the sample size of ε^*_Υ and ε_Υ.
- Estimate the covariance matrices $\Sigma_{\Upsilon,1:N/2} = \text{Cov}(\varepsilon_{\Upsilon,1:N/2})$ and $\Sigma^*_{\Upsilon,N/2+1:N} = \text{Cov}(\varepsilon^*_{\Upsilon,N/2+1:N})$, as well as the pooled covariance matrix $\Sigma_p = \frac{1}{N-2}\Big[(N/2-1)\Sigma_{\Upsilon,1:N/2} + (N/2-1)\Sigma^*_{\Upsilon,N/2+1:N}\Big]$
- Compute $M = (N-2)\ln\det(\Sigma_p) - (N/2-1)\ln\det(\Sigma_{\Upsilon,1:N/2}) - (N/2-1)\ln\det(\Sigma^*_{\Upsilon,N/2+1:N})$ and $C = \frac{2k^2+3k-1}{6(k+1)}\left(\frac{2}{N/2-1} - \frac{1}{N-1}\right)$, where k is equal to the dimension for the covariance matrices.
- Compute the test statistic as follows: $M(1-C)$
- Compare the test statistics with a χ^2 distribution with $\frac{k(k+1)}{2}$ degrees of freedom

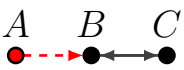

Figure 1.7: Causal Graph where Weak Exogeneity Does not Correspond to Strong Causal Exogeneity

1.5.3 Relationship with Standard Weak Exogeneity

The examination of the relationship between strong causal exogeneity and traditional weak exogeneity underscores significant disparities between these concepts, as demonstrated in the following corollaries:

Corollary 5 (***Second Corollary to Theorem 2***)**.** *Weak exogeneity is not a sufficient condition for strong causal exogeneity.*

Proof of Corollary 5. The corollary can be proven by providing a specific example where weak exogeneity does not imply strong causal exogeneity.

Consider the VECM below whose causal structure is described by the causal graph depicted in figure 1.7.

$$\Delta \begin{bmatrix} A \\ B \\ C \end{bmatrix}_t = \begin{bmatrix} 0 & 0 \\ a & 0 \\ 0 & b \end{bmatrix} \begin{bmatrix} c & 1 & d \\ 0 & e & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \end{bmatrix}_{t-1} + \varepsilon_t$$

Assume that the model user identifies B and C only, i.e., $X_o = \{B, C\}$. Since all the paths between the fundamental trend A and C are blocked by B, C belongs to $\beth$. Conversely, there exists a path between A and B that is not blocked by X_o, thus $B \in \aleph$. Despite

being weakly causally exogenous, B is not strongly causally exogenous due to the causal connection $C \to B$.

To assess standard weak exogeneity, we can use Equation (1.10). The state-space coefficient N_o contains a non-null entry only in the position relative to B. Consequently, its orthogonal complement will have a non-zero entry (exemplified by unity) only in the position relative to C. Without loss of generality, we set Ω_o equal to the identity matrix. The resulting adjustment matrix is $\alpha_o = \begin{bmatrix} 0 \\ \\ 1 \end{bmatrix}$, thus B is weakly exogenous despite not being strongly causally exogenous.

This completes the proof of the corollary.

□

Corollary 6 (***Third Corollary to Theorem 2***)**.** *Weak exogeneity is not a necessary condition for strong causal exogeneity.*

The necessity of providing a formal proof for this corollary is obviated, as the specific case presented in section 1.3 suffices to demonstrate that strong causal exogeneity and non weak exogeneity can co-exist.

In conclusion, the identification of weak exogeneity in a variable does not provide any information about the strong causal exogeneity property.

It must be emphasized that we have examined the concept of weak exogeneity with respect to the long-run parameter β_o. In case we change the parameters of interest, the relations

between standard weak exogeneity and strong causal exogeneity may vary. A thorough examination of this aspect is left to future research.

1.6 Example

The causal exogeneity analysis is illustrated in this section with the example of the theory of money, one of the most studied and contentious of economic concepts. We show in principle what our approach to causation can unearth about the role of money, assuming that the economic system adheres to the classical perspective of the quantity theory of money, as advocated by Hume. To construct a causal graph, we draw upon the conclusions outlined in section 1.1 of Hoover (2001), where the author synthesizes the predominant causal mechanisms inherent in Humean theory.

Hoover identifies five causal effects within Hume's major essays. Firstly, the *quantity theory of money* posits that the money supply (M) relative to available goods (Y) determines the price level (P), illustrated as $M \to P \leftarrow Y$.

The second causal mechanism, termed the *specie-flow mechanism* suggests that changes in relative prices influence the quantity of precious metal inversely, i.e. $P \to M$.

The third mechanism, known as the *loanable funds doctrine*, asserts that the level of interest rates (i) is determined by the supply and demand for loans (L),[14] implying the graphical representation $L \to i$.

[14] L is a variable denoting the interaction and relationship between the supply and demand for loans. For instance, it can represent their difference or their ratio. If it represents their ratio (i.e., Loan Demand / Loan Supply), then when $L > 1$, the interest rate i increases, while when $L < 1$, i decreases.

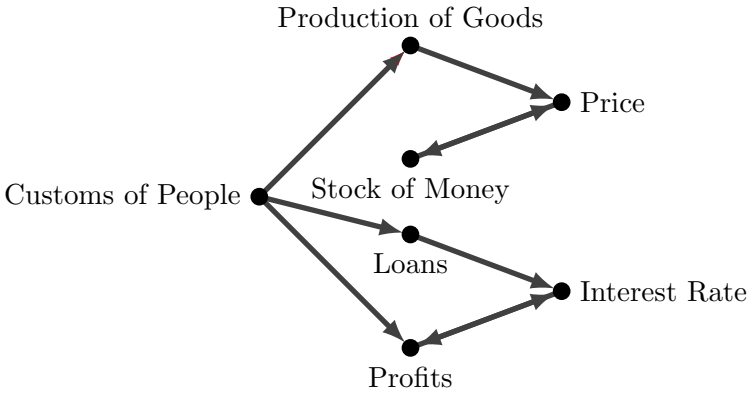

Figure 1.8: Graphical Structure of the Causal Links in the Humean Doctrine

The fourth mechanism extracted from Hume's work is the mutual causation between interest rates and profits. This phenomenon, known as the *arbitrage doctrine*, is represented by the cyclical graph $\pi \leftrightarrow i$.

Lastly, Hume emphasizes the pivotal role of societal customs in determining profit rates, as well as the demand and supply for goods and loans. Hume's *sociological doctrine* is graphically summarised as $CP \rightarrow \{Y, M, \pi\}$, where CP denotes the customs of people.

These relationships are combined together, and produce the cyclical directed graph depicted in Figure 1.8.

The fundamental origin of causality in the Humean model is traced back to societal norms and customs (CP). In long-run systems, the original source of causality is the source of non-stationarity, such that the variable CP constitutes the fundamental trend that induces non-stationarity in the ordinary variables $X = \{Y, M, L, \pi, P, i\}$.

The treatment of CP as a fundamental trend is arguably plausible, as it supposes the sociological factors are the accumulation of unpredictable shocks. Under this perspective,

CP aggregates all the non-economic influences on preferences, culture, and the traditions of individuals.

Following the methodology outlined in section 1.2 and in Hoover (2020), the graph can be translated into the following VECM representation of a C-VAR(1):

$$\Delta \begin{bmatrix} CP \\ Y \\ M \\ L \\ \pi \\ P \\ i \end{bmatrix}_t = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 & 0 \\ a & 0 & 0 & 0 & 0 & 0 \\ 0 & b & 0 & 0 & 0 & 0 \\ 0 & 0 & c & 0 & 0 & 0 \\ 0 & 0 & 0 & d & 0 & 0 \\ 0 & 0 & 0 & 0 & e & 0 \\ 0 & 0 & 0 & 0 & 0 & f \end{bmatrix} \begin{bmatrix} g & 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & h & 0 \\ i & 0 & 0 & 1 & 0 & 0 & 0 \\ j & 0 & 0 & 0 & 1 & 0 & k \\ 0 & l & m & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & n & p & 0 & 1 \end{bmatrix} \begin{bmatrix} CP \\ Y \\ M \\ L \\ \pi \\ P \\ i \end{bmatrix}_{t-1} + \varepsilon_t \tag{1.32}$$

The above VECM effectively captures the causal connections delineated in the graph. Notably, CP is weakly exogenous and remains unaffected by any equilibrium violation, consistent with the absence of edges pointing towards "Customs of People". Variable Y reacts to deviations from the equilibrium $Y = -gCP$, reflecting the causal relationship between Y and CP. Similarly, deviations from the equilibrium $M = -hP$ influence M, while deviations from $L = -iCP$ impact L. Profits (π) are influenced by multiple variables, and the economic forces influence their fluctuations when the equilibrium $\pi = -jCP - ki$ does not hold. This rationale extends to P and i.

The cointegration rank of the model is 6, equating to the difference between the total

number of variables and the number of fundamental trends.

The graph in Figure 1.8 embodies Hume's conceptualization of the economic system. We suppose Hume's conceptualization is accurate, in the sense that the graph depicts the true causal relations among macroeconomic variables, with Equation 1.32 serving as the data generating process. In essence, the graph illustrates natural orders, where causal connections are akin to natural laws. Other conceptualizations are of course possible, and might be more plausible *a priori*. The key point is of course that they too can be tested just as we outline how "Hume's model" can be tested.

Having described the functioning of the economic system, we now consider the econometrician's job. Numerous studies employ cointegrated systems to model monetary phenomena, as evidenced in works such as Johansen (1992c) and Johansen and Juselius (1990), among others. These studies commonly select money stock, GDP, price index, and interest rates[15] as relevant for the analysis. The analysis predominantly centers on the interplay between the money supply, represented by the money stock (M), and the determinants of money demand, namely Y, i, and P. The primary objective is to determine the nature and role of money in the system: whether and to what extent the demand of money influences the money supply, in other words, to what extent the money supply is exogenous. Following much of the literature, we solely consider M, Y, i, and P as modeled variables.

Thus, the sets are delineated as follows: the set encompassing the components of the fully observed model, is $\{CP, Y, M, L, \pi, P, i\}$; the set X_o comprising modeled variables is

[15]Both long and short-term interest rates have been utilized in the literature cited. For simplicity, we consider only one interest rate in this example.

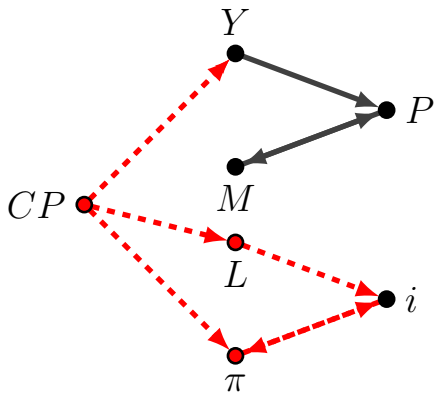

Figure 1.9: Observed and Latent Factors of Graph 1.8

$X_o = \{Y, M, P, i\}$; and the set Y_u containing latent elements is $Y_u = \{CP, L, \pi\}$. Given that the set T enclosing fundamental trends is the singleton set $T = \{CP\}$, the set X_u containing the unobserved ordinary variables is $X_u = \{L, \pi\}$. Figure 1.9 illustrates the relationship between observed and unobserved components, with red nodes and dotted edges representing latent variables and unobserved causal connections, respectively.

The graph shows the causal exogeneity properties of the observed variables. There exists a direct forward path between the fundamental trend CP and Y, while the forward paths between CP and i are mediated by L and π. Since those last two variables are unobserved, the paths between T and Y and T and i are not blocked by the components of X_o, implying $Y \in \aleph$ and $i \in \aleph$.

Practically, the exogenous nature of Y implies that, even with arbitrary manipulations of M, P, and i, control over the nominal level of Y remains unattainable. Its long-term fluctuations are directly influenced by levels of CP, which in turn remain unaffected by X_o. A similar reasoning applies to i: despite the forward paths between the fundamental

trend and i being indirect, arbitrary manipulations of Y, P, and M cannot regulate the nominal level of i.

Conversely, all the forward paths between T and $\{M, P\}$ are blocked by Y and the causal paths between Y and $\{M, P\}$ is direct, signifying that by modifying Y at will, one can control the long-run levels of both P and M. Consequently, both P and M are regarded as endogenously determined with respect to $X_o = \{Y, M, P, i\}$. $X_u = \{L, \pi\}$ are all parents of $i \in$ א. Thus, they belong to the set ך, while ל remains empty. Finally, the causal sets are as follows: $T = \{CP\}$, ך $= \{L, \pi\}$, א $= \{Y, i\}$, ב $= \{P, M\}$ and ל $= \emptyset$.

To illustrate the duality between causal sets and model restrictions, we need to obtain the coefficients of the partially observed models that can be estimated by the model user. We begin by demonstrating Theorem 1, i.e., the duality between linear dependence of rows of Ψ and elements of set א.

For this purpose, we require the matrices $\Sigma_o^{-1}\alpha_o = (M_{ou}V_{uT} + N_oV_T)_\perp$, β_o, and their product. As expounded in section 1.4, the rank of this model equals the cointegration rank of the partially observed system. Since N_o possesses full rank, it equals the number of modeled variables, i.e., 4, minus the number of elements in T, i.e., 1. Thus, the final rank is 3. The order for observed variables is Y, M, P, i, so that the first row-column of the coefficients related to the observed variables corresponds to Y, the second to M, the third to P and the last to i. The order of the unobserved ordinary variables is L, π such that the first row-column of the coefficients relative to X_u corresponds to L while the second corresponds to i.

The coefficients of the partially observed model are obtained from the parameters of the state-space representation in Equation (1.6). The specific entries are derived from the VECM representation of the fully observed system in equation (1.32):

$$M_o = \begin{bmatrix} a & 0 & 0 & 0 \\ 0 & b & bh & 0 \\ el & em & e & 0 \\ 0 & 0 & 0 & f \end{bmatrix} \qquad M_{ou} = \begin{bmatrix} 0 & 0 \\ 0 & 0 \\ 0 & 0 \\ fn & fp \end{bmatrix} \qquad N_o = \begin{bmatrix} ag \\ 0 \\ 0 \\ 0 \end{bmatrix}$$

$$M_u = \begin{bmatrix} c & 0 \\ 0 & d \end{bmatrix} \qquad M_{uo} = \begin{bmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & dk \end{bmatrix} \qquad N_u = \begin{bmatrix} ci \\ dj \end{bmatrix}$$

By inserting the matrices into equation (1.5), we obtain the following representation for the cointegration matrix β_o:

$$\beta_o' = \left(\begin{bmatrix} ag \\ 0 \\ 0 \\ -fj(n+p) \end{bmatrix}_\perp \right)' \begin{bmatrix} a & 0 & 0 & 0 \\ 0 & b & bh & 0 \\ el & em & e & 0 \\ 0 & 0 & 0 & f(1-pk) \end{bmatrix}$$

while the matrix $\Sigma_o^{-1}\alpha_o$ is

$$(M_{ou}V_{uT} + N_oV_T)_\perp = \begin{bmatrix} agV_{CP} \\ 0 \\ 0 \\ f\big(nV_{L,CP} + pV_{\pi,CP}\big) \end{bmatrix}_\perp \tag{1.33}$$

where $V_{A,B}$ represents the sub-matrix of V constructed by considering the rows corresponding to A and columns corresponding to B, while V_A is the square sub-matrix of V constructed from the rows and columns corresponding to A. An orthogonal complement does not have a unique representation. For consistency, we adopt the following form:

$$\begin{bmatrix} a \\ 0 \\ 0 \\ b \end{bmatrix}_\perp = \begin{bmatrix} -b & 0 & 0 \\ 0 & 1 & 0 \\ 0 & 0 & 1 \\ a & 0 & 0 \end{bmatrix}$$

Consequently, the final representation of the model in Equation (1.19) is

$$Q_t = \begin{bmatrix} f\big(nV_{L,CP} + pV_{\pi,CP}\big) & 0 & 0 \\ 0 & 1 & 0 \\ 0 & 0 & 1 \\ -agV_{CP} & 0 & 0 \end{bmatrix} \begin{bmatrix} afj(n+p) & 0 & 0 & afg(1-pk) \\ 0 & b & bh & 0 \\ el & em & e & 0 \end{bmatrix} R_{1,t} + \eta_t$$

The first and last row of Ψ are linearly dependent. This arises from both being derived by multiplying the first row of β'_o, denoted as $\beta'_{o(1)}$, by the scalars $\lambda = f\big(nV_{L,CP} + pV_{\pi,CP}\big)$

and $\delta = -agV_{CP}$. Consequently, $\Psi_{(1)} = \frac{\lambda}{\delta}\Psi_{(4)}$, confirming a linear dependency between the rows corresponding to $\aleph = \{Y, M\}$, in line with Theorem 1.

The second and third rows of Ψ correspond to the second and third rows of β_o'. Since the columns of the cointegration matrix are linearly independent by construction, $\Psi_{(2)}$ and $\Psi_{(3)}$ remain linearly independent. This result aligns with Corollary 1, given that the second and third modeled variables are respectively $M \in \beth$ and $P \in \beth$.

Thus far, we have used the duality set out in Theorem 1, and its first Corollary. Next, we outline practical steps for identifying $\aleph = \{Y, i\}$, as outlined in section 1.4.2.

To estimate model (1.19), it is necessary to obtain Σ_o^{-1}. This can be achieved by computing the residuals of the standard VECM fitted to X_o and estimating their covariance matrix. Subsequently, the series $\hat{Q}_t = \hat{\Sigma}_o^{-1} R_{0,t}$ is computed and regressed on $R_{1,t}$ using the reduced rank technique. The regression rank matches the original VECM's rank, which in our example is 3.

As specified in model (1.20), the substitution of the i^{th} row of $\hat{Q}_t$ with an independently generated white noise series renders the i^{th} variable weakly exogenous. Consequently, the i^{th} row of Ψ becomes a zero vector. Denoting $\tilde{\Psi}_A$ as the regression coefficient resulting after modifying the row of $\hat{Q}_t$ corresponding to A, the estimated outcomes for the various manipulations are as follows:

$$\tilde{\Psi}_Y = \begin{bmatrix} 0 & 0 & 0 & 0 \\ 0 & b & bh & 0 \\ el & em & e & 0 \\ -a^2gfj(n+p)V_{CP} & 0 & 0 & -(ag)^2f(1-pk)V_{CP} \end{bmatrix}$$

$$\tilde{\Psi}_M = \begin{bmatrix} f^2aj(n+p)\big(nV_{L,CP}+pV_{\pi,CP}\big) & 0 & 0 & f^2ag(1-pk)\big(nV_{L,CP}+pV_{\pi,CP}\big) \\ 0 & 0 & 0 & 0 \\ el & em & e & 0 \\ -a^2gfj(n+p)V_{CP} & 0 & 0 & -(ag)^2f(1-pk)V_{CP} \end{bmatrix}$$

$$\tilde{\Psi}_P = \begin{bmatrix} f^2aj(n+p)\big(nV_{L,CP}+pV_{\pi,CP}\big) & 0 & 0 & f^2ag(1-pk)\big(nV_{L,CP}+pV_{\pi,CP}\big) \\ 0 & b & bh & 0 \\ 0 & 0 & 0 & 0 \\ -a^2gfj(n+p)V_{CP} & 0 & 0 & -(ag)^2f(1-pk)V_{CP} \end{bmatrix}$$

$$\tilde{\Psi}_i = \begin{bmatrix} f^2aj(n+p)\big(nV_{L,CP}+pV_{\pi,CP}\big) & 0 & 0 & f^2ag(1-pk)\big(nV_{L,CP}+pV_{\pi,CP}\big) \\ 0 & b & bh & 0 \\ el & em & e & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}$$

We see that $\text{rank}(\tilde{\Psi}_Y) = \text{rank}(\tilde{\Psi}_i) = \text{rank}(\Psi) = 3$. This indicates that the manipulation of the rows corresponding to $\aleph = \{Y, i\}$ did not alter the overall rank. In contrast, $\text{rank}(\tilde{\Psi}_M) = \text{rank}(\tilde{\Psi}_P) = 2 < \text{rank}(\Psi)$, implying that the modification of the rows corre-

sponding to the elements of $\beth = \{M, P\}$ reduces the rank of the model.

The hypothesis $\text{rank}(\tilde{\Psi}_A) \leq 2$ can be tested employing the trace test or the maximum eigenvalue test. We expect to fail to reject the test for $\tilde{\Psi}_M$ and $\tilde{\Psi}_P$, but to reject for $\tilde{\Psi}_Y$ and $\tilde{\Psi}_i$.

Thus far, we have examined weak causal exogeneity. Now, we focus on the analysis of strong causal exogeneity.

As per section 1.3, a variable $x_i \in \aleph$ is deemed strongly causally exogenous if there exists no causal feedback from the elements in $\beth$ to x_i. No directed arrows originate from $\beth = \{P, M\}$ and point directly or indirectly towards $\aleph = \{Y, i\}$ in the graphical representation. Hence, both Y and i are strongly causally exogenous.

According to Theorem 2, the covariance matrix of the residuals associated with Y and i of the model fitted to $X_o = \{Y, M, P, i\}$ equals the covariance matrix of the residuals of the model fitted exclusively to Y and i.

To compute the covariance matrix Σ_o, we incorporate the coefficients of the state-space model into equation (1.8). The conditional variance matrix V contains non-zero entries as the covariance paths among the elements of Y_u are not blocked by X_o. The resulting covariance matrix for the model fitted to $X_o = \{Y, M, P, i\}$ is as follows:

$$\Sigma_o = \begin{bmatrix} (ag)^2 V_{CP} + \sigma_Y^2 & 0 & 0 & agf(nV_{L,CP} + pV_{\pi,CP}) \\ 0 & \sigma_M^2 & 0 & 0 \\ 0 & 0 & \sigma_P^2 & 0 \\ agf(nV_{L,CP} + pV_{\pi,CP}) & 0 & 0 & f^2(n^2 V_L + p^2 V_\pi + 2pnV_{L\pi}) + \sigma_i^2 \end{bmatrix} \tag{1.34}$$

When modeling $\aleph = \{Y, i\}$ only, variables M and P are treated as unobserved. Consequently, the conditional covariance matrix V is adjusted to correspond to $\text{Cov}(\{L, \pi, CP, P, M\} \mid \{Y, i\})$. As we can see from the graph in figure 1.9, the covariance paths between $\{CP, L, \pi\}$ and $\{P, M\}$ are blocked by the node corresponding to Y. Moreover, it follows from strong causal exogeneity that $\{T, \daleth\}$ and $\beth$ do not share the same conditional descendants, resulting in zero entries in the sub-matrices relating those two sets. Moreover, since P and M do not interfere with L, π and CP, the conditional covariance matrix $\text{Cov}(\{L, \pi, CP\} \mid \{Y, i\})$ equates $\text{Cov}(\{L, \pi, CP\} \mid X_o)$. The final representation for the residual covariance matrix is:

$$\Sigma_o^* = \begin{bmatrix} (ag)^2 V_{CP} + \sigma_Y^2 & agf(nV_{L,CP} + pV_{\pi,CP}) \\ agf(nV_{L,CP} + pV_{\pi,CP}) & f^2(n^2 V_L + p^2 V_\pi + 2pnV_{L\pi}) + \sigma_i^2 \end{bmatrix}$$

Σ_o^* equals the sub-matrix of Σ_o constructed by considering the rows and columns corresponding to Y and i. In fact, $\Sigma_{o,11}^* = \Sigma_{o,11}$, $\Sigma_{o,12}^* = \Sigma_{o,14}$, $\Sigma_{o,21}^* = \Sigma_{o,41}$ and $\Sigma_{o,22}^* = \Sigma_{o,44}$.

So far, we illustrated the duality discovered in section 1.5. To practically identify the strongly causally exogenous elements one can follow the procedure in section 1.5.2: Ini-

tially, the VECM is estimated for $X_o = \{Y, M, P, i\}$, and the residuals for variables Y and i are retrieved. Subsequently, $\aleph$ is modeled by fitting a VECM exclusively to Y and i. The rank of this model is 1, given that N_o is a 2×1 matrix of rank 1. The covariance among the two pairs of residuals is then compared using the Box's M test, as detailed in section 1.5.2. The anticipated outcome is the failure to reject the null hypothesis of equality between the two covariance matrices.

To illustrate the distinction between standard weak exogeneity and causal exogeneity, we compute the adjustment matrix α_o. This is obtained by inserting (1.33) and (1.34) into Equation (1.7). The resulting matrix is given by:

$$\alpha_o = \begin{bmatrix} \alpha_Y & 0 & 0 \\ 0 & \alpha_M & 0 \\ 0 & 0 & \alpha_P \\ \alpha_i & 0 & 0 \end{bmatrix}$$

where $\alpha_Y \neq 0$, $\alpha_M \neq 0$, $\alpha_P \neq 0$, and $\alpha_i \neq 0$.

This matrix demonstrates that α_o does not contain null rows, indicating that the hypothesis of weak exogeneity is anticipated to be rejected for all four modeled variables. Consequently, in this scenario, standard weak exogeneity is not informative from a causal perspective, and neither therefore are the notions of strong and super exogeneity (of Engle et al. (1983)), as these both require weak exogeneity. By way of contrast, in the example

model of this section, with the assumed set of observed variables, both Y, i are weakly and strongly causally exogenous. Our example shows that the conclusions we draw based on the definitions of weak and strong causal exogeneity may not match the statisitcal notion of weak exogeneity.

1.7 Conclusions

This chapter contributes to the literature on causality in economics by showing that some key aspects of latent causal graphs can be framed as restrictions on the coefficients of partially-observed vector error correction models, even in the absence of knowledge of all the relevant variables. The absence of this requirement - the requirement of complete specification - makes our approach practicable and reliable. Generally in statistical analysis the failure to adequately account for "confounding" factors or explanatory variables can severely limit what can be learnt from the analysis.

The duality we have discovered between causal graphs and restrictions on model parameters allows for the development of inferential procedures to determine which variables exhibit weak and strong causal exogeneity. Weak causal exogeneity refers to the capacity to influence the nominal value of endogenous variables, while strong causal exogeneity adds to this the absence of causal feedback on to the exogenous variable.

Our analytical framework facilitates a generic assessment of causality, circumventing the conventional assumptions typically imposed in causal inference literature, which include

the requirement for acyclicity. This framework is particularly advantageous in the context of long-run systems, where mutual influences in equilibrium adjustments are widely acknowledged in the economics literature. Our approach is based on the CVAR, and is ostensibly related to well known tests of weak exogeneity, and which variables can be regarded as "driving forces". However, we have shown that whether or not a variable is weakly exogenous is unrelated to whether or not it is strongly causal exogenous and is only a sufficient condition for weak causal exogeneity. The notions of weak and strong causal exogeneity we introduce in this paper are distinct from the Engle et al. (1983) notion of weak exogeneity, and the implementation of this notion in CVARs. This is illustrated by our Money Example in section 1.6 for an assumed "economic structure". The utility of our approach extends beyond monetary economics. For example we note that Franchi and Juselius (2007) find the *surprising* result that the weak exogeneity of private capital formation can be rejected, while the weak exogeneity of per capita consumption can not be rejected. This is surprising from the point of view of the Real Business Cycle school, as it suggests shocks to capital formation are not a driving stochastic trend, but shocks to per capita consumption are, at odds with what is expected to be the case. Nevertheless, it might be the case that private capital formation is weakly or strongly causally exogenous despite not being weakly exogeneous (matching Y, i in section 1.6), and the use of these concepts might be more relevant.

The following chapters further explore the concept of causal exogeneity. Specifically, Chapter 2 extends the analysis of weak causal exogeneity by identifying a further du-

ality between model characteristics and weak causal exogeneity, as well as by assessing test performance using simulated data. Additionally, it proposes an empirical illustration to demonstrate the practical utility of causal exogeneity.

Chapter 3 delves deeper into the causal information embedded in standard weak exogeneity. This chapter goes beyond weak and strong causal exogeneity, focusing on the concepts of non-causality and driving forces. It aims to provide a comprehensive understanding of how standard statistical notions contribute to the broader framework of long-run causal analysis in cointegrated systems.

Chapter 2

A New Log-Likelihood Ratio Test for the Identification of Long-Run Causal Endogeneity

2.1 Introduction

The meaning of exogenous and endogenous can be ambiguous, and may depend on contextual considerations. For instance, in monetary economics, Palley (2002) has noted various facets of money's endogeneity/exogeneity. If the focus is on the origin or the evolution of money, the analysis could be said to concern *Evolutionary Exogeneity* or the *Originary Endogeneity*. If, instead, what matters is not the nature of the variable of interest, but its properties within a particular institutional setting, the analysis could be termed the *Quantitative endogeneity/exogeneity* or *Control endogeneity/exogeneity*.

From an econometric or statistical perspective, the emphasis is on *Statistical endogene-*

ity/exogeneity. The seminal paper by Engle et al. (1983) proposed three different definitions of exogeneity, each serving a specific statistical purpose: weak exogeneity, strong exogeneity and super exogeneity. Broadly, statistical exogeneity implies a variable's independence from others, while endogeneity suggests its joint determination with other system variables. However, as argued by numerous scholars (see for instance Sieroń (2019)), variables are inherently interconnected, rendering a variable's exogeneity context-dependent within a specific model framework.

Clearly, the economic debate is not about statistical exogeneity, since what matters in this context is the appropriateness of the model rather than the nature of the economic magnitude. On the other hand, for macro-economists, what really matters is causality rather than the joint probability properties of the quantities under analysis.

The findings of the first chapter reconcile statistical and economic perspectives on exogeneity by proposing a definition that is related to the statistical notion, but nevertheless reflects the notion of control exogeneity. The proposed definition of exogeneity is context-dependent and relies on the variables included in the model. Specifically, this concept of exogeneity is not an inherent property of a variable but rather a relational property between a variable and a specific set of other variables. Consequently, whether a variable is classified as exogenous or endogenous depends on the particular variables included within the model. At the same time, the definition is grounded in the system's latent causal structure, which also encompasses unobserved (unidentified) components. Since the definition is anchored to the causal structure, the terminology causal exogeneity/endogeneity

will be used.

Causal endogeneity is related to the concept of governability. A variable within a set X is deemed causally endogenous if its expected nominal value can be fully controlled by other components in X. In other words, it is possible to obtain any level of the causally endogenous variables by arbitrarily manipulating the causally exogenous components. Naturally, this must be considered in a hypothetical scenario since the possibility of manipulating the causally exogenous variables at will is not ensured.

In terms of causal graphs, a set of variables X_W can control the expected nominal value of a generic X_i element if every directed forward path between X_i and the original source of causality is blocked by the elements in X_W and the transmission of causality between X_W and X_i is either direct or passes through unobserved components which do not in turn affect X_W. In cointegrated systems, the original source of causality corresponds to the primary source of non-stationarity, assumed to be generated by fundamental trends.[1] Coherently with the previous chapter, we will use the notation T to indicate the set containing the fundamental trends and X_o to indicate the set of modeled variables. X_o can be further split into two subsets: $\aleph$ and $\beth$. The former encloses causally exogenous variables while the latter embodies causally endogenous variables.

The ability (in principle) to manipulate a variable fails to capture certain nuances of exogeneity, especially in the presence of cyclical graphs. An example of this is that causal

[1]Non-stationarity can be generated either by the presence of fundamental trends or by a fine tuning of the parameters. However, this second scenario is less likely since even a small change in the parameter would lead to a loss of cointegration and would make the variables stationary. For further details, see the discussion in Hoover (2020)

exogeneity does not exclude causal feedbacks from ב to א. This creates the need for a twofold definition: on one hand, we have the variables in א-the ones that with a hypothetical experiment can control ב-on the other, we can delineate the subset of א containing the variables affected neither directly nor indirectly by the causally endogenous variables. Hence causal exogeneity can occur in weak and strong forms.

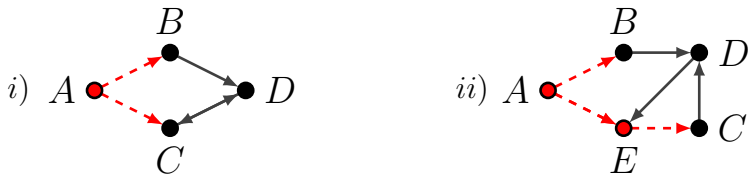

Figure 2.1: Causal Graphs Exemplifying Causal Exogeneity in its Weak and Strong Forms

The graphs in Figure 2.1 illustrate the difference between strong and weak causal exogeneity. The red nodes and edges represent the unobserved variables and latent causal path, respectively, thus, the exogenous characteristics should be determined based on the set $X_o = \{B, C, D\}$.

In both graphs, the primary source of causality is the unobserved A. In graph i), A is directly connected with B and C, so no variable interferes with the causal paths between them and the fundamental trends. Consequently, $\aleph = \{B, C\}$. Conversely, in graph ii), only B is directly connected with A, since the variable E stands between A and C. Nevertheless, E is unobserved, therefore no variable in X_o blocks the direct forward path between A and C. Similarly to the previous graph, $\aleph = \{B, C\}$. In both cases, the set ב is a singleton set containing D, as the forward paths between the fundamental trend and D are all blocked by B and C.

However, the degree of exogeneity of B differs from that of C because of the causal feedback between $\beth$ and C. In graph i), the causal connection between D and C is direct, while it passes through E in graph ii). In both cases, an intervention on C aimed at modifying the expected value of D will have a repercussion on C itself. This could nullify, mitigate, or amplify the planned influence on D. Consequently, only B is also strongly causally exogenous, while C is exclusively weakly causally exogenous.

This chapter focuses on weak causal exogeneity, and the problem of identifying it in the context of long-term causality. In the next section, we will review the results illustrated in the previous chapter and show how the coefficients of the partially observed Vector Error Correction Model (VECM) for X_o incorporate information about the weakly causally exogenous variables. Section 2.3 identifies a new duality between model characteristics and causal endogeneity and illustrates how to exploit it to construct inferential procedures for causal identification. Section 2.4 compares the performance of the two procedures for testing hypotheses on causal endogeneity. This comparison is conducted using simulated datasets generated from different types of causal structures. Section 2.5 presents an empirical illustration of monetary theory aimed at illustrating the practical value of the tools here introduced. Finally, section 2.6 provides concluding remarks and summarises the findings.

2.2 Properties and Inference of Causally Exogenous Variables

The analysis focuses on long-term models, where causality is defined in terms of equilibrium adjustments and violations of steady state. This section summarises the properties of the underlying model and the analyses of the partially observed models. The assumptions and notations align with those presented in Section 1.2, so this section serves as a reminder of the earlier content.

As outlined in the introduction, causal exogeneity is determined relative to observed variables. Let X_o denote the set of observed variables and X_u the set containing the unobserved ordinary variables. The set Y is defined as the union of X_o, X_u and T, namely, it is the set comprising all the variables in a system.

As shown in Hoover (2020), the causal structure of Y can be readily translated into a Vector Error Correction Model (VECM) by incorporating equilibrium relationships into the cointegration matrix and constructing the adjustment matrix α to ensure that affected variables react appropriately (see also chapter 1).

For illustration, consider $Y = \{A, B, C\}$ and the causal structure represented by the collider $A \rightarrow C \leftarrow B$. Since only C is causally affected, it is the sole variable reacting to steady state violations. Consequently, the adjustment matrix has a non-null entry exclusively in the row corresponding to C, and the cointegration matrix embodies the

equilibrium relationship among the three variables. The resulting VECM is illustrated below:

$$\Delta \begin{bmatrix} A \\ B \\ C \end{bmatrix}_t = \begin{bmatrix} 0 \\ 0 \\ a \end{bmatrix} \begin{bmatrix} b & c & d \end{bmatrix} \begin{bmatrix} A \\ B \\ C \end{bmatrix}_{t-1} + \varepsilon_t$$

Consider another example where the causal structure for Y is represented by the chain $A \rightarrow B \rightarrow C$. In this case, there are two causally affected variables, implying two equilibria: one affecting B and the other influencing C. The adjustment matrix must be configured to relate ΔB_t to violations of the equilibrium between A and B, and ΔC_t to violations of the equilibrium between B and C, as illustrated in the following VECM:

$$\Delta \begin{bmatrix} A \\ B \\ C \end{bmatrix}_t = \begin{bmatrix} 0 & 0 \\ a & 0 \\ 0 & b \end{bmatrix} \begin{bmatrix} c & d & 0 \\ 0 & e & f \end{bmatrix} \begin{bmatrix} A \\ B \\ C \end{bmatrix}_{t-1} + \varepsilon_t$$

To align with assumptions from previous literature on partially observed VECM, we assume that fluctuations of ΔY are solely generated by long-term dynamics. Thus, the model:

$$\Delta Y_t = \alpha \beta' Y_{t-1} + \varepsilon_t \tag{2.1}$$

is structural, with the term ε_t containing primitive shocks. Consequently, $\Omega = \text{Var}(\varepsilon)$ is diagonal by construction.

Consider the set $X_o \subseteq Y$ encompassing the variables identified and modeled by the user,

and denote Y_u as $Y \setminus X_o$. The elements in Y_u are treated as unobserved. Consequently, the model user cannot estimate the fully observed VECM in (2.1), and the analysis has to focus on the partially observed model below:

$$\Delta X_{o,t} = \alpha_o \beta_o' X_{o,t-1} + \sum_{i=1}^{\infty} \Gamma_{o,i} \Delta X_{o,t-i} + \varepsilon_{o,t} \tag{2.2}$$

where the subscript "o" stands for observed and distinguishes parameters and variables related to the estimated model from those of the fully observed VECM modeling Y.

It should be emphasized that the characteristics of model (2.1) do not generally extend to model (2.2). Consequently, $\Sigma_o = \text{Var}(\varepsilon_o)$ does not need to be diagonal and $\Gamma_{o,i=1:\infty}$ is not necessarily zero.

For estimation purposes, model (2.2) is expressed in a reduced form, eliminating short-term dependencies using the Frisch-Waugh theorem:

$$R_{0,t} = \alpha_o \beta_o' R_{1,t} + \varepsilon_{o,t}$$

In section 4 of Chapter 1, it has been proven the existence of a duality between weak causal exogeneity and the properties of the following "synthetic" model:

$$\Sigma_o^{-1} R_{0,t} = \Sigma_o^{-1} \alpha_o \beta_o' R_{1,t} + \Sigma_o^{-1} \varepsilon_{o,t}$$

which can be rewritten as:

$$Q_t = \gamma\beta_o' R_{1,t-1} + \eta_t = \Psi R_{1,t} + \eta_t \tag{2.3}$$

The matrix Σ_o is the covariance matrix of the residuals ε_o. Σ_o^{-1} is not directly observable, but it can be estimated from the residuals of the VECM fitted to X_o. Thus, in practical application Σ_o^{-1} is substituted with $\hat{\Sigma}_o^{-1}$ as well as Q_t, is substituted with $\hat{Q}_t$.

Since $\mathrm{Cov}(\varepsilon_o, \alpha_o\beta_o R_{1,t}) = 0$, it follows that the covariance between η_t and $\Psi R_{1,t}$ is zero, ensuring that standard estimators of Ψ produce unbiased estimates.

The basis for the inference of the elements in $\beth$ is the following statement summarizing the conclusions of Theorem 1 and Corollary 1: The i^{th} row in Ψ is linearly dependent if the i^{th} element of X_o belongs to $\aleph$. Conversely, it is linearly independent if the i^{th} element of X_o belongs to $\beth$.

The statement on the rows of Ψ can be readily extended to the rows of γ: The i^{th} row in γ is linearly dependent if the i^{th} element of X_o belongs to $\aleph$. Conversely, it is linearly independent if the i^{th} element of X_o belongs to $\beth$. The proof is trivial since $\Psi = \gamma\beta_o'$. In fact, if there exists a column vector $\boldsymbol{\lambda}$ with non zero entries in the i^{th} and some other positions such that $\boldsymbol{\lambda}'\gamma = 0$, it follows that $\boldsymbol{\lambda}'\gamma\beta_o' = 0$. Conversely, if $\boldsymbol{\lambda}'\gamma \neq 0$ for any non-trivial vector, then it necessarily holds that $\boldsymbol{\lambda}'\gamma\beta_o' \neq 0$.

The duality between causal endogeneity and linear dependence can be leveraged to identify $\beth$. The mechanism of the inferential procedure proposed in Chapter 1 exploits the effect

that the substitution of a row of Ψ with a null vector has on the matrix rank. In fact, by substituting a linearly independent row with a null vector, the rank of the modified matrix decreases by a unit. If a linearly dependent row is substituted, the rank of the modified matrix equals the rank of the original matrix.

In general, by substituting n linearly independent rows with null vectors, the rank of the modified matrix reduces by n units. Conversely, if not all the substituted rows were linearly independent, the rank of the modified matrix decreases by k units, where $0 \leq k < n$.

Given an arbitrarily selected set Ξ, the causal hypothesis

$$\mathbf{H_{0c}}: \textit{The elements in } \Xi \subset X_o \textit{ belong to } \beth$$

can be translated into a sentence relative to model characteristics:

$$\mathbf{H_{0m}}: Rank(\Psi_{(-\Xi)}) = Rank(\Psi) - |\Xi|$$

where $\Psi_{(-\Xi)}$ is the matrix obtained by substituting the rows of Ψ corresponding to Ξ with null vectors.

Similarly, the alternative causal hypothesis

$$\mathbf{H_{1c}}: \textit{Not every element in } \Xi \textit{ is causally endogenous}$$

translates to:

$$\mathbf{H_{1m}}: Rank(\Psi_{(-\Xi)}) > Rank(\Psi) - |\Xi|$$

The duality between causal and model hypotheses is important since the pair $\{\mathbf{H_{0m}}, \mathbf{H_{1m}}\}$ corresponds to the null and alternative of the maximum eigenvalue and trace test known

as Johansen's tests.[2] This implies that standard techniques for inferring the regression rank can be employed to indirectly test causal hypotheses.

To substitute the rows of Ψ with zero vectors, one could manipulate the left-hand side of regression (2.3) by replacing the variables relative to Ξ with independent processes. These substituted variables will be uncorrelated with $\beta_o' X_{o,t-1}$, resulting in null entries in the corresponding rows of the theoretical representation of γ. A null row in γ necessarily produces a null row in Ψ, thus $\Psi_{(-\Xi)}$ can be estimated by regressing $Q_{(-\Xi),t}$ on R_1, where $Q_{(-\Xi)}$ substitutes the columns of Q relative to Ξ with independent processes (e.g. randomly generated white noises). Thus, the regression for the causal endogeneity test is

$$Q_{(-\Xi),t} = \Psi_{(-\Xi)} R_{1,t-1} + \nu_t$$

This model is a reduced rank regression, and its coefficients need to be estimated using the procedures described in Anderson (1951). Since $\Psi_{(-\Xi)}$ is equivalent to the impact matrix of a standard VECM, its rank can be inferred using procedures described in Johansen (1995) and Juselius (2006).

This chapter analyses the performance of this test through simulations and compares the results with an alternative procedure proposed in the next section. In section 2.5, both tests on causal endogeneity are applied to an empirical study on money endogeneity.

[2]The null hypothesis of the Johansen's tests should be $\text{Rank}(\Psi_{(-\Xi)}) \leq \text{Rank}(\Psi) - |\Xi|$. However, the hypothesis $\text{Rank}(\Psi_{(-\Xi)}) < \text{Rank}(\Psi) - |\Xi|$ is not admissible since the reduction of the rank generated by the substitution of n rows can be by a maximum of n units.

2.3 Alternative Test on Causal Endogeneity

This section introduces an alternative procedure for identifying causally endogenous variables based on the restrictions of the orthogonal complement of the parameter γ.

The analysis relies on the formulae proposed by Johansen (2019) in his analysis of partially observed vector error correction models. His formulae provide a representation for α_o, β_o and Σ_o starting from the state-space arrangement of a cointegrated system:

$$\Delta X_{o,t} = M_o X_{o,t-1} + M_{ou} X_{u,t-1} + N_o T_{t-1} + \nu_{o,t}$$

$$\Delta X_{u,t} = M_{uo} X_{o,t-1} + M_u X_{u,t-1} + N_u T_{t-1} + \nu_{u,t}$$

$$\Delta T_t = \nu_{T,t}$$

Here, the coefficients M_o, M_{ou} and N_o relate X_o to itself, X_u and T respectively. Likewise, the coefficients M_u, M_{uo} and N_u relate X_u to itself, X_o and T respectively.

To clarify, consider the VECM below, and set $X_o = \{B, D\}$

$$\Delta \begin{bmatrix} A \\ B \\ C \\ D \end{bmatrix}_t = \begin{bmatrix} 0 & 0 & 0 \\ a & 0 & 0 \\ 0 & b & 0 \\ 0 & 0 & c \end{bmatrix} \begin{bmatrix} d & 1 & 0 & 0 \\ 0 & e & 1 & 0 \\ 0 & f & g & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \\ D \end{bmatrix}_{t-1} + \begin{bmatrix} \varepsilon_A \\ \varepsilon_B \\ \varepsilon_C \\ \varepsilon_D \end{bmatrix}$$

Clearly, the fundamental trend is A, while X_u is a singleton set composed of C. In this specific example, the state-space model is

$$\Delta \begin{bmatrix} B \\ D \end{bmatrix}_t = \begin{bmatrix} a & 0 \\ cf & c \end{bmatrix} \begin{bmatrix} B \\ D \end{bmatrix}_{t-1} + \begin{bmatrix} 0 \\ cg \end{bmatrix} C_{t-1} + \begin{bmatrix} ad \\ 0 \end{bmatrix} A_{t-1} + \begin{bmatrix} \varepsilon_B \\ \varepsilon_D \end{bmatrix}_t$$

$$\Delta C_t = bC_{t-1} + \begin{bmatrix} be & 0 \end{bmatrix} \begin{bmatrix} B \\ D \end{bmatrix}_{t-1} + 0 \times A_{t-1} + \varepsilon_{C,t}$$

$$\Delta A_t = \varepsilon_{A,t}$$

It is evident that the state-space representation is a trivial adjustment of the fully observed model delineated in vector error correction form.

Starting from the coefficients of the state-space representation, Johansen's analysis discovered that the covariance matrix of the residuals of the partially observed model finds the following representation:

$$\Sigma_o = \mathrm{Var}(\varepsilon_o) = \begin{bmatrix} M_{ou} & N_o \end{bmatrix} V \begin{bmatrix} M'_{ou} \\ N'_o \end{bmatrix} + \Omega_o \tag{2.4}$$

where V is

$$V = \mathrm{Var} \begin{bmatrix} X_{u,t} \\ T_t \end{bmatrix} \Big| X_{o,t} \Big] = \begin{bmatrix} V_u & V_{u,T} \\ V_{T,u} & V_T \end{bmatrix}$$

The adjustment and cointegration matrices are retrieved as follows:

$$\alpha_o = \Sigma_o \Big(M_{ou} V_{uT} + N_o V_T \Big)_\perp \tag{2.5}$$

$$\beta_o' = \big(N_o - M_{ou} M_u^{-1} N_u \big)_\perp' \Big(M_o - M_{ou} M_u^{-1} M_{uo} \Big) \tag{2.6}$$

Starting from representation (2.5), the next subsection discusses some mathematical properties of $\gamma_\perp$ and demonstrates a duality between its restrictions and causal endogeneity. Subsequently, section 2.3.2 elucidates how to exploit this duality to develop a statistical test.

2.3.1 Model Manifestations of Weak Causal Exogeneity

Following the representation in (2.5) and the construction of model (2.3), γ can be expressed as:

$$\gamma = \Big(M_{ou} V_{uT} + N_o V_T \Big)_\perp$$

Therefore $\gamma_\perp$ is simply:

$$\gamma_\perp = \left[\Big(M_{ou} V_{uT} + N_o V_T \Big)_\perp \right]_\perp \tag{2.7}$$

which reduces to

$$\gamma_\perp = M_{ou} V_{uT} + N_o V_T$$

if $M_{ou} V_{uT} + N_o V_T$ has full rank.

M_{ou} is the coefficient of the state-space model relating the observed variables to the ordinary unobserved variables, while N_o relates X_o to the fundamental trends. It is convenient to decompose the set of unobserved ordinary variables into two subsets: $X_u = \{ד, ל\}$. Here, ד includes the ancestors of א, while ל includes the ancestors of ב not contained in ד as per definitions 3 and 4.

By definition, elements in ב cannot be caused by the fundamental trends or by the components of ד. Moreover, by construction, the variables in א cannot be caused by components of ל. Consequently, the coefficients of the state-space model assume the following form:

$$M_{ou} = \begin{matrix} \\ |א| \\ |ב| \end{matrix} \begin{matrix} \begin{matrix} |ד| & |ל| \end{matrix} \\ \begin{bmatrix} \boldsymbol{\mathcal{A}} & \mathbf{0} \\ \mathbf{0} & \boldsymbol{\mathcal{C}} \end{bmatrix} \end{matrix} \qquad N_o = \begin{matrix} \\ |א| \\ |ב| \end{matrix} \begin{matrix} \begin{matrix} |T| \end{matrix} \\ \begin{bmatrix} \boldsymbol{\mathcal{D}} \\ \mathbf{0} \end{bmatrix} \end{matrix} \tag{2.8}$$

As illustrated in Chapter 1, V_{uT} contains null entries in the rows corresponding to ל. To understand the reason, it is instructive to undertake a covariance analysis grounded in the concept of blocked and unblocked paths. The conditional covariance between $\{T, ד\}$ and ל is zero since א blocks the paths among them and only the former points towards א. Consequently, collider-type structures of the form $ל \rightarrow X_o \leftarrow T \cup ד$ are excluded. On the other hand, X_o cannot block the paths between ד and T; thus the sub-matrix corresponding to ד has not particular constraints. The same goes for the covariance paths among components of T. Consequently, the forms of V_{uT} and V_T are as follows:

$$V_{uT} = \begin{matrix} \\ |\daleth| \\ |\gimel| \end{matrix} \begin{matrix} |T| \\ \begin{bmatrix} \boldsymbol{\mathcal{E}} \\ \mathbf{0} \end{bmatrix} \end{matrix} \qquad V_T = |T| \begin{matrix} |T| \\ \begin{bmatrix} \boldsymbol{\mathcal{F}} \end{bmatrix} \end{matrix} \tag{2.9}$$

Before drawing further conclusions, it is necessary to study the properties of the non-zero sub-matrix $\boldsymbol{\mathcal{A}}\boldsymbol{\mathcal{E}} + \boldsymbol{\mathcal{D}}\boldsymbol{\mathcal{F}}$. This matrix can be interpreted as the product of coefficients linking the elements of $\aleph$ to their parents and the covariance between them and the fundamental trends. Given that X_o does not interfere with the fundamental trends and either does not block the paths between $\daleth$ and T or unblock them, each row of $\boldsymbol{\mathcal{E}}$ and $\boldsymbol{\mathcal{F}}$ will contain at least one non-zero entry. Furthermore, every component of $\aleph$ is linked to at least one variable in either $\daleth$ or T. Hence, the matrix $\begin{bmatrix} \boldsymbol{\mathcal{A}} & \boldsymbol{\mathcal{D}} \end{bmatrix}$ contains at least one non-null entry in every row. Consequently, $\boldsymbol{\mathcal{A}}\boldsymbol{\mathcal{E}} + \boldsymbol{\mathcal{D}}\boldsymbol{\mathcal{F}}$ contains at least one non-null entry for each row. This is naturally true since every element in $\aleph$ must be either directly or indirectly connected to T.

Similarly, every element of T must be connected with at least one element of $\aleph$. To suggest otherwise would imply that $\aleph$ and some components of T belong to disjoint graphs, hence to different systems. Consequently, the columns of $\boldsymbol{\mathcal{A}}\boldsymbol{\mathcal{E}} + \boldsymbol{\mathcal{D}}\boldsymbol{\mathcal{F}}$ must also contain at least one non-null entry. The specific entries of the coefficients are independent of each other, thus linear dependence between the rows or columns of $\boldsymbol{\mathcal{A}}\boldsymbol{\mathcal{E}} + \boldsymbol{\mathcal{D}}\boldsymbol{\mathcal{F}}$ is attained only through fine-tuning of the parameters. Here, we find it reasonable to exclude this scenario, thus considering $\boldsymbol{\mathcal{A}}\boldsymbol{\mathcal{E}} + \boldsymbol{\mathcal{D}}\boldsymbol{\mathcal{F}}$ as a full-rank matrix with rank $\min(|\aleph|, |T|)$.

The discussion above leads to the following fundamental conclusion:

Theorem 3 (***Weak Causal Exogeneity and Restrictions of*** $\gamma_\perp$)**.** *The rows of the matrix* $\gamma_\perp$ *corresponding to elements within the set* $\beth$ *are zero vectors.*

Proof of Theorem 3. The theorem can be readily proved by substituting (2.8) and (2.9) into (2.7). The resulting expression for $M_{ou}V_{uT} + N_oV_T$ is

$$M_{ou}V_{uT} + N_oV_T = \begin{matrix} \\ |\aleph| \\ |\beth| \end{matrix} \begin{matrix} |T| \\ \begin{bmatrix} \boldsymbol{\mathcal{AE}} + \boldsymbol{\mathcal{DF}} \\ \mathbf{0} \end{bmatrix} \end{matrix}$$

if $M_{ou}V_{uT} + N_oV_T$ has full rank, it equates $\gamma_\perp$, thus immediately proving the theorem.

If $M_{ou}V_{uT} + N_oV_T$ is of reduced rank, it follows that $\text{Rank}(M_{ou}V_{uT} + N_oV_T) = |\aleph|$ and $|T| > |\aleph|$.[3] Therefore, if $M_{ou}V_{uT} + N_oV_T$ is of reduced rank, its orthogonal complement, i.e. γ, is a $|X_o| \times (|X_o| - |\aleph|) = |\beth|$ matrix of the form

$$\gamma = \begin{matrix} \\ |\aleph| \\ |\beth| \end{matrix} \begin{matrix} |\beth| \\ \begin{bmatrix} \mathbf{0} \\ \boldsymbol{\mathcal{G}} \end{bmatrix} \end{matrix}$$

where $\boldsymbol{\mathcal{G}}$ is of full rank, and the null vectors in the rows corresponding to $\aleph$ result from the fact that $\left(\boldsymbol{\mathcal{AE}} + \boldsymbol{\mathcal{DF}}\right)_\perp$ is a $|\aleph| \times 0$ matrix when its rank equals $|\aleph|$.

Since $\boldsymbol{\mathcal{G}}$ is a full-rank square matrix, its nullity is zero, meaning the only vector that yields zero when multiplied by it is the trivial vector. Therefore, the representation of $\gamma_\perp$ must be

[3] $\boldsymbol{\mathcal{AE}}+\boldsymbol{\mathcal{DF}}$ has rank $|T|$ if $|T| \leq |\aleph|$. Given that $M_{ou}V_{uT}+N_oV_T$ is a $|X_o|\times|T|$ matrix where $|T| < |X_o|$, it would have full rank if $|T| \leq |\aleph|$.

$$\gamma_{\perp} = \begin{matrix} \\ |\aleph| \\ |\beth| \end{matrix} \begin{matrix} |\aleph| \\ \left[\begin{array}{c} \boldsymbol{\mathcal{H}} \\ \mathbf{0} \end{array}\right] \end{matrix} \tag{2.10}$$

where $\boldsymbol{\mathcal{H}}$ is any full rank square matrix of dimension $|\aleph|$

This is sufficient to prove the theorem.

□

Corollary 7 (***Corollary to Theorem 3***)**.** *Every row of $\gamma_{\perp}$ corresponding to the elements in $\aleph$ contains at least one non-null entry.*

Proof of Corollary 7. If $M_{ou}V_{uT} + N_oV_T$ is of reduced rank, $\gamma_{\perp}$ assumes the form in equation (2.10). Since $\gamma_{\perp}$ has full rank by definition, the matrix $\boldsymbol{\mathcal{H}}$ is a full-rank square matrix. It follows that all of its rows contain at least a non-null entry.

To prove the corollary for the case where $M_{ou}V_{uT} + N_oV_T$ is not rank deficient, we must demonstrate that $\boldsymbol{\mathcal{AE}} + \boldsymbol{\mathcal{DF}}$ does not contain null rows.

By definition, every element in $\aleph$ must be directly causally connected with at least one variable in $\daleth \cup T$. Consequently, either $\boldsymbol{\mathcal{A}}_{(i)}$ or $\boldsymbol{\mathcal{D}}_{(i)}$ (or both) must contain one non-null entry, where $A_{(i)}$ denotes the i^{th} row of A.

Without loss of generality, we set that either $\boldsymbol{\mathcal{A}}_{ij} \neq 0$ or $\boldsymbol{\mathcal{D}}_{ij} \neq 0$. Consequently, $\gamma_{(i)} = \mathbf{0}$ if $\boldsymbol{\mathcal{E}}^{(j)} = \mathbf{0}$ or $\boldsymbol{\mathcal{F}}^{(j)} = \mathbf{0}$, where $A^{(i)}$ denotes the i^{th} column of A. Nevertheless, since X_o either does not block or unblock every path between $\daleth_j$ and T, the conditional covariance

between $\top_j$ and at least one component of T must be different from zero, therefore $\boldsymbol{\mathcal{E}}^{(j)}$ cannot be the null vector.

Moreover, X_o does not interfere with the relationships among the components of T, thus the main diagonal of $\boldsymbol{\mathcal{F}}$ contains the variances of T, i.e. non-zero entries.

We can conclude that $\boldsymbol{\mathcal{A}}_{(i)}\boldsymbol{\mathcal{E}} + \boldsymbol{\mathcal{D}}_{(i)}\boldsymbol{\mathcal{F}} = 0$ can occur only for a fine-tuning of the parameters, since the values of the state-space coefficients can freely vary independently of the values of V. One might argue that specific policies, reaction functions, or particular economic choices could lead to fine-tuned parameters. However, even a minor deviation from these coefficients would render the matrix full rank. This implies that a fine-tuned parameter resulting from a policy function can cause rank reduction only if the policy-maker has perfect control over the variable used as the policy instrument. If even the slightest deviation occurs, the parameter would no longer be fine-tuned, and the rank would be restored to full. Therefore, assuming that the coefficients can be finely tuned is an inherently weak assumption. Consequently, there are no compelling reasons to think that this scenario would occur with a probability measure different from zero.

This proves the Corollary

□

To clarify the concept of Theorem 3 and its Corollary, consider the graph in figure 2.2 and

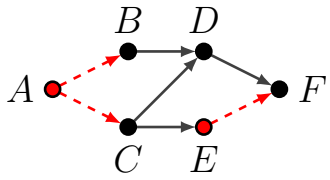

Figure 2.2: Example to Illustrate the Findings of Theorem 3 and its Corollary

the corresponding VECM shown below:

$$\Delta \begin{bmatrix} A \\ B \\ C \\ D \\ E \\ F \end{bmatrix}_t = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 \\ a & 0 & 0 & 0 & 0 \\ 0 & b & 0 & 0 & 0 \\ 0 & 0 & c & 0 & 0 \\ 0 & 0 & 0 & d & 0 \\ 0 & 0 & 0 & 0 & e \end{bmatrix} \begin{bmatrix} f & 1 & 0 & 0 & 0 & 0 \\ g & 0 & 1 & 0 & 0 & 0 \\ 0 & h & i & 1 & 0 & 0 \\ 0 & 0 & j & 0 & 1 & 0 \\ 0 & 0 & 0 & k & l & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \\ D \\ E \\ F \end{bmatrix}_{t-1} + \varepsilon_t$$

Suppose the model user observes only B, C, D, and F, so that $Y = \{A, B, C, D, E, F\}$, $X_o = \{B, C, D, F\}$, $T = \{A\}$, and $X_u = \{E\}$. As observed, the forward path between T and both B and C are direct and thus not blocked by any other variable within X_o. It follows that both B and C belong to $\aleph$. On the other hand, the forward paths between T and D as well as the ones between T and F are indirect and blocked by B and C. Additionally, causality from X_o to D is direct, thus satisfying condition *ii*) of Definition 1. Conversely, causality between C and F is mediated by E. Nevertheless, this does not create any problem since the causal transmission does not reverse, given that E does not, in turn, affect C. It follows that $\beth = \{D, C\}$. Since E is a parent of $F \in \beth$ but is not an

ancestor of the components of א, it will be included in ל in accordance to definition 4. In summary, the causal sets are א $= \{B, C\}$, ב $= \{D, F\}$, ל $= \{E\}$, ך $= \emptyset$, and $T = \{A\}$.

The coefficients M_{ou} and N_o of the state-space representation can be derived from the fully observed VECM. Specifically, in this example, they are:

$$M_{ou} = \begin{bmatrix} 0 \\ 0 \\ 0 \\ el \end{bmatrix} \qquad N_o = \begin{bmatrix} af \\ bg \\ 0 \\ 0 \end{bmatrix}$$

The matrix V is instead a diagonal matrix, given that the covariance paths between A and E are blocked by C. Moreover, the absence of feedback from E to א ensures that no covariance path between A and E is unblocked by X_o. Therefore, the coefficients V_{uT} and V_T assumes the following structures:

$$V_{uT} = \begin{bmatrix} 0 \end{bmatrix} \qquad V_T = \begin{bmatrix} \sigma^2_{T|X_o} \end{bmatrix}$$

By substituting the above matrices into Equation 2.7, we obtain the following representa-

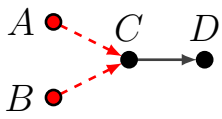

Figure 2.3: Example to Illustrate the Findings of Theorem 3 when $M_{ou}V_{uT}+N_oV_T$ is Rank Deficient

tion:

$$\gamma_\perp = \left(\begin{bmatrix} af\sigma^2_{T|X_o} \\ bg\sigma^2_{T|X_o} \\ 0 \\ 0 \end{bmatrix}_\perp \right)_\perp$$

Since $M_{ou}V_{uT} + N_oV_T$ is a 4×1 matrix of full rank, the two $\perp$ operators simplify. Thus it results:

$$\gamma_\perp = \begin{bmatrix} af\sigma^2_{T|X_o} \\ bg\sigma^2_{T|X_o} \\ 0 \\ 0 \end{bmatrix}$$

As observed, the third and fourth row of $\gamma_\perp$ are null vectors. This is coherent with Theorem 3, as the third and fourth variables are $D \in \beth$ and $F \in \beth$. Conversely, the first two rows are non-zero. This aligns with Corollary 7, as the variables corresponding to the first two rows are B and C, which are the elements of $\aleph$.

It should be noted that the simplification $\left[(M_{ou}V_{uT} + N_oV_T)_\perp\right] = M_{ou}V_{uT} + N_oV_T$ is not always feasible. Specifically, if $M_{ou}V_{uT} + N_oV_T$ has reduced rank, it holds the inequality

$\left[(M_{ou}V_{uT} + N_oV_T)_\perp\right] \neq M_{ou}V_{uT} + N_oV_T$. To illustrate, consider the graph in Figure 2.3 together with its corresponding VECM reported below:

$$\Delta \begin{bmatrix} A \\ B \\ C \\ D \end{bmatrix}_t = \begin{bmatrix} 0 & 0 \\ 0 & 0 \\ a & 0 \\ 0 & b \end{bmatrix} \begin{bmatrix} c & d & 1 & 0 \\ 0 & 0 & e & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \\ D \end{bmatrix}_{t-1} + \varepsilon_t$$

Here, the only unobserved are the fundamental trends A and B, while $X_o = \{C, D\}$. D respects both requirements i) and ii) of definition 1, thus $D \in \beth$. Conversely, C is directly linked to T, so that $C \in \aleph$.

Since $X_u = \emptyset$, the matrices V_{uT} and M_{ou} do not exist, therefore the parameters of interest are:

$$N_o = \begin{bmatrix} ac & ad \\ 0 & 0 \end{bmatrix} \qquad V_T = \begin{bmatrix} \sigma^2_{A|X_o} & \sigma_{AB|X_o} \\ \sigma_{AB|X_o} & \sigma^2_{B|X_o} \end{bmatrix}$$

By substituting the above matrices into Equation 2.7, we obtain the following representation:

$$\gamma_\perp = \left(\begin{bmatrix} ac\sigma^2_{A|X_o} + ad\sigma_{AB|X_o} & ac\sigma_{AB|X_o} + ad\sigma^2_{B|X_o} \\ 0 & 0 \end{bmatrix}_\perp \right)_\perp$$

Since $N_o V_T$ is a 2×2 matrix of reduced rank 1, it cannot be equal to $\gamma_\perp$. This follows from the fact that the orthogonal complement is inherently constructed as a full-rank matrix. Therefore, it is necessary to solve the equation step by step.

The internal orthogonal complement is a 2×1 matrix such that $(M_{ou} V_{uT} + N_o V_T)'_\perp (M_{ou} V_{uT} + N_o V_T) = 0$. The only admissible solution is a matrix with a zero element in the first row and a generic non-zero entry in its second position. It can be immediately observed that by constructing in this way the orthogonal complement, the equality $(M_{ou} V_{uT} + N_o V_T)'_\perp (Mou V_{uT} + N_o V_T) = 0$ holds:

$$\begin{bmatrix} 0 & x \end{bmatrix} \begin{bmatrix} ac\sigma^2_{A|X_o} + ad\sigma_{AB|X_o} & ac\sigma_{AB|X_o} + ad\sigma^2_{B|X_o} \\ 0 & 0 \end{bmatrix} = \mathbf{0}$$

where $x \in (\infty, 0) \cup (0, \infty)$.

To obtain $\gamma_\perp$, it is necessary to reiterate the procedure by computing the orthogonal complement of $\begin{bmatrix} 0 \\ x \end{bmatrix}$. Naturally, the only solution for this problem is a 2×1 matrix containing a null entry in the second row and a generic non-null entry in its first row:

$$\gamma_\perp = \begin{bmatrix} y \\ 0 \end{bmatrix}$$

where $y \in (\infty, 0) \cup (0, \infty)$.

As anticipated, the non-zero row corresponds to $B \in \aleph$, while the null row corresponds to

$D \in \beth$, confirming the duality between causal properties and model constraints identified by Theorem 3 and Corollary 7.

The next subsection demonstrates how to exploit Theorem 3 and its corollary to implement an inferential procedure for identifying the causal endogeneity.

2.3.2 Testing Hypotheses on Causal Endogeneity

Theorem 3 highlights a duality between model characteristics and the concept of causal endogeneity, suggesting that hypotheses regarding the latter can be reformulated as hypotheses concerning model constraints. Specifically, given an arbitrarily selected set Ξ, the causal null hypothesis:

$$\mathbf{H_{0c}}: \textit{The elements in } \Xi \subseteq X_o \textit{ belong to } \beth$$

can be translated as

$$\mathbf{H_{0m}}: \textit{The rows of } \gamma_\perp \textit{ corresponding to the variables in } \Xi \subseteq X_o \textit{ are zero vectors}$$

The estimation of the coefficients of model (2.3) can be undertaken using reduced rank regression technique on model

$$Q_t = \gamma \beta_o' R_{1,t} + \nu_t$$

The estimate of β_o is found as the eigenvectors corresponding to the largest r eigenvalues of the following problem:

$$\det\left(\lambda S_1 - S_{1Q} S_Q^{-1} S_{Q1}\right) = 0$$

where the product matrices S are calculated as follows:

$$S_Q = N^{-1} \sum Q_t Q_t' \qquad S_1 = N^{-1} \sum R_{1t} R_{1t}'$$

$$S_{Q1} = N^{-1} \sum Q_t R_{1t}' \qquad S_{1Q} = N^{-1} \sum R_{1t} Q_t'$$

To test the restrictions of $\gamma_\perp$ we use the tecniques developed in Gonzalo and Granger (1995).In the standard VEC model, there is a strict relation between the estimate of the cointegration matrix and the estimate of the orthogonal complement of the adjustment matrix (see, e.g., Chapter 8 of Johansen (1995)). While one can interpret the cointegration matrix as determining the variables that cointegrates, the orthogonal complement of the adjustment matrix determines the coefficients of the common trends.

Similarly, the coefficient $\gamma_\perp$ is related to β_o and can be estimated by modifying the eigenvector problem employed to retrieve $\hat{\beta}_o$. Thus, extending the work of Gonzalo and Granger (1995) to our scenario, the estimate of $\gamma_\perp$ is found as the eigenvectors corresponding to the smallest $|X_o| - r$ eigenvalues of the problem:

$$\det\left(\lambda S_Q - S_{Q1} S_1^{-1} S_{1Q}\right) = 0 \tag{2.11}$$

Testing restrictions on the rows of $\gamma_\perp$ can be conducted in a manner similar to hypothesis testing on restrictions on β (see for instance Morin (2010)). The typology of restriction allows a straightforward formalization since it concerns every row of $\gamma_\perp$ in the same manner. The most intuitive way of articulating the restriction is through the indirect representation:

$$R'\gamma_\perp = 0$$

R is a $|X_o| \times \big(|X_o| - |\Xi|\big)$ matrix with zero elements in the rows corresponding to Ξ and an identity matrix formed by considering the rows corresponding to $\Upsilon = X_o \setminus \Xi$.

For practical purposes, it is convenient to express the constrained relationship in terms of $H = R_\perp$, where H is a $|X_o| \times |\Upsilon|$ matrix. This alternative depiction allows the direct expression of the restricted matrix as depicted below:

$$\gamma_\perp = H\varphi$$

where φ is a $|\Upsilon| \times |T|$ matrix containing the estimated parameters.

The test statistic compares the smallest $|X_o| - r$ eigenvalues of the unrestricted estimate found from the problem in Equation (2.11), with the smallest $|X_o| - r$ eigenvalues of problem relative to the restricted estimate:

$$\det\big(\lambda_r H' S_Q H - H' S_{Q1} S_1^{-1} S_{1Q} H\big) = 0$$

The LR statistic is computed as

$$N\Big[\ln(1-\lambda_{r,1}) - \ln(1-\lambda_1) + \cdots + \ln(1-\lambda_{r,|X_o|-r}) - \ln(1-\lambda_{|X_o|-r})\Big]$$

Under the null, the statistic is distributed as a $\chi^2(\nu)$, where $\nu = |T| \times |\Xi|$ are the degrees of freedom.

2.4 Simulation

This section analyses the performance of the two tests for causal endogeneity using simulated data. Simulations are conducted on eight distinct causal structures, where the compositions of the causal sets and the presence of strong causal exogeneity differ, in order to understand what the factors negatively influencing the tests' performance are.

The next subsection describes the causal structures and the data-generating process used in the simulations, outlining the main properties of the resulting partially observed models. Subsection 2.4.2 details the simulation process and presents the pseudo-codes used to compute the test statistics. Finally, subsection 2.4.3 presents and discusses the simulation outcomes.

2.4.1 Data Generating Processes

Figure 2.4 illustrates the causal structures employed in the data generating process of the simulated time-series. Red nodes and red dotted edges denote unobserved variables and

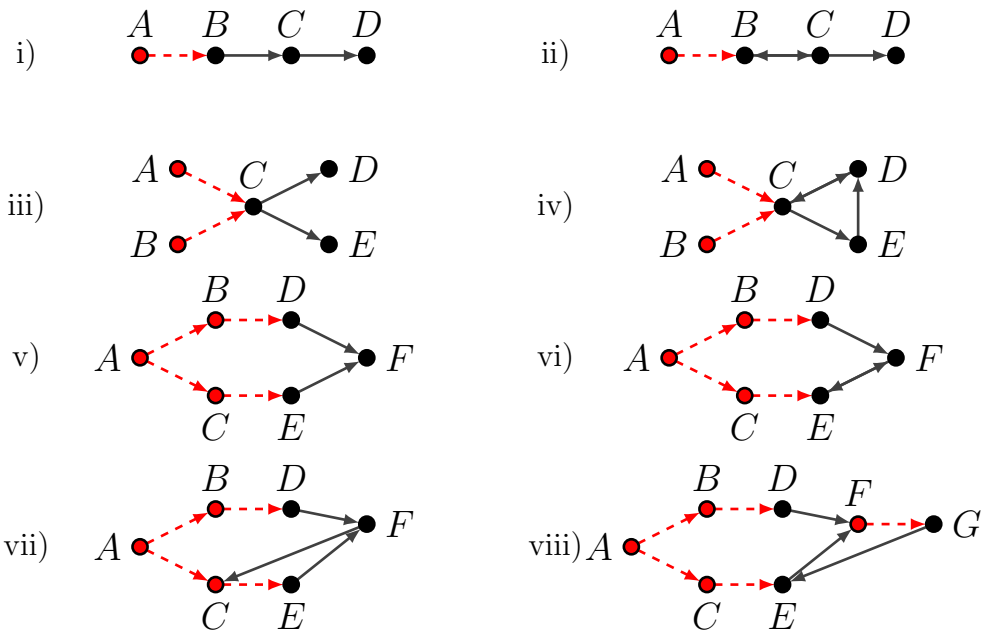

Figure 2.4: Causal Structures for Simulations

latent causal path, respectively, while observed variables are depicted in black.

Structures *i*) and *ii*) are causal chains with four nodes. The fundamental trend is the variable A and it is directly connected to B. Here, B blocks the forward paths between A and all other variables, and it is directly connected to C. Consequently, arbitrary manipulations of B can control the nominal values of both C and D. The set $\aleph$ is thus singleton and contains B only, while $\beth = \{C, D\}$.

Graph *ii*) differs from *i*) for the presence of cyclicality. Since B and C influence each other, and $C \in \beth$, $B \in \aleph$, B is not strongly causally exogenous in graph *ii*).

Graphs i) and ii) can be represented using vector error correction models as follows:

$$\Delta \begin{bmatrix} A \\ B \\ C \\ D \end{bmatrix}_t = \begin{bmatrix} 0 & 0 & 0 \\ a & 0 & 0 \\ 0 & b & 0 \\ 0 & 0 & c \end{bmatrix} \begin{bmatrix} d & 1 & 0 & 0 \\ 0 & e & 1 & 0 \\ 0 & 0 & f & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \\ D \end{bmatrix}_{t-1} + \varepsilon_t \tag{2.12}$$

$$\Delta \begin{bmatrix} A \\ B \\ C \\ D \end{bmatrix}_t = \begin{bmatrix} 0 & 0 & 0 \\ a & 0 & 0 \\ 0 & b & 0 \\ 0 & 0 & c \end{bmatrix} \begin{bmatrix} d & 1 & e & 0 \\ 0 & f & 1 & 0 \\ 0 & 0 & g & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \\ D \end{bmatrix}_{t-1} + \varepsilon_t \tag{2.13}$$

The difference in their representation lies in the first row of β, where the term e in model (2.13) captures the influence of C on B.

Since A is treated as unobserved, the focus is on models that can be estimated by observing only $X_o = \{B, C, D\}$. We consider the synthetic model in Equation (2.3), derived using Johansen's formulae in equations (2.4), (2.5), and (2.6) as previously discussed.

The resulting partially observed model has the following structure for both graphs i) and

ii):

$$\begin{bmatrix} Q_B \\ Q_C \\ Q_D \end{bmatrix}_t = \begin{bmatrix} 0 & 0 \\ \gamma_1 & 0 \\ 0 & \gamma_2 \end{bmatrix} \begin{bmatrix} \beta_1 & \beta_2 & 0 \\ 0 & \beta_3 & \beta_4 \end{bmatrix} \begin{bmatrix} R_{1B} \\ R_{1C} \\ R_{1D} \end{bmatrix}_{t-1} + \varepsilon_t$$

The matrix γ is a rectangular matrix of rank 2 with two linearly independent rows, i.e. the rows corresponding to C and D. Conversely, the row corresponding to B contains only null entries, making it linearly dependent.

The causal endogeneity test based on rank reduction examines whether the rank of $\gamma_{(-i)}$ is less than or equal to 1, indicating a reduction in rank by one unit. $\gamma_{(-i)}$ is equivalent to γ but with the row corresponding to i replaced by a null vector:

$$\gamma_{(-B)} = \begin{bmatrix} 0 & 0 \\ \gamma_1 & 0 \\ 0 & \gamma_2 \end{bmatrix} \qquad \gamma_{(-C)} = \begin{bmatrix} 0 & 0 \\ 0 & 0 \\ 0 & \gamma_2 \end{bmatrix} \qquad \gamma_{(-D)} = \begin{bmatrix} 0 & 0 \\ \gamma_1 & 0 \\ 0 & 0 \end{bmatrix}$$

It is immediately evident that the rank of $\gamma_{(-C)}$ and $\gamma_{(-D)}$ is one, while the rank of $\gamma_{(-B)}$ is two. This aligns with expectations, as we anticipate rejecting the hypothesis $r \leq 1$ when the row corresponding to $B \in \aleph$ is modified, but not rejecting the null when the rows corresponding to $C \in \beth$ and $D \in \beth$ are altered.

The second test for causal endogeneity examines the restrictions on $\gamma_{\perp}$. Given that γ is a

3×2 matrix with rank 2, $\gamma_\perp$ will be a 3×1 matrix with rank 1. Due to the structure of γ, its orthogonal complement can only have null entries in the last two rows. Thus, the only feasible representation is the following:

$$\gamma_\perp = \begin{bmatrix} x \\ 0 \\ 0 \end{bmatrix}$$

where x is any nonzero real number.

Consistent with expectations, the rows of $\gamma_\perp$ corresponding to $\beth$ contain only zero entries. Therefore, we anticipate rejecting the hypothesis that the first row is zero, while we expect not to reject the restrictions for the rows corresponding to C and D.

Structure *iii*) is the union of a collider and a fork, while graph *iv*) maintains the same connections as *iii*) while introducing cyclical behaviours.

The fundamental trends are A and B, both of which cause a single variable, i.e. C. This means that T forms a collider with C. The connection between C and T is direct, thus $C \in \aleph$. Conversely, the forward paths between the two fundamental trends and $\{D, E\}$ are blocked by C and the latter exerts direct causality to $\{D, E\}$. This enables C to control the nominal values of D and E, thus considering them as causally endogenous, i.e., $\aleph = \{C\}$ and $\beth = \{D, E\}$.

Graphs *iii*) and *iv*) are translated into models as follows:

$$\Delta \begin{bmatrix} A \\ B \\ C \\ D \\ E \end{bmatrix}_t = \begin{bmatrix} 0 & 0 & 0 \\ 0 & 0 & 0 \\ a & 0 & 0 \\ 0 & b & 0 \\ 0 & 0 & c \end{bmatrix} \begin{bmatrix} d & e & 1 & 0 & 0 \\ 0 & 0 & f & 1 & 0 \\ 0 & 0 & g & 0 & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \\ D \\ E \end{bmatrix}_{t-1} + \varepsilon_t \tag{2.14}$$

$$\Delta \begin{bmatrix} A \\ B \\ C \\ D \\ E \end{bmatrix}_t = \begin{bmatrix} 0 & 0 & 0 \\ 0 & 0 & 0 \\ a & 0 & 0 \\ 0 & b & 0 \\ 0 & 0 & c \end{bmatrix} \begin{bmatrix} d & e & 1 & f & 0 \\ 0 & 0 & g & 1 & h \\ 0 & 0 & i & 0 & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \\ D \\ E \end{bmatrix}_{t-1} + \varepsilon_t \tag{2.15}$$

The cyclical connections in graph *iv*) are captured by the coefficients f and h of equation (2.15), which introduce in the equilibrium connections affecting C and D, respectively, the variables D and E.

The synthetic model, which can be estimated by an econometrician observing $X_o = \{C, D, E\}$ only, is derived using Johansen's formulae. For graph *iii*), it takes the fol-

lowing architecture:

$$\begin{bmatrix} Q_C \\ Q_D \\ Q_E \end{bmatrix}_t = \begin{bmatrix} 0 & 0 \\ \gamma_1 & 0 \\ 0 & \gamma_2 \end{bmatrix} \begin{bmatrix} \beta_1 & \beta_2 & 0 \\ \beta_3 & 0 & \beta_4 \end{bmatrix} \begin{bmatrix} R_{1C} \\ R_{1D} \\ R_{1E} \end{bmatrix}_{t-1} + \varepsilon_t$$

while for graph iv), it is

$$\begin{bmatrix} Q_C \\ Q_D \\ Q_E \end{bmatrix}_t = \begin{bmatrix} 0 & 0 \\ \gamma_1 & 0 \\ 0 & \gamma_2 \end{bmatrix} \begin{bmatrix} \beta_1 & \beta_2 & \beta_3 \\ \beta_4 & 0 & \beta_5 \end{bmatrix} \begin{bmatrix} R_{1C} \\ R_{1D} \\ R_{1E} \end{bmatrix}_{t-1} + \varepsilon_t$$

The cyclical behaviour in graph iv) results in a slight modification of the coefficient β_o. Specifically, the influence of E on D introduces a nonzero entry in position $\{3, 1\}$.

The matrix γ is a rectangular matrix of rank 2. Therefore, we anticipate that substituting the rows corresponding to D and E with null vectors, the rank reduces to one, while substituting the row corresponding to C, the rank remains stable at 2. This can be readily observed after computing $\gamma_{(-C)}$, $\gamma_{(-D)}$, and $\gamma_{(-E)}$:

$$\gamma_{(-C)} = \begin{bmatrix} 0 & 0 \\ \gamma_1 & 0 \\ 0 & \gamma_2 \end{bmatrix} \qquad \gamma_{(-D)} = \begin{bmatrix} 0 & 0 \\ 0 & 0 \\ 0 & \gamma_2 \end{bmatrix} \qquad \gamma_{(-E)} = \begin{bmatrix} 0 & 0 \\ \gamma_1 & 0 \\ 0 & 0 \end{bmatrix}$$

It is expected a rejection of the hypothesis $r \leq 1$ for the model associated with $\gamma_{(-C)}$, while a failure to reject is anticipated for the models associated with $\gamma_{(-D)}$ and $\gamma_{(-E)}$.

Under this scenario, the matrix $\gamma_{\perp}$ does not coincide with

$$M_{ou}V_{uT} + N_oV_T = \begin{bmatrix} y & z \\ 0 & 0 \\ 0 & 0 \end{bmatrix}$$

In this case, $M_{ou}V_{uT} + N_oV_T$ has a reduced rank, which is incompatible with $\gamma_{\perp}$, since by construction the orthogonal complement needs to have full rank. Therefore, matrix $\gamma_{\perp}$ must be obtained from $\gamma = \begin{bmatrix} 0 & 0 \\ \gamma_1 & 0 \\ 0 & \gamma_2 \end{bmatrix}$. $\gamma_{\perp}$ is a 3×1 matrix of rank 1. Consequently, to ensure $\gamma'\gamma_{\perp} = 0$, its last two entries must be zero, while the first can be any nonzero number.

The resulting structure is

$$\gamma_{\perp} = \begin{bmatrix} x \\ 0 \\ 0 \end{bmatrix}$$

which aligns with our expectations. Specifically, we expect to reject the null hypothesis $\gamma_{\perp(C)} = 0$, while we anticipate a failure to reject the hypotheses $\gamma_{\perp(D)} = 0$ and $\gamma_{\perp(E)} = 0$.

Graphs *v*)-*viii*) exhibit a collider structure among the observed components, while the

variables introducing non-stationarity into the systems form a fork with the fundamental trends.

In all four graphs, the fundamental trend is A, and the forward paths between T and $\{D, E\}$ are not blocked by the other components in X_o. It follows that $\{D, E\} \in$ א in all four graphs. Conversely, D and E block the forward paths between A and F in graphs v)-vii), as well as the forward paths between A and G in graph $viii$). While D and E in $v) - vii$) directly cause the other components of X_o, the transmission of causality between $\{D, E\}$ and G in $viii$) passes through the unobserved F. However, this does not affect the endogeneiety property of G since it does not in turn affects D and E. Consequently, א $= \{D, E\}$ in all the four graphs, while ב $= \{F\}$ in $v) - vii$) and ב $= \{G\}$ in $viii$)

The distinction among the four graphs lies in the presence of cyclicality and the way it is transmitted. In graph v), there is no causal feedback between א and ב, while in graphs vi) and vii), only D is strongly causally exogenous. Specifically, E is directly caused by ב $= \{F\}$ in graph vi) and indirectly through its effect on C in graph vii). This implies that in the latter scenario, causality from ב to א passes through ד.

Similarly to the previous graphs, in $viii$) only D is strongly causally exogenous because of the feedback that G has on E. The distinction between this and the other cases stands on the indirect transmission of causality between א and ב. Consequently, the set ל is not empty and intermediate the connection between א and ב.

The VECM corresponding to v) is

$$\Delta \begin{bmatrix} A \\ B \\ C \\ D \\ E \\ F \end{bmatrix}_t = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 \\ a & 0 & 0 & 0 & 0 \\ 0 & b & 0 & 0 & 0 \\ 0 & 0 & c & 0 & 0 \\ 0 & 0 & 0 & d & 0 \\ 0 & 0 & 0 & 0 & e \end{bmatrix} \begin{bmatrix} f & 1 & 0 & 0 & 0 & 0 \\ g & 0 & 1 & 0 & 0 & 0 \\ 0 & h & 0 & 1 & 0 & 0 \\ 0 & 0 & i & 0 & 1 & 0 \\ 0 & 0 & 0 & j & k & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \\ D \\ E \\ F \end{bmatrix}_{t-1} + \varepsilon_t \tag{2.16}$$

To add causality from F to E, it is necessary to include a coefficient in the column corresponding to F in the equilibrium relation affecting the dynamic of E. Thus, the VECM for vi) is:

$$\Delta \begin{bmatrix} A \\ B \\ C \\ D \\ E \\ F \end{bmatrix}_t = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 \\ a & 0 & 0 & 0 & 0 \\ 0 & b & 0 & 0 & 0 \\ 0 & 0 & c & 0 & 0 \\ 0 & 0 & 0 & d & 0 \\ 0 & 0 & 0 & 0 & e \end{bmatrix} \begin{bmatrix} f & 1 & 0 & 0 & 0 & 0 \\ g & 0 & 1 & 0 & 0 & 0 \\ 0 & h & 0 & 1 & 0 & 0 \\ 0 & 0 & i & 0 & 1 & j \\ 0 & 0 & 0 & k & l & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \\ D \\ E \\ F \end{bmatrix}_{t-1} + \varepsilon_t \tag{2.17}$$

Similarly, in graph vii), cyclicality is added by including F in the equilibrium affecting C:

$$\Delta \begin{bmatrix} A \\ B \\ C \\ D \\ E \\ F \end{bmatrix}_t = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 \\ a & 0 & 0 & 0 & 0 \\ 0 & b & 0 & 0 & 0 \\ 0 & 0 & c & 0 & 0 \\ 0 & 0 & 0 & d & 0 \\ 0 & 0 & 0 & e & \end{bmatrix} \begin{bmatrix} f & 1 & 0 & 0 & 0 & 0 \\ g & 0 & 1 & 0 & 0 & h \\ 0 & i & 0 & 1 & 0 & 0 \\ 0 & 0 & j & 0 & 1 & 0 \\ 0 & 0 & 0 & k & l & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \\ D \\ E \\ F \end{bmatrix}_{t-1} + \varepsilon_t \tag{2.18}$$

Graph *viii*) adds variable G, and the resulting model representation is as follows:

$$\Delta \begin{bmatrix} A \\ B \\ C \\ D \\ E \\ F \\ G \end{bmatrix}_t = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 & 0 \\ a & 0 & 0 & 0 & 0 & 0 \\ 0 & b & 0 & 0 & 0 & 0 \\ 0 & 0 & c & 0 & 0 & 0 \\ 0 & 0 & 0 & d & 0 & 0 \\ 0 & 0 & 0 & 0 & e & 0 \\ 0 & 0 & 0 & 0 & 0 & f \end{bmatrix} \begin{bmatrix} g & 1 & 0 & 0 & 0 & 0 & 0 \\ h & 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & i & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & j & 0 & 1 & 0 & k \\ 0 & 0 & 0 & l & m & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & n & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \\ D \\ E \\ F \\ G \end{bmatrix}_{t-1} + \varepsilon_t \tag{2.19}$$

The synthetic model that can be estimated using X_o is:

$$\begin{bmatrix} Q_D \\ Q_E \\ Q_F \end{bmatrix}_t = \begin{bmatrix} \gamma_1 & 0 \\ \gamma_2 & 0 \\ 0 & \gamma_3 \end{bmatrix} \begin{bmatrix} \beta_1 & \beta_2 & 0 \\ \beta_3 & \beta_4 & \beta_5 \end{bmatrix} \begin{bmatrix} R_{1D} \\ R_{1E} \\ R_{1F} \end{bmatrix}_{t-1} + \varepsilon_t$$

for $v) - vi)$, while it is

$$\begin{bmatrix} Q_D \\ Q_E \\ Q_F \end{bmatrix}_t = \begin{bmatrix} \gamma_1 & 0 \\ \gamma_2 & 0 \\ 0 & \gamma_3 \end{bmatrix} \begin{bmatrix} \beta_1 & \beta_2 & \beta_3 \\ \beta_4 & \beta_5 & \beta_6 \end{bmatrix} \begin{bmatrix} R_{1D} \\ R_{1E} \\ R_{1F} \end{bmatrix}_{t-1} + \varepsilon_t$$

for graphs $vii) - viii)$.

In contrast to the previous scenarios, the linearly dependent rows of γ do not coincide with the null rows. In this case, the linear dependence between the first and second rows arises from the non-zero entries in the same column. Upon substituting the rows of γ with zeros, we obtain:

$$\gamma_{(-D)} = \begin{bmatrix} 0 & 0 \\ \gamma_2 & 0 \\ 0 & \gamma_3 \end{bmatrix} \qquad \gamma_{(-E)} = \begin{bmatrix} \gamma_1 & 0 \\ 0 & 0 \\ 0 & \gamma_3 \end{bmatrix} \qquad \gamma_{(-F)} = \begin{bmatrix} \gamma_1 & 0 \\ \gamma_2 & 0 \\ 0 & 0 \end{bmatrix}$$

where $\gamma_{(-F)}$ becomes $\gamma_{(-G)}$ in graph $viii)$

Consistent with expectations, the first two matrices have a rank of 2, while the latter is rank-deficient. Therefore, we anticipate rejecting the null $r \leq 1$ only when the substitution concerns the variables in $\aleph$.

To ensure that $\gamma'_{\perp}\gamma = 0$, the orthogonal complement cannot have a non-null entry in its

last row. Therefore, its representation is:

$$\gamma_{\perp} = \begin{bmatrix} x(-\gamma_2) \\ x(\gamma_1) \\ 0 \end{bmatrix}$$

where x is any non-null real number.

As expected, we anticipate rejecting the restriction only for the rows corresponding to א, while we expect a failure to reject the constraint for the row corresponding to ב.

2.4.2 Time-Series Simulation and Testing Procedures

The time-series are simulated according to the VECM specified in Equations (2.12)-(2.19). The individual coefficients, denoted by small letters, are randomly drawn from a normal distribution. The drawing process continues until the stability condition is satisfied, i.e., the eigenvalues of the rows and columns of $\Pi = \alpha\beta'$ corresponding to the ordinary variables are less than one in absolute value.

The decision not to select specific parameter values is based on the premise that discussing local alternatives is meaningless when addressing causal hypotheses. In fact, the focus is not on the specific values of the coefficients, but on the causal properties themselves. Causality exists independently of the absolute value of the causal coefficient relating two variables. Thus, there is no distinction in causality produced by causal relations of different coefficient magnitudes.

Moreover, in Test 2, it is not feasible to set a proper local alternative starting from the values of the coefficients in the partially observed model. Test 2 addresses the orthogonal complement of the model coefficients, which has an infinite number of possible representations. For instance, if the coefficient γ derived from the coefficients of a vector error correction model is

$$\gamma = \begin{bmatrix} 0.5 \\ \\ 0 \end{bmatrix}$$

its orthogonal complement is

$$\gamma_{\perp} = \begin{bmatrix} 0 \\ \\ x \end{bmatrix}$$

where x can assume any non-zero value. This means that selecting either $x = 10^{10}$ or $x = 10^{-10}$ still leads to a correct specification, making the search for a local alternative elusive. A specific estimator will naturally pick one of the infinitely possible specifications of $\gamma_{\perp}$. However, to the best of our knowledge, it is not possible to know *a priori* which exact specification will be chosen by the standard maximum-likelihood estimator. Therefore, even if one wants to assign a strength to causal relations, this inherent uncertainty complicates the process of setting a local alternative for Test 2.

The observations are generated starting from a vector of zeros as initial values for all the variables. The coefficients are randomly drawn until they meet the stability condition. Each simulated series consists of 1,000 observations and each scenario is simulated 1,000 times. Algorithm 1 shows an example of the code used to generate the observations for

graph i), corresponding to the VECM in Equation (2.12):

Algorithm 1 Algorithm for Data Simulation

▷ Generate random α and β

$\alpha \leftarrow \text{zeros}(4,3)$
$\beta \leftarrow \text{zeros}(4,3)$
$\alpha(2,1) \leftarrow \text{randn}, \quad \alpha(3,2) \leftarrow \text{randn}, \quad \alpha(4,3) \leftarrow \text{randn}$
$\beta(1,1) \leftarrow \text{randn}, \quad \beta(2,2) \leftarrow \text{randn}, \quad \beta(3,3) \leftarrow \text{randn}$
$\beta(2,1) \leftarrow 1, \quad \beta(3,2) \leftarrow 1 \quad \beta(4,3) \leftarrow 1$

▷ Generate impact matrix

$\Pi \leftarrow \alpha\beta'$

▷ Continue generating adjustment and cointegration matrices as long as the stability condition is not met i.e. until all the eigenvalues of the matrix resulting from summing the identity matrix and the sub-matrix of Π constructed by considering rows and columns relative to the ordinary variables ($\Pi(2:\text{end}, 2:\text{end})$ in this specific example) lie inside the unit circle

while $\text{sum}(\text{abs}(\text{eig}(\text{I} + \Pi(2:\text{end}, 2:\text{end}))) > 1) \neq 0$ **do**
    $\alpha(2,1) \leftarrow \text{randn}, \quad \alpha(3,2) \leftarrow \text{randn}, \quad \alpha(4,3) \leftarrow \text{randn}$
    $\beta(1,1) \leftarrow \text{randn}, \quad \beta(2,2) \leftarrow \text{randn}, \quad \beta(3,3) \leftarrow \text{randn}$
    $\Pi \leftarrow \alpha\beta'$
end while

▷ Create series of shocks

$\varepsilon \leftarrow \text{randn}(N, 4)$

▷ Generate the observations

$Y \leftarrow \text{zeros}(N, 4)$
for $i \leftarrow 1 : (N-1)$ **do**
    $\Delta Y \leftarrow \alpha\beta' Y(i,:)' + \varepsilon(i,:)'$
    $Y(i+1,:) \leftarrow Y(i,:) + \Delta Y'$
end for

▷ Delete the first variable to create X_o

$X_o \leftarrow Y(:, [2,3,4])$

The testing procedure follows the steps described in Chapter 1 for the test based on rank reduction and the steps outlined in section 2.3 for the test based on the restriction of $\gamma_\perp$.

Algorithms 2 and 3 provide the pseudo-code for the computation of the test statistics. The commands are written in a generic form but specific functions such as the computation of eigenvalues track MATLAB syntax.

Algorithm 2 Rank Reduction Test for Causal Endogeneity

▷ Wipe out the short-term dependencies

$Z_0 \leftarrow \Delta X_o(k+1:\text{end})$
$Z_1 \leftarrow X_o(k:\text{end}-1)$
$Z_2 \leftarrow [\Delta X_o(k:\text{end}-1), \ldots, X_o(1:\text{end}-k-1)]$
$M_{02} \leftarrow (Z0'Z2)/N$
$M_{22} \leftarrow (Z2'Z2)/N$
$M_{12} \leftarrow (Z1'Z2)/N$
$R_0 \leftarrow Z_0 - Z_2(M_{02}M_{22}^{-1})'$
$R_1 \leftarrow Z_1 - Z_2(M_{12}M_{22}^{-1})'$

▷ Estimate Σ_o, Q and $Q_{(-\Xi)}$

$\Sigma_o \leftarrow \text{estimate}(\text{vecm}, X_o).\text{Covariance}$
$Q \leftarrow R_0\Sigma_o^{-1}$
$Q(:,\Xi) \leftarrow \text{randn}$

▷ Compute the product matrices

$S_{00} \leftarrow (Q'Q)/N$
$S_{01} \leftarrow (Q'R_1)/N$
$S_{10} \leftarrow (R_1'Q)/N$
$S_{11} \leftarrow (R_1'R_1)/N$

▷ Solve eigenvalue problem and sort the eigenvalues in descending order

$\lambda \leftarrow \text{eig}(S_{10}S_{00}^{-1}S_{01}, S_{11})$
$\lambda \leftarrow \text{sort}(\lambda)$

▷ Compute the statistic

Statistic $\leftarrow 0$
for $i \leftarrow (r+1-|\Xi|) : |X_o|$ **do**
    Statistic $\leftarrow$ Statistic $+ \ln(1-\lambda(i))$
end for

Statistic $\leftarrow -N \times$ Statistic

Algorithm 3 Test on the Restrictions of $\gamma_\perp$ for Causal Endogeneity Hypotheses

▷ Wipe out the short-term dependencies

$Z_0 \leftarrow \Delta X_o(k+1 : \text{end})$
$Z_1 \leftarrow X_o(k : \text{end} - 1)$
$Z_2 \leftarrow [\Delta X_o(k : \text{end} - 1), \ldots, X_o(1 : \text{end} - k - 1)]$
$M_{02} \leftarrow (Z0'Z2)/N$
$M_{22} \leftarrow (Z2'Z2)/N$
$M_{12} \leftarrow (Z1'Z2)/N$
$R_0 \leftarrow Z_0 - Z_2(M_{02}M_{22}^{-1})'$
$R_1 \leftarrow Z_1 - Z_2(M_{12}M_{22}^{-1})'$

▷ Estimate Σ_o and Q

$\Sigma_o \leftarrow \text{estimate}(\text{vecm}, X_o).\text{Covariance}$
$Q \leftarrow R_0\Sigma_o^{-1}$

▷ Compute matrix H

$\Upsilon \leftarrow X_o \setminus \Xi$
$H \leftarrow \text{zeros}(|X_o|, |\Upsilon|)$
$H(\Upsilon, :) \leftarrow \text{I}$

▷ Compute the product matrices

$S_{00} \leftarrow (Q'Q)/N$
$S_{01} \leftarrow (Q'R_1)/N$
$S_{10} \leftarrow (R_1'Q)/N$
$S_{11} \leftarrow (R_1'R_1)/N$

▷ Solve restricted and unrestricted eigenvalue problems and sort the eigenvalues in ascending order

$\lambda \leftarrow \text{eig}(S_{01}S_{11}^{-1}S_{10}, S_{00})$
$\lambda_r \leftarrow \text{eig}(H'S_{01}S_{11}^{-1}S_{10}H, H'S_{00}H)$
$\lambda \leftarrow \text{sort}(\lambda)$
$\lambda_r \leftarrow \text{sort}(\lambda)$

▷ Compute the statistic

Statistic $\leftarrow 0$
for $i \leftarrow 1 : (|X_o| - r)$ **do**
  Statistic $\leftarrow$ Statistic $+ \ln(1 - \lambda_r(i)) - \ln(1 - \lambda(i))$
end for

Statistic $\leftarrow -N \times$ Statistic

Graph	Test 1			Test 2		
	B	C	D	B	C	D
$i)$	99.8%	3%	3.2%	99.9%	6.9%	7.8%
$ii)$	99.2%	6.1%	6.2%	99.5%	7.6%	6.7%
	C	D	E	C	D	E
$iii)$	99.7%	1.6%	2.1%	100%	5.7%	7%
$iv)$	99%	5.7%	4.2%	99.7%	6.9%	9.1%
	D	E	F	D	E	F
$v)$	86.8%	85.3%	2.1%	91.1%	89.8%	7.8%
$vi)$	87.1%	82.1%	2.3%	91.1%	86.9%	7.5%
$vii)$	90.9%	66%	2.4%	93.3%	73.3%	6.8%
	D	E	G	D	E	G
$viii)$	91.3%	82.3%	2.3%	93.9%	87.2%	8%

Table 2.1: Rejection Rate for Rank Test (Test 1) and for Test on Restrictions of $\gamma_\perp$ (Test 2). Significance Level 5%

2.4.3 Simulation outcome

This subsection presents the outcomes of the simulations. The importance of evaluating performance hinges on two key aspects: the first one is related to the study of the test behaviour under the alternative hypothesis. The second aspect is relevant in the specific case of the hypothesis testing on causal endogeneity. In fact, in both tests, the left-hand side of the model is not directly observable but is estimated by multiplying the inverse of $\hat{\Sigma}_o$ with $R_{0,t}$. This affects the distribution of the test statistic in unknown ways, potentially leading to results that do not perfectly align with expected rejection rates.

Table 2.1 records the simulation outcomes, where percentages indicate rejection rates over 1,000 simulated scenarios at a 5% significance level. Test 1 refers to the causal endogeneity test based on rank reduction, while Test 2 pertains to the causal endogeneity test based on restrictions of $\gamma_\perp$.

The table indicates satisfactory results, with rejection rates significantly different between tests applied to elements of $\aleph$ and $\beth$. As anticipated, the size does not align perfectly with the theoretical 5% value, likely due to discrepancies between the distribution under the null of the standard test and the actual distribution generated by the substitution of Q with $\hat{Q}$. Nevertheless, the results are reasonable, with an average rejection rate under the null of 3.77% for Test 1 and 7.73% for Test 2. The power of both tests also appears reasonable, with an average value of 87.75% for Test 1 and 91.17% for Test 2.

Neither test consistently outperforms the other overall; however, Test 1 yields better results under the null, while Test 2 exhibits higher power. This is evidenced by consistently higher rejection rates for Test 2 respect to Test 1. This indicates that the former performs better when the null is false and worse when the null is true.

Graph structure significantly impacts test performance. Results are highly satisfactory for graphs $i) - iv)$, with the worst empirical power values being 99% and 99.5% for tests 1 and 2, respectively. However, the accuracy heavily decreases for graphs $v) - viii)$, particularly in terms of power, reaching a minimum in graph $vii)$ for the test on E, with values of 66% and 73.3% for tests 1 and 2, respectively. The difference between the two groups of graphs stands on the presence of $\gimel$. In fact, in graphs $i) - iv)$, X_o is directly connected to the fundamental trends, while in graphs $v) - viii)$, the connection between T and $\aleph$ passes through $\gimel = \{B, C\}$.

Another factor that is observed to affect the test accuracy is the presence of cyclicality. Comparing the outcomes of graphs $i)$, $iii)$, and $v)$ with those of graphs $ii)$, $iv)$, and

$vi) - viii)$, it is evident that cyclicality negatively impacts causal endogeneity tests. For example, in graphs $vi) - vii)$, where cyclicality involves only variable E, there is a significant difference in rejection rates between D and E. It is also evident that an indirect feedback between ב and א more heavily affects the test results compared to a direct feedback. In fact, the gap in test power between D and E is around 5% in graph $v)$, increasing to approximately 20% in graph $vi)$.

To understand the impact of sample size, we performed simulations for two smaller sample sizes, specifically $N = 100$ and $N = 500$. Table 2.2 reports the outcomes of the first test under the null hypothesis. The simulations do not exhibit any consistent patterns in rejection rates across different sample sizes. The rejection rates appear to fluctuate around 5%, with no significant difference between $N = 100$ and $N = 1,000$. Notably, there seems to be a tendency for the rejection rate to be lower than the significance level, even for the smallest sample size. The reason behind this phenomenon remains unclear and requires further investigation.

Table 2.3 reports the outcomes under the null hypothesis for the second test on causal endogeneity. Unlike the first test, the table shows a significant improvement in performance when the sample size is increased from 100 to 500, with the rejection rate approaching the 5% significance level. Notably, this improvement is evident when increasing the sample size from 100 to 500 observations. However, there is no significant impact when the number of observations is doubled from 500 to 1,000.

Table 2.4 reports the rejection rates when the null hypothesis is false for the first test on

Graph N=1,000	Variable	N=100	N=500
i)	C	3.17%	4.4%
i)	D	2.9%	1.6%
ii)	C	2.54%	6.9%
ii)	D	2.86%	6.9%
iii)	D	1.9%	1%
iii)	E	4.8%	1%
iv)	D	1.3%	4.1%
iv)	E	0.9%	2.9%
v)	F	1.9%	2.9%
vi)	F	4.8%	2.9%
vii)	F	3.5%	4.1%
viii)	G	2.9%	1.3%

Table 2.2: Rejection Rate for Rank Test (Test 1) Under the Null Hypothesis for Different Sample Sizes. Significance Level 5%

Graph	Variable	N=100	N=500
i)	C	15.9%	7.6%
i)	D	11.8%	7.6%
ii)	C	13.3%	8.6%
ii)	D	10.5%	8.5%
iii)	D	12.4%	5.7%
iii)	E	9.2%	9.2%
iv)	D	11.8%	7.6%
iv)	E	8.9%	7.3%
v)	F	8.7%	6.8%
vi)	F	11.4%	7.9%
vii)	F	14.9%	7.3%
viii)	F	9.2%	9.2%

Table 2.3: Rejection Rate for Orthogonal Complement's Restrictions Test (Test 2) Under the Null Hypothesis for Different Sample Sizes. Significance Level 5%

Graph	Variable	N=100	N=500
i)	B	47%	99.1%
ii)	B	45.7%	98.4%
iii)	C	51,1%	100%
iv)	C	44.4%	98.4%
v)	D	24.8%	79.1%
v)	E	23.8%	80%
vi)	D	36.8%	84.1%
vi)	E	31.1%	81%
vii)	D	44.1%	86%
vii)	E	21%	60%
viii)	D	39.4%	86%
viii)	E	32.1%	77.5%

Table 2.4: Rejection Rate for Rank Test (Test 1) Under the Alternative Hypothesis for Different Sample Sizes. Significance Level 5%

causal endogeneity. As anticipated, the rejection rate increases significantly as the sample size grows. The table indicates that the test based on the cointegration rank performs poorly in terms of power when the sample size is 100. Notably, the empirical power exceeds 50% only once, with a minimum value of 21%.

Table 2.5 records the test outcomes when the alternative hypothesis is true. As anticipated, the power increases as the number of observations grows. However, unlike the first test, the reported outcomes show decent power rates even for the smallest sample size. Specifically, for $N = 100$ the empirical power fluctuates around 70%, reaching a maximum of 93%.

To summarise, the simulations demonstrated that the tests generally perform satisfactorily. Test 1 is more effective when the null hypothesis is true, while Test 2 exhibits higher power. The accuracy of the tests is influenced by the graph structure and the presence of cyclicality. Specifically, outcomes deviate slightly from expectations when ך is non-empty

Graph	Variable	N=100	N=500
i)	B	92.4%	99.4%
ii)	B	88.9%	99.4%
iii)	C	93%	100%
iv)	C	90.5%	99.1%
v)	D	65.7%	89.2%
v)	E	66.4%	85.4%
vi)	D	68.9%	88.3%
vi)	E	69.2%	87.3%
vii)	D	77.5%	89.8%
vii)	E	51.4%	70.5%
viii)	D	75.9%	89.8%
viii)	E	63.8%	83.8%

Table 2.5: Rejection Rate for Orthogonal Complement's Restrictions Test (Test 2) Under the Alternative Hypothesis for Different Sample Sizes. Significance Level 5%

or when ב causes א, in particular when this causal feedback is indirect. Furthermore, Test 2 demonstrates satisfactory performance for small sample sizes, whereas the rejection rates under the alternative hypothesis for Test 1 are particularly low when the number of observations is limited. This suggests that Test 2 should be preferred when the analysis involves small samples.

2.5 Empirical Illustration

This section offers an empirical illustration of the methodologies previously discussed by testing the causal endogeneity properties of supply and demand of money. It is important to interpret the results with caution due to limited emphasis on specification issues. Additionally, given the preliminary nature of this research, certain considerations, such as the presence of deterministic trends within the cointegration relation, were not fully

addressed in the theoretical framework. Consequently, we will not use the raw data; instead, the observations will be detrended and normalized. This preprocessing of variables may result in a loss of information crucial for analyzing the endogeneity of money. Hence, these findings should be viewed as illustrative, with the primary aim of demonstrating the practical implementation of the developed tests.

This section is structured as follows: next subsection discusses the theories advocating for money exogeneity and endogeneity. Subsection 2.5.2 describes the data, while Subsection 2.5.3 reports the results of the empirical illustration.

2.5.1 Causal Endogeneity and Money Theories

Understanding whether money is endogenous or exogenous entails comprehending the causal relationships between money supply and money demand. Exogeneity implies that the supply of money is causally independent of demand, while endogeneity implies the opposite. Monetarists have traditionally supported the exogeneity view, whereas horizontalists and post-Keynesian economists see money supply as inherently endogenous. The structuralist school acknowledges a degree of endogeneity while recognizing some control over narrow money aggregates.

Since money demand is challenging to measure directly, studying the nature of money supply requires examining its relation with the driving forces behind money demand. It is widely accepted that money demand is influenced by monetary variables like interest rates and inflation, as well as real factors such as economic output and in general economic

activity. If the money supply is exogenous, it remains unaffected by the determinants of money demand, and vice versa if the money supply is endogenous.

The monetarist view is the most extreme version of money exogeneity. Monetarists consider the LM curve in the IS/LM model as vertical, with the money supply fixed and unresponsive to money demand. According to this view, monetary authorities have full control over monetary aggregates, modifying the monetary base at will. Obviously, the central bank does not directly control the total money supply, but monetarists believe that the central banks fully control the monetary base and that there exists a strong, stable link between it and broader monetary aggregates.

On the other extreme, horizontalists deny the central banks' ability to control the monetary base. They assert that banks grant loans based on demand, and central banks accommodate the demand for reserves to preserve financial system stability. In other words, the horizontalists claim that money is endogenous, because central banks willingly and deliberately supply the reserves on demand. The money supply curve is thus horizontal, as the suppliers of money always fully accommodate the demand for money at a given interest rate.

The monetarist school posits that money generation stems primarily from the decisions of monetary authorities, while the horizontalist school considers the level of even narrower money aggregates as a result of demand forces.

To adapt these theories to the framework of causal endogeneity, it is necessary to speak in terms of causality. Lavoie (1984) summarised the monetarist and post-Keynesian (hor-

izontalist) perspectives with a stylized system of structural equations, where causality is read from right to left. Denote M as the money supply and B as the monetary base. the monetarist viewpoint, which, for causality purposes, is similar to the view endorsed by the great majority of economists, is represented by the structural equation $M = \varphi B$, where φ is a causal coefficient. On the other hand, the post-Keynesian view can be summarised by the equation $B = \theta M$. Naturally, despite the quantity of money in the second view is considered as the independent variable, the value of M cannot be considered the primary source of causality. In fact, money supply accommodates money demand, which is in turn influenced by economic factors such as interest rates, income and prices. Thus, the complete mathematical description of the post-Keynesian view should be summarised with the following system of equations:

$$\begin{cases} B = \theta M \\ \\ M = f(Y, i, P) \end{cases}$$

where Y, i, P represent income, interest rate and prices respectively.

A third perspective, the structuralist view, lies midway between these extremes. Structuralists, like monetarists, acknowledge the influence of monetary authorities on the monetary base but question the close link between the base and broader money aggregates. They argue that commercial banks can choose to hold excess reserves and that the private non-bank sector can alter its preference for cash, making the monetary multiplier unstable. The structuralists thus accept an upward-sloping money supply curve, presenting a view

between the horizontal and vertical LM curves.

Structuralists are generally considered proponents of an endogenous view of money. The distinction with the horizontalists lies in the structure and type of exogeneity. Horizontalists claim that money is endogenous, because central banks willingly and deliberately supply the reserves on demand. The money supply curve is thus horizontal, as the suppliers of money always fully accommodate the demand for money at a given interest rate. Meanwhile, the structuralists argue that money is endogenous, because—thanks to innovative techniques of management of assets and liabilities—commercial banks can lend largely free of any central bank constraint (Howells (2006)).[4]

In causal terms, structuralists recognize the structural equation $M = f(Y, i, P)$, while also acknowledging the control that monetary authorities exert over the monetary base, which in turn affects M. Hence, structuralists include B and indirectly the choices of monetary authorities among the determinants of the money supply.

Understanding the primary source of causality implied by these theories is complex. From an economic perspective, monetarists treat the monetary base B as the variable introducing non-stationarity in the economic dynamics. However, the monetary base cannot be considered as the real fundamental trend. According to this view, B is modified at will by policymakers, making the true original source of causality the decisions of monetary authorities. Likewise, horizontalists consider the driving force to be money demand and its determinants. Nevertheless, they cannot be considered the fundamental trends since

[4]For a thorough discussion over the debate between the horizontalists and structuralists, refer to Pollin (1991), Fontana and Venturino (2003), and Fontana (2004)

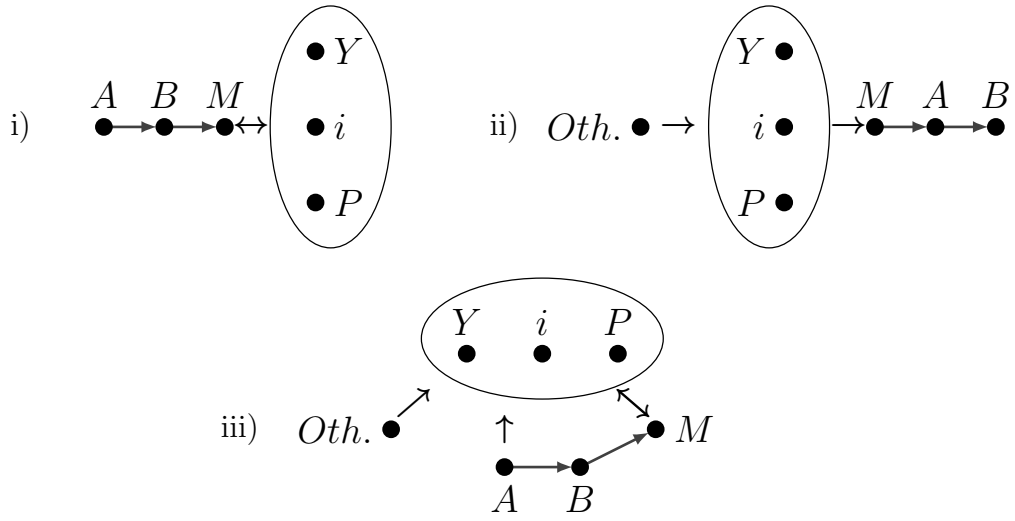

Figure 2.5: Causal Graphs summarizing *i*) the monetarist view, *ii*) the Post-Keynesian (Horizontalist) view, and *iii*) the Structuralist theory. *Oth.* stands for *Other Variables*, A stands for *Decision of the Authorities*, B is the monetary base, M is broad money, Y is income, P denotes prices, and i represents interest rates

it is widely acknowledged that they are in turn influenced by consumer preferences and the decisions of both fiscal and monetary authorities. For this reason, in the fully observed graphs constructed according to these theories, we will add a generic term called *Oth.* which groups all the other factors causing the treated variables. According to these considerations, Figure 2.5 graphically represents the causal structures underlying the theories. The cyclical behaviour between the determinants of money demand and money supply results from the components of the money multiplier.

It should be emphasized that the debate among these schools of thought is intricate, and the distinctions presented are simplified for the purposes of this paper.[5] For a comprehensive review of the theories on money exogeneity and endogeneity, see Sieroń (2019).

[5]For example, some economists also distinguish supporters of liquidity preference who agree with the core tenets of money endogeneity (Palley (1991)). Rochon and Rossi (2013) further differentiates revolutionary post-Keynesians, for whom money is endogenous irrespective of specific institutional arrangements.

Consistent with previous illustrative examples in the cointegration literature (e.g. Johansen and Juselius (1990)), we will include in the analysis proxies of the income, prices, interest rates and a monetary aggregate. Consequently, the set X_o will be composed by Y, P, i and M, while A, B as well as the other factors are treated as unobserved.

Examining the relationships in Figure 2.5, we can determine under which theory the money supply can be labeled as causally endogenous. In the monetarist graph (network i)), the primary source of causality is A. No variable in X_o blocks the forward paths between A and M, making the money supply weakly causally exogenous.[6] The opposite situation occurs in the horizontalist case (network ii)). Here, the original source of causality is represented by unknown components. However, all forward paths between $Oth.$ and M are blocked by Y, i, and P, thus M is treated as causally endogenous.

The structuralist view acknowledges the power of the monetary authority to govern the monetary base, which in turn influences M. Since A is considered among the fundamental trends, and its path to M is not blocked by Y, i, and P, the causal conceptualization of the structuralist view implies weak causal exogeneity for the monetary aggregate.

In the next subsections, we will empirically test the composition of א and ב to gather evidence supporting or contrasting the causal connections implied by these theories.

[6]Due to the feedback that Y, i, and P have on M, M is not strongly causally exogenous.

2.5.2 Data

The empirical analysis utilizes quarterly U.S. data spanning from Q2-1964 to Q3-2023, sourced from the FRED database. The determinants of money demand include the long-term rate (Bond Yields 10-year maturity), the short-term interest rate (3-month interbank rates), the Consumer Price Index (CPI), and the Economic Output (GNP), which has been seasonally adjusted to align with the assumptions underlying the causal exogeneity analysis. The proxy for money supply is the broader monetary aggregate (M3). Appendix A provides the links to the data sources and details the characteristics of the data. The variables indicating Money supply, Prices and Gross Domestic Product are transformed into logarithmic form to account for multiplicative effects.[7]

The data are visualized in Figures 2.6 -2.10. Since the current framework for long-term causal analysis and partially observed models does not address the role of constant terms and deterministic trends in studying causal endogeneity, it has been removed a deterministic linear trend to the series of money supply, GNP, and CPI. To verify whether the de-trending of these three variables results in $I(1)$ processes, a Dickey-Fuller test was applied to the modified time-series. The results are reported in Table 2.6 for the variables in levels and in Table 2.7 for the variables in first differences. Hereafter, the notations M, P, and Y denote the de-trended series of the logarithm of money supply, prices, and Gross Domestic Product, respectively.

[7]The multiplicative effects are derived from the identity $MV = PY$, where M denotes the quantity of money, V represents money velocity, P indicates prices, and Y corresponds to real GDP. This identity is prominently featured in the quantity theory of money

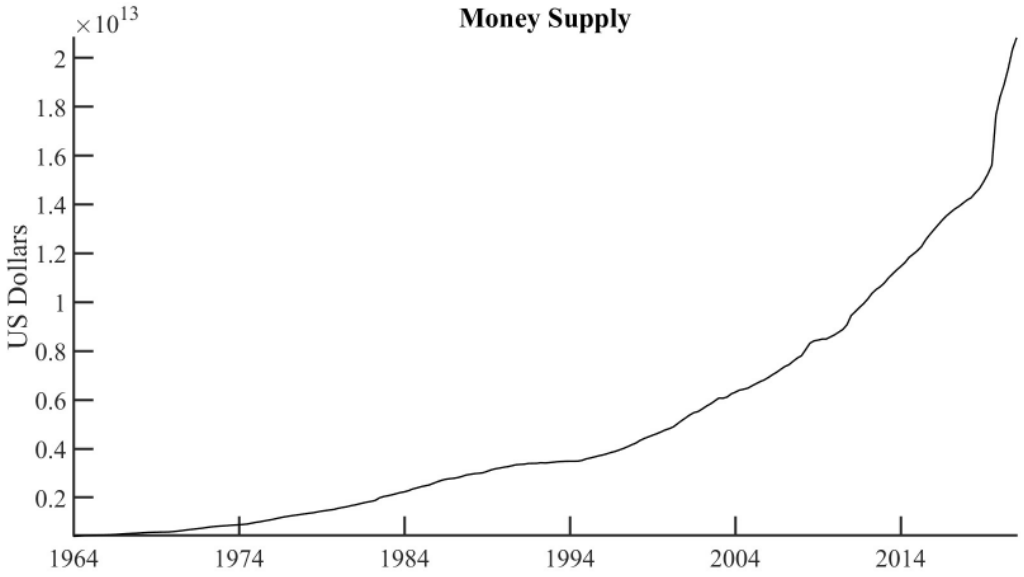

Figure 2.6: Broad Money Aggreagate M3. Retrieved from FRED, Federal Reserve Bank of St. Louis.

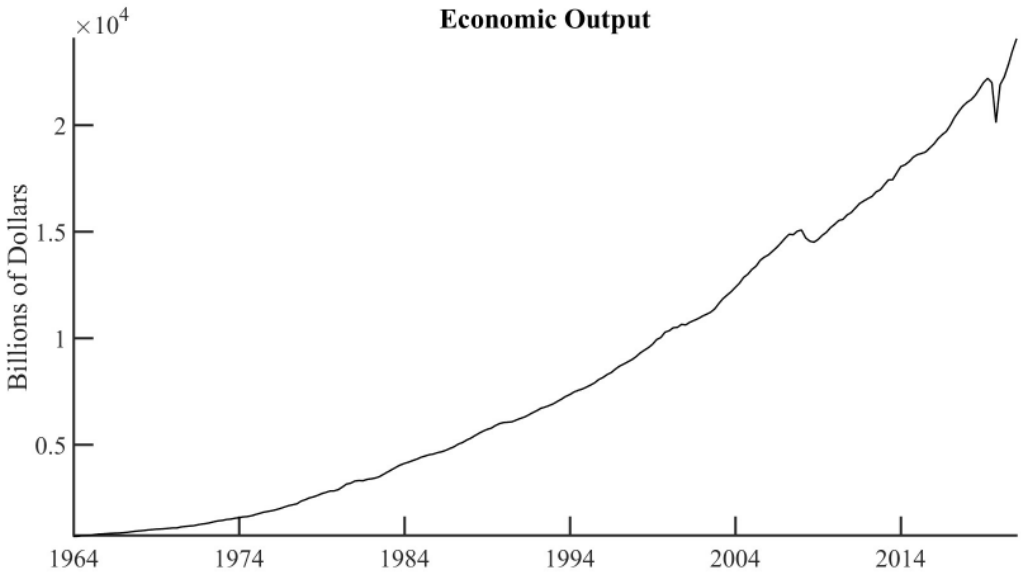

Figure 2.7: Gross National Product. Retrieved from FRED, Federal Reserve Bank of St. Louis.

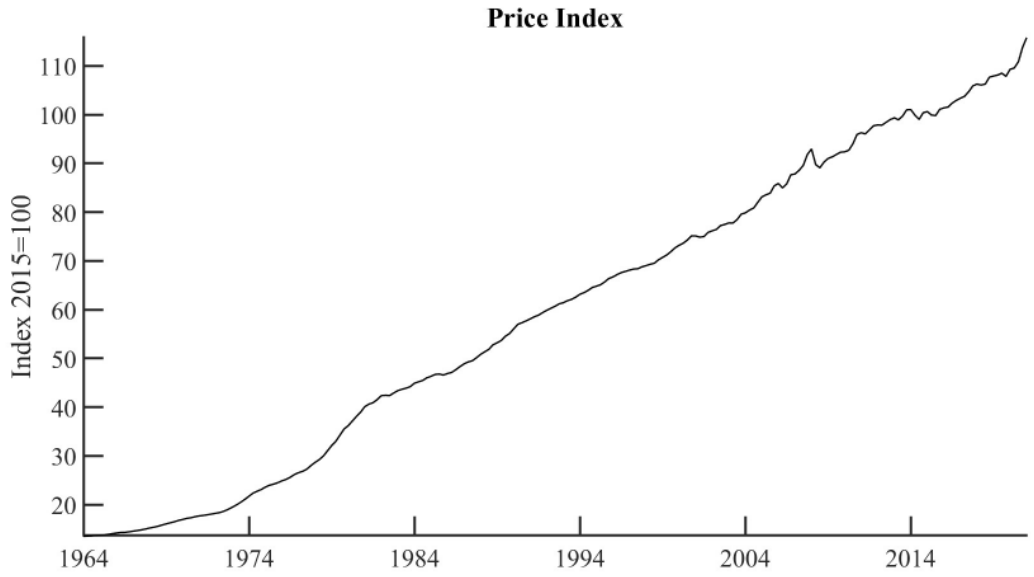

Figure 2.8: Consumer Price Index All Items. Retrieved from FRED, Federal Reserve Bank of St. Louis.

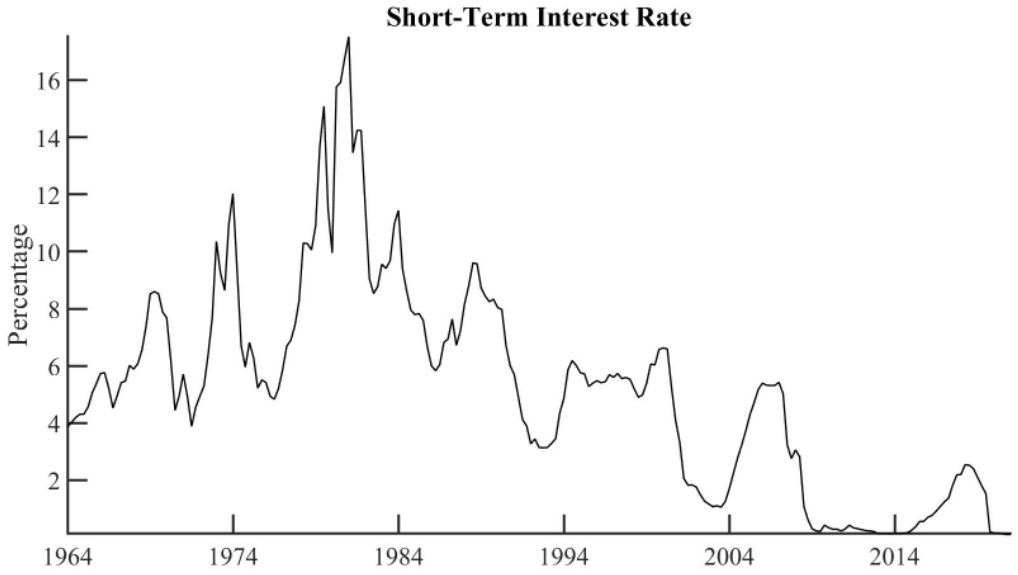

Figure 2.9: 3-month interbank rates. Retrieved from FRED, Federal Reserve Bank of St. Louis.

Figure 2.10: Bond Yields 10-year maturity. Retrieved from FRED, Federal Reserve Bank of St. Louis.

	P-Value	Statistic	C-Value
M	0.1083	-1.5776	-1.9427
Y	0.4108	-0.6559	-1.9427
P	0.1839	-1.2834	-1.9427

Table 2.6: Results of the Dickey-Fuller Test after Logarithmic Transformation and De-trending of the Raw Time-Series.

	P-Value	Statistic	C-Value
ΔM	**1.0000e-03**	**-8.0800**	-1.9427
ΔY	**1.0000e-03**	**-7.0545**	-1.9427
ΔP	**1.0000e-03**	**-13.1339**	-1.9427

Table 2.7: Results of the Dickey-Fuller Test Applied to the First Difference of the Series after Logarithm Transformation and De-trending. Bold Figures Indicate Rejections

	P-Value	Statistic	C-Value
r	0.2762	-1.0236	-1.9427
i	0.4479	-0.5546	-1.9427

Table 2.8: Results of the Dickey-Fuller Test For Raw Series of Interest Rates.

	P-Value	Statistic	C-Value
Δr	**1.0000e-03**	**-11.8761**	-1.9427
Δi	**1.0000e-03**	**-12.3397**	-1.9427

Table 2.9: Results of the Dickey-Fuller Test For Raw Series of Interest Rates. Bold Figures Indicate Rejections

The hypothesis of a unit root cannot be rejected for the series of the de-trended logarithms of money supply, GDP, and CPI. Conversely, there is strong evidence against the presence of unit roots in their first differences. These results suggest that the modified variables follow an $I(1)$ process, hence suitable for the analysis.

The behaviour of the two interest rate series does not indicate any deterministic trends. Therefore, these variables have not been modified. To verify whether the raw data are appropriate for the VECM representation examined in this study, the presence of a unit root was tested using the Dickey-Fuller test. The results are reported in Table 2.8 for the variables in levels and in Table 2.9 for the variables in first differences. Hereafter, the notations r and i denote the raw series of the short-term and long-term interest rates, respectively.

The Dickey-Fuller test fails to reject the null hypothesis for the variables in levels, whereas it strongly rejects the presence of a unit root for the first differences. These outcomes suggest that the variables are $I(1)$ and thus compatible with the cointegration analysis proposed so far.

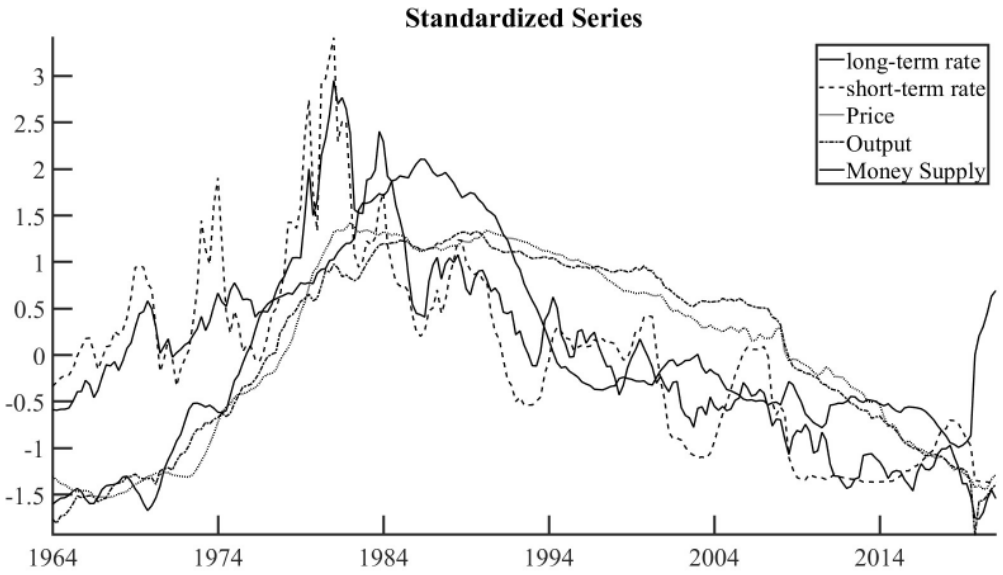

Figure 2.11: Final Series employed for the empirical analysis

Figure 2.11 presents the final dataset used for the empirical analysis. The series are normalized to facilitate graphical interpretation.

2.5.3 Empirical Results

The optimal order of the Vector Autoregressive model is determined using the Akaike Information Criterion (AIC). The results for various VAR(k) models are presented in Table 2.10. The optimal VAR order is 4, as the AIC value is minimized for the VAR(4) model. This high order is consistent with the findings of Johansen and Juselius (2014), who demonstrated that a partially observed C-VAR(1) model behaves as a VECM with exponentially decreasing short-term coefficients.

An accurate determination of the cointegration rank r is crucial as it influences subsequent procedures, especially the first test for the hypothesis of causal endogeneity. The trace test

Lag	AIC
0	1.7560e+03
1	-2.4293e+03
2	-2.6534e+03
3	-2.6676e+03
4	**-2.6837e+03**
5	-2.6472e+03
6	-2.6392e+03
7	-2.6243e+03

Table 2.10: AIC Values for the VAR(k) Fitted to the Levels of the Variables. The Minimal Value is Reported in Bold

results are reported in Table 2.11. The estimated rank is found to be 3, as the hypothesis $r \leq 3$ cannot be rejected, while evidence suggests rejecting the hypothesis $r \leq 2$.

H_0	Statistic	C-Values
$r = 0$	**101.9871**	60.0623
$r \leq 1$	**61.4263**	40.1751
$r \leq 2$	**33.3536**	24.2747
$r \leq 3$	11.9598	12.3206
$r \leq 4$	2.2671	4.1302

Table 2.11: Statistics of the Trace Test for the Identification of Cointegration Rank and Critical Values for a 5% Significance Level. Bold Numbers Represent Rejections of the Null

Table 2.12 reports the outcomes of the first test on causal endogeneity. The test result is not unique due to the procedure involving the replacement of the row corresponding to the tested variable with randomly generated numbers. Consequently, the statistic varies every time the series is generated. To address this variability, the test is conducted 100 times, and the table displays the average, maximum, and minimum statistics, along with the percentage of instances where the hypothesis of causal endogeneity could not be rejected.

$H_0: \tilde{r} \leq 2$	M	P	Y	r	i	C-Value
Avg. Stat.	**26.5071**	19.6224	**32.2107**	20.9124	**29.5339**	24.2747
Min. Stat.	23.9734	17.2613	**29.4157**	18.4149	**27.1660**	24.2747
Max. Stat.	**34.9996**	**28.8617**	**38.3539**	**29.1308**	**36.0220**	24.2747
Stat<Crit.	4%	98%	0%	92%	0%	

Table 2.12: Average, Minimum and Maximum Statistics for the Causal Endogeneity Test. $\tilde{r}$ is the Rank of the Manipulated Model. Critical Values and Number of Non-Rejections are Generated for a Significance Level of 5%

The null is rejected for i and Y in 100% of cases. Similarly, causal endogeneity for M is rejected in the vast majority of implementations, with the statistic lower than the critical value in only 4 out of 100 tests. Conversely, the results for P and r suggest that the price index and the short-term interest rate are causally endogenous, as the hypothesis is rejected in only 2% and 8% of cases, respectively.

H_0	Statistic	C-Value
M	**11.9857**	5.9915
P	5.2473	5.9915
Y	**17.4234**	5.9915
r	**6.3660**	5.9915
i	**15.0839**	5.9915

Table 2.13: Statistics and Critical Values for Weak Exogeneity Test. Bold Numbers Represent Rejection of the Null for a Significance Level of 5%

Table 2.13 reports the outcomes of the second test on causal endogeneity. Given that $\gamma_\perp$ is a 5×2 matrix, the approximated distribution under the null hypothesis concerning singleton sets is a $\chi^2(2)$. Consistent with the first test, the analysis of restrictions on $\gamma_\perp$ rejects the hypothesis of causal endogeneity for M, Y, and i, while it cannot reject the null for P. The two tests yield different results regarding r since the second test rejects the hypothesis of causal endogeneity for the short-term interest rate. However, it should

be noted that the statistic is close to the critical value, with a p-value of 0.0415, indicating that the hypothesis would not have been rejected at a 1% significance level.

To further verify the results, a standard weak exogeneity test was conducted. As proved in the previous Chapter, standard weak exogeneity is a sufficient condition for weak causal exogeneity. The test results are detailed in Table 2.14. Coherently with the previous findings, the hypothesis of weak exogeneity cannot be rejected for M. It should be emphasized that the rejection of the null for Y and i is not inconsistent with the results of the causal endogeneity test, as weak exogeneity is not a necessary condition for causal exogeneity.

H_0	Statistic	C-Value
M	7.2963	7.8147
P	**11.4840**	7.8147
Y	**9.5080**	7.8147
r	**15.0575**	7.8147
i	**18.2708**	7.8147

Table 2.14: Statistics and critical Values for Weak Exogeneity Test. Bold Numbers Represent Rejection of the Null for a Significance Level of 5%

The tests on causal endogeneity, as well as the standard weak exogeneity test, provide evidence supporting weak causal exogeneity for the log of the detrended and normalized series of money supply, suggesting that changes in the nominal value of M cannot be readily influenced by manipulating i, r, Y, and P. Conversely, there is insufficient evidence against the hypothesis of causal endogeneity for the logarithm of the price index, indicating that interventions on $\{M, Y, r, i\}$, or subsets thereof, can control the nominal value of P.

To summarise, these findings challenge the horizontalist perspective, which posits that fluctuations in the monetary base are driven by factors related to money demand, and in-

stead support the structuralist and monetarist views. Furthermore, they provide evidence in favor of policies targeting price levels through adjustments in Y, M, and interest rates.

2.6 Conclusions

This chapter identifies a novel duality between causal endogeneity and the mathematical properties of a manipulated version of the vector error correction model. Leveraging this duality, we constructed a log-likelihood ratio test to assess causal hypotheses. In section 2.4, we evaluated the performance of the two tests on causal endogeneity, demonstrating how these alternative procedures can provide complementary insights—one performs better under the null hypothesis, while the other exhibits higher power. Additionally, we observed that the structure of the graph and the presence of cyclicality negatively affect test performance. Finally, an empirical illustration demonstrated the practical relevance of the discussed causal tests.

The findings face some challenges. Firstly, the distribution employed under the null for both tests on causal endogeneity is an approximation, which could affect test size and lead to rejection rates under the null hypothesis that do not align with the significance level. Consequently, it is essential to extend the findings and identify the exact distribution under the null hypothesis.

The empirical illustration also highlighted practical implementation challenges. One issue is the presence of deterministic components, such as linear trends. In macroeconomic

analysis, these deterministic components are common, and their removal, as done in our illustration, might lead to information loss. Moreover, the determination of the cointegration rank is often unclear, as seen in the trace test results where the statistic can be very close to the critical value.

To summarise, the chapter shows that causal endogeneity tests generally perform well; however, there is a need to account for more complex processes and refine the testing procedures.

Chapter 3

Weak Exogeneity, Driving Variables and Non-Causality

3.1 Introduction

Whether a variable can be considered exogenous depends on whether it can be treated as given without losing information relevant to the model's purpose. Exogeneity is a multifaceted concept, with its definition varying depending on the parameter of interest and the model's objectives. The most common forms of statistical exogeneity are weak, strong, and super exogeneity. These concepts, grounded in the work of Koopmans (1950), have been refined by Engle et al. (1983) in conjunction with Richard (1980), Florens and Mouchart (1985a), and Florens and Mouchart (1985b).

The primary concept is weak exogeneity, a necessary condition for the more stringent forms. Strong exogeneity is the conjunction of weak exogeneity with Granger non-causality,

while super exogeneity combines weak exogeneity with invariance. Weak exogeneity ensures valid conditional forecasting, and strong exogeneity ensures effective policy simulations. This chapter focuses only on weak exogeneity, as it is closely related to model restrictions in long-term systems.

Weak exogeneity ensures that inference and hypothesis testing about some parameters of interest can be conducted by looking to a conditional rather than a joint density. Consider a set of variables Y split into X and Z:

$$Y = \begin{bmatrix} X \\ Z \end{bmatrix}$$

Studying exogeneity properties helps determine whether modeling Z is necessary to understand X. This is particularly useful in complex systems, such as economic models with many variables.

The starting point for defining weak exogeneity is the decomposition of the joint probability function. Denoting $F_u()$ as the density function of a variable u, we factorize $F_Y(Y_t; \theta, Y_{t-1:0})$ into a conditional and a marginal density function, where θ is the vector of parameters of the process Y and $Y_{t-1:0}$ represents past observations, the decomposition is as follows:

$$F_Y(Y_t; \theta, Y_{t-1:0}) = F_{X|Y}(X_t|Y_t; \lambda_1, Y_{t-1:0}) F_Z(Z_t; \lambda_2, Y_{t-1:0})$$

where λ_1 and λ_2 are the parameters of the conditional and marginal models, respectively.

In general, assessing only the conditional probability function $F_{X|Y}(X_t|Y_t;\lambda_1,Y_{t-1:0})$ leads to a loss of information about the conditional model itself. The weak exogeneity condition ensures that all relevant information about the conditional model is contained within $F_{X|Y}(X_t|Y_t;\lambda_1,Y_{t-1:0})$, obviating the need to evaluate $F_Z(Z_t;\lambda_2,Y_{t-1:0})$. This occurs when the parameters of interest, denoted by ψ, are functions exclusively of λ_1 and the factorization allows a sequential cut (see Florens et al. (2019), Chapter 6).

The definition of parameters of interest is crucial since models can be structured in various ways, and researchers may not be concerned with all parameters influencing the data density. The factorization performs a sequential cut if λ_1 and λ_2 are variation-free. Sequential cuts address the dependencies between the parameter spaces of λ_1 and λ_2. In essence, λ_1 and λ_2 are variation-free if the value of one does not restrict the possible values of the other. Define Λ_1 and Λ_2 as the parameter spaces of λ_1 and λ_2, respectively. Mathematically, the parameters are variation-free if the space of (λ_1,λ_2) equals $\Lambda_1 \times \Lambda_2$, where $\times$ denotes the Cartesian product. To summarise, the two conditions for weak exogeneity are:

- The parameters of interest ψ are a function of λ_1 only: $\psi = f(\lambda_1)$
- The range of the parameter (λ_1,λ_2) is $\Lambda_1 \times \Lambda_2$

To clarify, we report an example taken from Ericsson and Irons (1994): consider x_t and z_t jointly normally distributed with mean vector μ and covariance matrix Ω,

$$Y_t = \begin{bmatrix} x_t \\ \\ z_t \end{bmatrix} \sim N(\mu,\Omega)$$

with

$$\mu = \begin{bmatrix} \mu_x \\ \mu_z \end{bmatrix} \qquad \Omega = \begin{bmatrix} \omega_x^2 & \omega_{x,z} \\ \omega_{z,x} & \omega_z^2 \end{bmatrix}$$

The joint distribution can be factorized as

$$x_t | z_t \sim N(a + bz_t, \sigma_x^2)$$

$$z_t \sim N(\mu_2, \omega_z^2)$$

where $b = \frac{\omega_{x,z}}{\omega_z^2}$, $a = \mu_x - b\mu_z$, $\sigma_x^2 = \omega_x^2 - \frac{\omega_{x,z}^2}{\omega_z^2}$. In model form, Y, x, and z would become

$$Y_t = \mu + \varepsilon_{Y,t} \qquad \varepsilon_{Y,t} \sim (0, \Omega)$$

$$x_t = a + bz_t + \varepsilon_{x,t} \qquad \varepsilon_{x,t} \sim (0, \sigma_x^2)$$

$$z_t = \mu_z + \varepsilon_{z,t} \qquad \varepsilon_{z,t} \sim (0, \omega_z^2)$$

Set the parameter of interest be the ones of the conditional model, i.e. $\psi = \lambda_1 = \{a, b, \sigma_x^2\}$.

The mean μ and covariance matrix Ω of the joint model as a function of λ_1 and λ_2 are

$$\mu = \begin{bmatrix} a + b\mu_z \\ \mu_z \end{bmatrix} \qquad \Omega = \begin{bmatrix} \sigma_x^2 + b^2\omega_z^2 & b\omega_z^2 \\ b\omega_z^2 & \omega_z^2 \end{bmatrix}$$

The parameter space of μ is the whole $\mathbb{R}^2$, therefore a particular value of μ_z does not

constrain the possible values of a and b. On the other hand, the requirement "Ω is positive definite" is automatically satisfied for every admissible value of ψ regardless of the realization λ_2 (see Drèze and Richard (1983)). Consequently, z is weakly exogenous for the parameters ψ and the conditional model can be estimated taking z as given.

There is an analytical relationship between cointegration and weak exogeneity, in the sense that in cointegrated systems, weak exogeneity manifests through restrictions on the cointegrated models. Consider the VECM:

$$\Delta Y_t = \alpha\beta' Y_{t-1} + \varepsilon_t$$

where the short-term dynamic as well as constant and deterministic terms are excluded without loss of generality. By splitting Y into X and Z, one can decompose the model as follows:

$$\Delta X_t = \alpha_x\beta' Y_{t-1} + \varepsilon_{X,t}$$

$$\Delta Z_t = \alpha_z\beta' Y_{t-1} + \varepsilon_{Z,t}$$

where

$$Y_t = \begin{bmatrix} X \\ Z \end{bmatrix} \quad \alpha = \begin{bmatrix} \alpha_x \\ \alpha_z \end{bmatrix} \quad \varepsilon = \begin{bmatrix} \varepsilon_x \\ \varepsilon_z \end{bmatrix}$$

The conditional model for ΔX_t given the past and ΔZ_t is:

$$\Delta X_t = \xi\Delta Z_t + (\alpha_x - \xi\alpha_Z)\beta' Y_{t-1} + \varepsilon_{X,t} - \xi\varepsilon_{Z,t}$$

where $\xi = \Omega_{12}\Omega_{22}^{-1}$ and $Var(\varepsilon) = \Omega$ is divided as follows

$$\Omega = \begin{bmatrix} \Omega_{11} & \Omega_{12} \\ \Omega_{21} & \Omega_{22} \end{bmatrix}$$

Consider the parameter of interest the long-run coefficient, i.e. $\psi = \{\beta\}$. Johansen (1995) proves that if $\alpha_z = 0$, then Z_t is weakly exogenous for the parameter β and the maximum likelihood estimator of α_x and β can be calculated from the conditional model. This can be easily verified by noticing that when $\alpha_z = 0$, the conditional and marginal models become

$$\Delta X_t = \xi \Delta Z_t + \alpha_x \beta' Y_{t-1} + \varepsilon_{X,t} - \xi \varepsilon_{Z,t}$$

$$\Delta Z_t = \varepsilon_{Z,t}$$

$\alpha_z = 0$ ensures that ψ enters the partial model only, in fact, the sets of parameters in the marginal and conditional models are

$$\theta_x = (\Omega_{22})$$

$$\theta_z = (\alpha_x, \beta, \xi, \Omega_{11.2} = \Omega_{11} - \Omega_{12}\Omega_{22}^{-1}\Omega_{21})$$

The parameter space of

$$\theta = (\alpha_1, \beta, \Omega)$$

is decomposed into a product space of the parameters in the marginal and conditional

models. This follows from the properties of the Gaussian distribution, which ensure that the parameters in the two models are variation-free (see, for instance, Johansen (1992c)). This can be verified by selecting arbitrary values for θ_x and θ_z and then constructing θ. If Ω_{22} and $\Omega_{11.2}$ are positive definite matrices, then Ω will also be positive definite. In fact, for multivariate Gaussian distributions, Ω_{22} is variation independent of the parameters ξ and $\Omega_{11.2}$ (see Barndorff-Nielsen (2014)). This proves that weak exogeneity can be expressed as a restriction on the rows of the adjustment matrix α.

Inference of statistical exogeneity permits simpler modeling strategies, reduces computational expenses, and helps isolate invariant mechanisms. However, in cointegrated systems, the close connection between weak exogeneity and model restrictions has led to associating this statistical concept with conclusions regarding the structure of economic systems. Specifically, this pertains to the identification of driving forces, the introduction of non-stationarity, and absence of causal influence.

For example, in Franchi and Juselius (2007), the rejection of the hypothesis of weak exogeneity for shocks to capital stock led the authors to conclude that this variable cannot be considered part of the driving stochastic trends. Conversely, the non-rejection of weak exogeneity for per capita consumption suggested that this variable is one of the driving forces, contradicting a key assumption of the real business cycle model.

In Juselius et al. (2014), the authors used weak exogeneity tests to identify the absence of influence. They assessed the restrictions of the adjustment matrix to determine whether foreign aid remains unaffected in the long run by key macroeconomic variables. Addition-

ally, they used weak exogeneity tests alongside other restrictions to test explicit causal statements.

While causal conclusions based on the presence of weak exogeneity are reasonable when all significant factors affecting a phenomenon are modeled, these connections may not extend to cases where not all variables involved in a causal structure are identified. Common causal sources and potential cyclical behaviour might disrupt the one-to-one mapping between weak exogeneity, driving forces, and non-causality.

This chapter incorporates recent developments in the literature on partially observed cointegrated systems in the study of the causal meaning of weak exogeneity. Investigating weak exogeneity properties in partially observed models helps to understand when weak exogeneity embodies causal understanding, even if not all factors affecting a system are identified. To define driving trends and absence of influence in causal terms, we follow approaches similar to those employed in the previous chapters and the ones utilized by Hoover (2020). The chapter also presents simulation results, highlighting a practical issue with testing restrictions on the adjustment matrix in partially observed systems. This issue involves a misalignment between the theoretical form of the adjustment matrix and the average outcome of the weak exogeneity test. Specifically, a low power of the weak exogeneity test is observed when the rows of the adjustment matrix correspond to weakly causally exogenous variables. We will describe potential difficulties arising from this issue and discuss the consequences concerning the interpretation of the results.

The chapter is structured as follows: The next section discusses long-term causality and

provides examples showing how the concepts of non-causality and driving forces do not necessarily align with weak exogeneity when dealing with latent components. Section 3.3 provides formal definitions of leading trend and non-causality. Section 3.4 explores the interrelations between weak exogeneity and the causal idea of driving trends, illustrating instances where testing restrictions on the adjustment matrix aids in causal understanding. Section 3.5 illustrates the relation between restrictions on the adjustment matrix and non-causality in its weak and strong form as explained in section 3.3. Using simulated data, section 6 highlights that under certain circumstances, the weak exogeneity test performs poorly in partially observed systems. Section 3.7 presents an empirical application to illustrate the correct causal interpretation of weak exogeneity. Finally, section 3.8 provides concluding remarks.

3.2 Causality and Model Restrictions

Cointegration pertains to long-term relationships, and the structural analysis in cointegrated systems is inherently concerned with long-run aspects. Thus, the counterfactual assessment emphasizes the limiting values of the variables rather than specific points in the future. In the context of statistical causality, such as Granger causality, the distinction between long-term and short-term causality is well-established. Granger and Lin (1995) highlighted this distinction through spectral densities, which was further elaborated by Dufour and Renault (1998) and Dufour and Taamouti (2010) within the temporal domain. Intuitively, causality from X_t to Y_{t+h_i} can be labeled as "long-run" when the temporal lag

h_i is "large enough".

We propose a straightforward extension of this distinction, based on incremental predictability, to structural causality by emphasizing the indices $\boldsymbol{h} = h_1, h_2, \ldots$. Short-term causality refers to scenarios where manipulation of variable X affects the dynamic behaviour of variable Y up to a specific future point. Long-term causality occurs when the effect triggered by the intervention persists over time and influences the limiting value of Y. Naturally, these considerations assume the "ceteris paribus clause":

Definition 8 (***Short-Term Structural Causality***)**.** *A variable X is deemed to exert short-term causality on variable Y when an arbitrary manipulation of X_t induces alterations in the values of $\Delta Y_{t+\boldsymbol{h}}$, where $\boldsymbol{h}$ represents a set of integer indices whose generic entry satisfies $0 \leq h_i < \infty$. The influence on $\Delta Y_{t+\boldsymbol{h}}$ is contingent upon the condition that all other factors remain constant.*

Definition 9 (***Long-Term Structural Causality***)**.** *A variable X is deemed to exert long-term causality on variable Y when an arbitrary manipulation of X_t induces alterations in the values of $\Delta Y_{t+\boldsymbol{h}}$, where $\boldsymbol{h}$ represents a set with generic entries satisfying $0 \leq h_i$ and $\boldsymbol{h} \to \infty$. The influence on $\Delta Y_{t+\boldsymbol{h}}$ is contingent upon the condition that all other factors remain constant.*

Short-term causality implies that the increments of Y are a function of current and past h increments of X, i.e. $\Delta Y_t = f(\Delta X_t, \Delta X_{t-1}, \ldots, \Delta X_{t-h})$ with $h < \infty$. An intervention in X at time t (i.e. $\Delta X_t \neq 0$), impacts Y up to $t + h$, after which ΔY_t reverts to zero. Figure 3.1 depicts the short-term effects on Y following a unitary intervention in X at

$t = 5$. As we can observe, the generated response in the subsequent values of ΔY_t has a short-term nature since it concludes after 5 time periods; indeed, the level of Y ceases to increase after $t = 10$ (i.e., $\Delta Y_t = 0$ for $t > 10$).

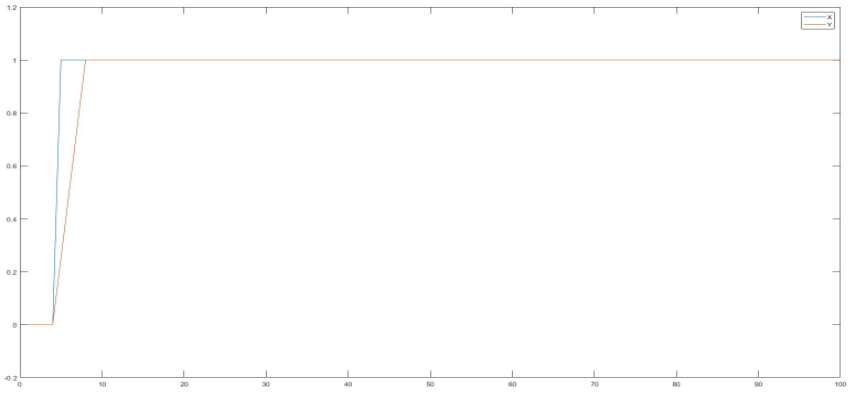

Figure 3.1: Simulation of Short-Term Effects on Y (Red Line) After a Unitary Intervention on X at $t = 5$

Conversely, long-term causality entails continuous increments perpetually influencing Y, as $\Delta Y_t = f(\Delta X_t, \Delta X_{t-1}, \ldots, \Delta X_{t-\infty})$. Figure 3.2 illustrates the enduring effects on Y following a unitary intervention in X at $t = 5$. The intervention induces a response in the subsequent values of ΔY_t which persists over time, and despite its diminishing effect, it never disappears.

From an economic perspective, the distinction between long-term and short-term causality is particularly interesting when examining how long-term causality manifests within a model. Specifically, when market forces instigate permanent changes to reestablish stable relationships, long-term causality can be effectively described using an error correction model.

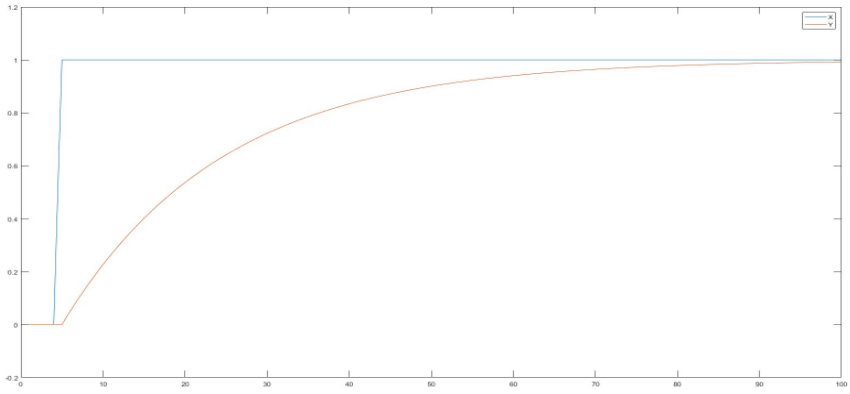

Figure 3.2: Simulation of Long-Term Effects on Y (Red Line) After a Unitary Intervention on X at $t = 5$

In examining this facet, the differentiation between long-term and short-term causality extends beyond mere temporal considerations to encompass the stability of relationships among variables. Consider two variables, X and Y, where X causes Y in the long-term. An intervention in X at time t triggers a permanent response in Y. Economically, we are interested in scenarios where these variables have stable relationships, meaning the reaction following the intervention aims to achieve a steady-state position. Assume the equilibrium is given by the simple relation $X = Y$. This implies that when $X - Y = 0$, there is no tendency for the system to deviate from this position, and the expected value for both positive and negative increments of the variables is zero.

Consider an external shock to X at time t, creating the inequality $X_t - Y_t \neq 0$. Since X causes Y in the long term, this inequality generates a perpetual effect on ΔY. If the long-term connection is stable, the sum $\sum_{i=t+1}^{\infty} \Delta Y_i$ cannot diverge. Specifically, it should equal the disequilibrium caused by the external shock, i.e., $\lim_{t\to\infty} \sum_{i=t+1}^{\infty} \Delta Y_i = X_t - Y_t$.

A straightforward way to mathematically describe this behaviour is to assume that each period ΔY reacts in order to adjust a stable portion φ of the disequilibrium generated in the previous period. For instance, if φ equals 0.5, then at time $t+1$, ΔY adjusts to half the disequilibrium generated at time t, i.e., $X_t - Y_t$. Similarly, at time $t+2$, ΔY adjusts to half the remaining disequilibrium at time $t+1$, and this process continues perpetually.

The model describing the value of ΔY_t is thus a linear function of $X_{t-1} - Y_{t-1}$:

$$\Delta Y_t = \varphi(X_{t-1} - Y_{t-1})$$

To generalize, consider the equilibrium relation $Y = \beta X$. In this generic case, the mathematical representation of the long-term causal relation $X \to Y$ becomes

$$\Delta Y_t = \alpha_Y(Y_{t-1} - \beta X_{t-1})$$

where α captures the speed to which Y adjusts to equilibrium.

Causality need not be symmetric. For instance, given the causal graph $X \to Y$, an arbitrary manipulation of Y does not affect the limiting value of X (assuming all other factors remain constant). This non-causality implies that positive and negative increments of X are not influenced by the disequilibrium between X and Y, i.e., no reaction is triggered in ΔX when $Y - \beta X \neq 0$. The error correction model representation for X is thus:

$$\Delta X_t = \alpha_X(Y_{t-1} - \beta X_{t-1})$$

where $\alpha_X = 0$.

The equations for X and Y can be combined into a Vector Error Correction Model (VECM):

$$\Delta \begin{bmatrix} X \\ Y \end{bmatrix}_t = \begin{bmatrix} 0 \\ \alpha_Y \end{bmatrix} \begin{bmatrix} -\beta & 1 \end{bmatrix} \begin{bmatrix} X \\ Y \end{bmatrix}_{t-1} + \varepsilon_t$$

where the term ε_t includes unpredictable shocks, adding stochastic behaviour to the system.

The asymmetry in the causal relationship is captured in the adjustment matrix, where non-causality produces a zero vector in the row corresponding to the non-caused variable. Moreover, the null row corresponds to the variable introducing Non-stationarity into the system. For instance, in the example $X \to Y$, the adjustment matrix contains a zero in the row corresponding to X. This restriction indicates that the dynamic of X is independent of the equilibrium between X and Y, meaning X is not caused by Y.

At first glance, these findings may suggest that the concept of weak exogeneity aligns with non-causality. However, this holds only partially true. On one hand, in a fully observed model, weak exogeneity necessarily corresponds to the variables whose nodes in the causal graph have no incoming edges, since a zero row implies that the corresponding variable remains unaffected by steady state violations. On the other hand, the one-to-one mapping between weak exogeneity and causal properties is lost when we fail to identify every variable involved in the unobserved data generating process.

To illustrate, we decompose a set of variables Y, representing the set containing every

factor affecting a system, into observed variables X_o, unobserved variables X_u, and a set T containing fundamental trends. As illustrated in the previous chapters, starting from the state space representation

$$\Delta X_{o,t} = M_o X_{o,t-1} + M_{ou} X_{u,t-1} + N_o T_{t-1} + \nu_{o,t}$$

$$\Delta X_{u,t} = M_{uo} X_{o,t-1} + M_u X_{u,t-1} + N_u T_{t-1} + \nu_{u,t}$$

$$\Delta T_t = \nu_{T,t}$$

one can derive the adjustment, cointegration, and covariance matrix of the residuals in the model:

$$\Delta X_{o,t} = \alpha_o \beta_o' X_{o,t-1} + \sum_{i=1}^{\infty} \Gamma_{o,i} \Delta X_{o,t-i} + \varepsilon_{o,t}$$

Specifically

$$\Sigma_o = \mathrm{Var}(\varepsilon_o) = \begin{bmatrix} M_{ou} & N_o \end{bmatrix} V \begin{bmatrix} M_{ou}' \\ N_o' \end{bmatrix} + \Omega_o \tag{3.1}$$

$$\alpha_o = \Sigma_o \Big(M_{ou} V_{uT} + N_o V_T \Big)_\perp \tag{3.2}$$

and

$$\beta_o' = \big(N_o - M_{ou} M_u^{-1} N_u \big)_\perp' \Big(M_o - M_{ou} M_u^{-1} M_{uo} \Big) \tag{3.3}$$

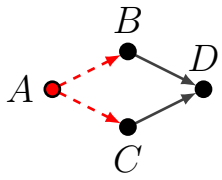

Figure 3.3: Causal Graph Generating Misalignment Between Weak Exogeneity and Non-Causality Example 1

where V is

$$V = \text{Var}\begin{bmatrix} X_{u,t} \\ T_t \end{bmatrix} \Big| X_{o,t} \Big] = \begin{bmatrix} V_u & V_{u,T} \\ V_{T,u} & V_T \end{bmatrix}$$

If only fundamental trends are unobserved, equation 3.2 reduces to

$$\alpha_o = N_{o\perp}\Omega_o \tag{3.4}$$

To demonstrate the loss of duality between non-causality and weak exogeneity, consider the causal graph in Figure 3.3. The error correction model for variables A, B, C, and D includes three causal connections: $A \rightarrow B$, $A \rightarrow C$, and $B \rightarrow D \leftarrow C$. These connections correspond to equilibrium relations: $B = \beta_B A$, $C = \beta_C A$, and $D = \beta_{DB} B + \beta_{DC} C$. The first equilibrium connection affects the increments of B. Consequently, the corresponding column in the adjustment matrix contains a non-null entry in the row relative to B. Similarly, the second and the third equilibra influence the dynamics of C and D respectively. Therefore, the second and third columns will contain a non-null entry

in the rows corresponding to C and D respectively. The final VECM is thus

$$\Delta \begin{bmatrix} A \\ B \\ C \\ D \end{bmatrix}_t = \begin{bmatrix} 0 & 0 & 0 \\ \alpha_B & 0 & 0 \\ 0 & \alpha_C & 0 \\ 0 & 0 & \alpha_D \end{bmatrix} \begin{bmatrix} -\beta_B & 1 & 0 & 0 \\ -\beta_C & 0 & 1 & 0 \\ 0 & -\beta_{DB} & \beta_{DC} & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \\ D \end{bmatrix}_{t-1} + \begin{bmatrix} \varepsilon_A \\ \varepsilon_B \\ \varepsilon_C \\ \varepsilon_D \end{bmatrix}_t$$

As anticipated, in the fully observed model weak exogeneity aligns with non-causality. In fact, a zero row in the adjustment matrix is associated with A, which is not caused by any other variable within the system. Nevertheless, if we compute the adjustment matrix of the partially observed model, this mapping fails. To prove it, we need to retrieve α_o. Since the only unobserved variable is the fundamental trend A, the adjustment matrix of the partially observed model can be computed with (3.4).

The coefficient N_o relating X_o to T is

$$N_o = \begin{bmatrix} -\alpha_B\beta_B \\ -\alpha_C\beta_C \\ 0 \end{bmatrix}$$

whose orthogonal complement is a 3×2 matrix of full rank. Therefore, by construction, it cannot contain two zero rows. Furthermore, if the row corresponding to B is non-zero, the row corresponding to C is necessarily non-null in order to ensure the equality $N_o' N_{o\perp}$. Given that Ω_o is diagonal, the resulting α_o matrix maintains the same characteristic of

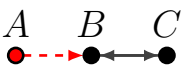

Figure 3.4: Causal Graph Generating Misalignment Between Weak Exogeneity and Non-Causality Example 2

$N_{o\perp}$, i.e., it does not contain zero rows. An admissible realization of α_o is

$$\alpha_o = \begin{bmatrix} \alpha_{o1} & 0 \\ \alpha_{o2} & 0 \\ 0 & \alpha_{o3} \end{bmatrix}$$

which suggests that weak exogeneity in the partially observed model might not necessarily imply non-causality.

If we consider cyclical graphs, it might even occurs that a variable influenced by others exhibits weak exogeneity. To illustrate, consider the cyclical graph depicted in Figure 3.4. In this scenario, the error correction representation involving variables A, B, and C displays two causal connections: $A \to B \leftarrow C$, and $B \to C$. Each connection corresponds to an equilibrium relation: $B = \beta_{BA}A + \beta_{BC}C$, and $C = \beta_C B$. The first equilibrium influences changes in B, resulting in a non-null entry in the adjustment matrix's corresponding column only in the row relative to B. Likewise, the second equilibrium influences C, leading to a non-null entry in the second column of the adjustment matrix only in the row relative

to C. Hence, the final Vector Error Correction Model is:

$$\Delta \begin{bmatrix} A \\ B \\ C \end{bmatrix}_t = \begin{bmatrix} 0 & 0 \\ \alpha_B & 0 \\ 0 & \alpha_C \end{bmatrix} \begin{bmatrix} -\beta_{BA} & 1 & -\beta_{BC} \\ 0 & -\beta_C & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \end{bmatrix}_{t-1} + \begin{bmatrix} \varepsilon_A \\ \varepsilon_B \\ \varepsilon_C \end{bmatrix}_t$$

Similar to the previous scenario, in the fully observed model, weak exogeneity aligns with non-causality and the introduction of non-stationarity. In fact, the zero row in the adjustment matrix corresponds to the variable A, which is unaffected by any other variable within the system. To analyse the partially observed model involving $\{B, C\}$, we utilize equation (3.2) in its reduced form (Equation (3.4)) since the only unobserved variable is the fundamental trend A. The coefficient N_o relating X_o to T takes the form:

$$N_o = \begin{bmatrix} -\alpha_B \beta_{BA} \\ 0 \end{bmatrix}$$

Its orthogonal complement, a 2×1 matrix of full rank, must be constructed in such a way to ensure the equality $N_o' N_{o\perp} = 0$. This necessarily implies a zero entry in the first row of $N_{o\perp}$. Given that Ω_o is diagonal, the resulting α_o matrix maintains the same characteristic of $N_{o\perp}$, containing a null vector in the row corresponding to B:

$$\alpha_o = \begin{bmatrix} 0 \\ \alpha_{o1} \end{bmatrix}$$

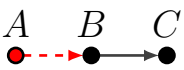

Figure 3.5: Causal Graph Generating Alignment Between Weak Exogeneity and Non-Causality

indicating that B exhibits weak exogeneity.

This example underscores that weak exogeneity not only does not always match non-causality, but it can even characterize a variable influenced by others. In fact, despite being caused by C, B is weakly exogenous.

However, this discrepancy does not imply that non-causality in partially observed models always deviates from the restrictions of the adjustment matrix. Consider the graph depicted in Figure 3.5. Here, the error correction representation involving A, B, and C contains two causal connections: $A \to B$, and $B \to C$. Each connection corresponds to an equilibrium relation: $B = \beta_B A$, and $C = \beta_C B$. The first equilibrium affects changes in B, resulting in a non-null entry in position $\{2, 1\}$ of the adjustment matrix. Likewise, the second equilibrium influences C, leading to a non-null entry in position $\{3, 2\}$ of α. Hence, the final VECM is:

$$\Delta \begin{bmatrix} A \\ B \\ C \end{bmatrix}_t = \begin{bmatrix} 0 & 0 \\ \alpha_B & 0 \\ 0 & \alpha_C \end{bmatrix} \begin{bmatrix} -\beta_B & 1 & 0 \\ 0 & -\beta_C & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \end{bmatrix}_{t-1} + \begin{bmatrix} \varepsilon_A \\ \varepsilon_B \\ \varepsilon_C \end{bmatrix}_t$$

Again, in the fully observed model, weak exogeneity aligns with non-causality. If we compute the adjustment matrix of the partially observed model, we find that the mapping

between weak exogeneity and non-causality extends to the error correction representation involving B and C only. To illustrate, we first retrieve N_o:

$$N_o = \begin{bmatrix} -\alpha_B \beta_B \\ \\ 0 \end{bmatrix}$$

whose orthogonal complement is a 2×1 matrix of full rank. To ensure the equality $N_o' N_{o\perp}$, the first row of $N_{o\perp}$ cannot contain non-zero entry. Since the only unobserved variable is the fundamental trend A, the adjustment matrix of the partially observed model can be computed with (3.4). Given that Ω_o is diagonal, the resulting α_o matrix maintains the same characteristic of $N_{o\perp}$, containing a null vector in the row corresponding to B:

$$\alpha_o = \begin{bmatrix} 0 \\ \\ \alpha_{o1} \end{bmatrix}$$

This demonstrates that weak exogeneity can correspond to non-causality in partially observed models.

In summary, the extension of weak exogeneity test outcome to causal conclusions is not automatic unless all variables affecting a system are adequately modeled. Consequently, conclusions regarding the causal structure and driving forces derived from testing restrictions of the adjustment matrix should be approached with caution. This chapter aims to analyse the circumstances under which weak exogeneity in partially observed models aligns with non-causality and when it is appropriate to draw causal conclusions from the

analysis of VECM coefficients.

3.3 Driving Trends and Non-Causality

As illustrated in the introduction, weak exogeneity is often associated with driving trends and non-causality. In this section, we clarify what is meant by these concepts.

We will first list the classical definitions and then propose alternative concepts based on the graphs representing the causal connections of the fully observed system. Subsequently, sections 3.4 and 3.5 will analyse whether weak exogeneity can be associated with leading or unaffected variables.

Subsection 3.3.1 presents the standard definition of driving trends and proposes an alternative definition based on the properties of latent causal structures. Subsection 3.3.2 articulates the idea of absence of influence, proposing a weak and a strong form of non-causality.

3.3.1 Driving Trends

As illustrated in the introduction, weak exogeneity is often associated with driving trends. This subsection aims to clarify the idea of driving variables.

The notion of driving forces is not unambiguous, and various authors have provided different definitions. Nonetheless, all definitions proposed thus far are grounded in the representation of X_t as a function of the error term ε. This representation allows for the

decomposition of X_t into stationary and non-stationary components. The driving trends are then identified from a transformation of the non-stationary component of X_t.

The standard Vector Error Correction Model (VECM)

$$\Delta X_{o,t} = \alpha_o \beta_o' X_{o,t-1} + \sum \Gamma_{oi} \Delta X_{o,t-i} + \varepsilon_t$$

finds the following solution for X_t as a function of the error term ε:

$$X_{o,t} = \boldsymbol{C} X_{o,0} + \boldsymbol{C} \sum_{i=1}^{t} \varepsilon_i + \boldsymbol{Y}_t \tag{3.5}$$

where $\boldsymbol{Y}_t$ is a stationary process, $X_{o,0}$ is the initial value of X_o, and $\boldsymbol{C} = \beta_{o\perp}(\alpha_{o\perp}'\beta_{o\perp})^{-1}\alpha_{o\perp}'$. Here, $\Gamma = \sum \Gamma_{oi} - I$.

Clearly, non-stationarity arises from the term $\boldsymbol{C} \sum_{i=1}^{t} \varepsilon_{oi}$. Consequently, authors have defined driving forces starting from the representation

$$\beta_{o\perp}(\alpha_{o\perp}'\beta_{o\perp})^{-1}\alpha_{o\perp}' \sum_{i=1}^{t} \varepsilon_{oi}$$

The matrix $\boldsymbol{C}$ is a reduced rank matrix that can be decomposed similarly to $\Pi_o = \alpha_o \beta_o'$:

$$\tilde{\beta}_o \alpha_{o\perp}' \sum_{i=1}^{t} \varepsilon_i$$

where $\tilde{\beta}_o = \beta_{o\perp}(\alpha_{o\perp}'\beta_{o\perp})^{-1}$. This representation prompts the definition of driving trends

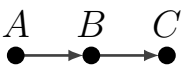

Figure 3.6: Causal Graph to Illustrate the Idea of Driving Forces

as

$$\alpha'_{o\perp} \sum_{i=1}^{t} \varepsilon_i$$

Similar to Π, $\boldsymbol{C}$ does not have a unique decomposition. For any given full rank matrix A, it holds that $\boldsymbol{C} = \beta_{o\perp}(\alpha'_{o\perp}\beta_{o\perp})^{-1}A^{-1}A\alpha'_{o\perp}$. Consequently, the fundamental trends can also be defined as $A\alpha'_{o\perp}\sum_{i=1}^{t}\varepsilon_i$. This variability has led the literature to introduce different definitions of driving trends.

In this thesis, we depart from definitions based on representation (3.5), and instead anchor the definition to the latent causal graphs describing the causal connections of the system at hand.

Intuitively, driving trends are variables that introduce non-stationarity into the system. The driving forces par excellence are the fundamental trends, and the common long-term dynamic among all the variables in the system is generated by the non-stationary behaviour of T. If T is kept fixed, the ordinary variables X would not cointegrate.

To clarify consider the graph in Figure 3.6 and its VECM representation:

$$\Delta \begin{bmatrix} A \\ B \\ C \end{bmatrix}_t = \begin{bmatrix} 0 & 0 \\ a & 0 \\ 0 & b \end{bmatrix} \begin{bmatrix} c & 1 & 0 \\ 0 & d & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \end{bmatrix}_{t-1} + \varepsilon_t$$

The original source of non-stationarity is variable A. By fixing $A = A_0$, the dynamics of B reduces to:

$$\Delta B_t = a(cA_0 + B_{t-1}) + \varepsilon_{B,t}$$

Expanding ΔB_t, we obtain the following AR representation for B:

$$B_t = \mu + (a+1)B_{t-1} + \varepsilon_{B,t}$$

where $\mu = acA_0$. It follows from the stability conditions of the C-VAR model that $a + 1$ is less than 1 in absolute value. Hence, by fixing A, B becomes a stationary process.

By fixing A, C also loses its non-stationary behaviour, resulting in a representation for ΔC_t similar to the one illustrated for ΔB_t. The only difference is that the mean term μ is no longer constant, but it is a stochastic process with stationary behaviour.

This example provides an intuitive understanding of what driving trends should capture. Heuristically, driving trends are variables that, when fixed, cause the system to lose its non-stationary behaviour. In a fully observed model, identifying driving trends is straightforward, as it aligns with T, which are by construction weakly exogenous. However, extending these concepts to partially observed systems is less intuitive.

When only a subset X_o of the entire set of variables affecting a system is observed, the focus shifts from the variables generating non-stationarity to the variables that bring the non-stationary behaviour inside X_o. These variables are the ones directly or indirectly connected with T that cannot be made stationary by keeping other variables within X_o

constant.

Understanding the properties of variables that can be labeled as driving forces requires analyzing how non-stationarity is transmitted from the fundamental trends to the observed variables. Notably, fixing X_o does not render T stationary. This follows from the fact that the fundamental trends are inherently non-stationary, and their long-term dynamics do not depend on connections with other variables. All variables directly affected by T are non-stationary unless arbitrarily fixed. Their indirect descendants are non-stationary unless arbitrarily fixed or the indirect connection between T and the descendant is blocked by fixing the variables transmitting causality from T to the variable under analysis. Thus, the introduction of non-stationarity is related to the concept of blocked and unblocked forward paths between T and the observed variables. Specifically, a variable $x_i \in X_o$ introduces non-stationarity into the system if some of the paths between T and x_i remain unblocked by the observed variable X_o.

It should be noted that this definition does not align with the notion of weak causal exogeneity introduced in Chapter 1. It is indeed more stringent since not all the variables in ℵ introduce non-stationarity into X_o. A variable belongs to ⊐ if it meets two requirements: *i*) all paths with T must be blocked by X_o, and *ii*) there must be no feedback in the transmission of causality from ℵ to ⊐ through latent factors. The second requirement is not applicable when defining driving forces. Therefore, a variable must only meet the first requirement to be excluded from the set of driving trends. Consequently, the variables introducing non-stationarity within X_o are a subset of ℵ.

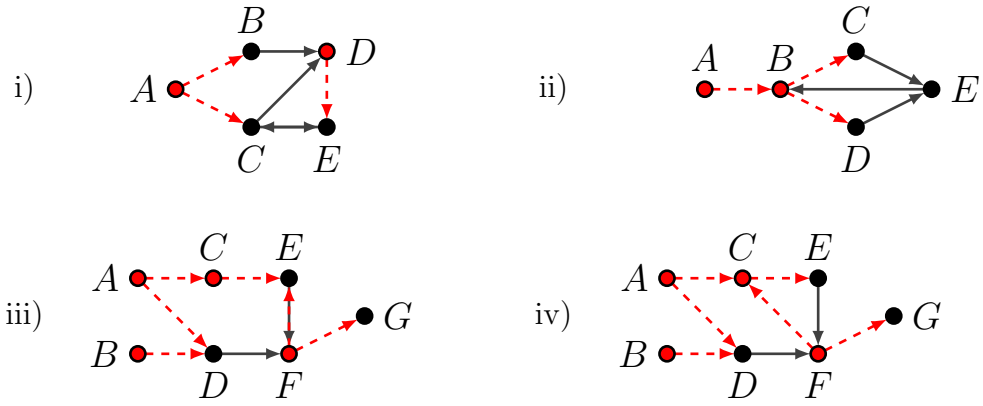

Figure 3.7: Causal Structures for Simulations

To clarify, consider the graphs in Figure 3.7. In graph i), the forward paths between $T = A$ and $\{B, C\}$ are direct, thus unblocked by X_o. Conversely, causality between A and E is mediated by B and C. By keeping C and E constant, the non-stationarity of B is unaffected. Specifically, ΔB reacts to follow the non-stationary fundamental trend A, which is unaffected by X_o. The same applies to C despite the causal feedback from E: even if E is kept constant, C remains non-stationary due to A's influence. Conversely, by fixing B and C, E becomes a stationary process, despite its influence from the unobserved D. This occurs because B and C block the forward paths from T and D, making D stationary. Consequently, E tracks a stationary process (D) and the constants B_0 and C_0.

A similar reasoning applies to graph ii). By fixing C and D, E remains constant despite causing B. This is because E adjusts to track a linear combination of B and C, which are constant in this hypothetical scenario. Conversely, both C and D maintain their non-stationarity even if E is held constant. Specifically, B remains non-stationary as it continues to track A, thereby transmitting non-stationarity to C and D.

In graphs *iii*) and *iv*), the variables whose forward paths with T are unblocked by X_o are D and E. Similar to the previous cases, despite the direct and indirect feedback of G on E, the latter is not made stationary by fixing the former. Moreover, G loses its long-term dynamic if both E and D are kept constant. This occurs because the variable driving its long-term behaviour (F) would become stationary by fixing E and D. These graphs are particularly interesting because they show instances where the variables introducing non-stationarity do not align with the set $\aleph$. The unobserved F transmits causality from $\{E, D\}$ to G and, in turn, affects E. This makes G fail the second requirement to be included in $\beth$, thus $G \in \aleph$. As mentioned earlier, this is an instance where the driving forces are a subset of $\aleph$, since E and D introduce the fundamental trends into X_o while $\aleph = \{D, E, G\}$.

To provide a more detailed definition of the concept of driving variables, we need the following partition of $\aleph$:

Definition 10 (***Set*** $\aleph_T$)**.** *A variable $x_i \in X_o$ belongs to the set $\aleph_T$ if at least one of the forward paths from T to x_i is not blocked by X_o*

Definition 11 (***Set*** $\aleph_B$)**.** *The set $\aleph_B$ is defined as $\aleph \setminus \aleph_T$*

We can now define what is meant by driving variables. The term "causal driving trend" will be used to distinguish the concept of leading variables based on the unobserved causal structure from the definition of driving trends based on the moving average representation of the VECM:

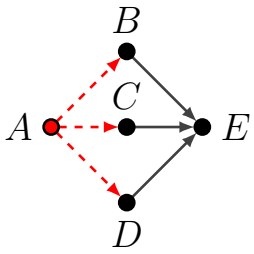

Figure 3.8: Causal Graph Generating a Misalignment Between Weak Exogeneity and Causal Driving Forces Example 1

Definition 12 (***Causal Driving Variables***)**.** *A variable $x_i \in X_o$ is a causal driving variable with respect to X_o if it belongs to the set $\aleph_T$.*

To understand the distinction between the standard and the causal concept of driving variables, we provide three different examples.

Consider the graph in Figure 3.8, translated into a VECM as follows:

$$\Delta \begin{bmatrix} A \\ B \\ C \\ D \\ E \end{bmatrix}_t = \begin{bmatrix} 0 & 0 & 0 & 0 \\ a & 0 & 0 & 0 \\ 0 & b & 0 & 0 \\ 0 & 0 & c & 0 \\ 0 & 0 & 0 & d \end{bmatrix} \begin{bmatrix} & & & & \\ e & 1 & 0 & 0 & 0 \\ f & 0 & 1 & 0 & 0 \\ g & 0 & 0 & 1 & 0 \\ 0 & h & i & j & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \\ D \\ E \end{bmatrix}_{t-1} + \varepsilon_t$$

Here, A is the only unobserved variable, as well as the sole fundamental trend. Its paths with B, C, and D are evidently not blocked by X_o, while the connection between A and E is fully mediated by B, C, and D. Consequently, the causal driving trends are B, C, and D.

Conversely, according to the definition based on the orthogonal complement of $\alpha_\perp$, the

driving trend is one and is retrieved as a linear combination of the residuals relative to $\aleph_T$. This can be observed by computing $\alpha_\perp$ using (3.4):

$$\alpha_{o\perp} = \begin{bmatrix} ae \\ bf \\ cg \\ 0 \end{bmatrix}$$

Thus, the driving trend $\alpha_\perp \varepsilon$ is:

$$\hat{T}_t = \begin{bmatrix} ae & bf & cg \end{bmatrix} \begin{bmatrix} \varepsilon_B \\ \varepsilon_C \\ \varepsilon_D \end{bmatrix}$$

By observing the difference between the causal and the standard definitions, we notice a different interpretation of what is meant by driving trend. In the standard interpretation, the trend does not have to be among the observed variables; it could be a linear combination of the residuals of some components in X_o. Conversely, in the causal interpretation, the set containing the driving forces is always a subset of X_o.

The standard approach estimates the unobserved T, while the causal approach focuses on identifying which variables in X_o are necessary to ensure the long-term dynamics. In fact, $\alpha_\perp \varepsilon$ results in only one variable, aligning with the cardinality of T, while the causal interpretation identifies three variables as fundamental trends. The focus of the causal

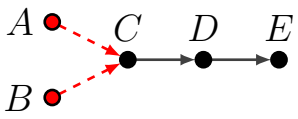

Figure 3.9: Causal Graph Generating a Misalignment Between the Number of Estimated Fundamental Trends and the Actual Cardinality of T

interpretation is on the variables in X_o leading the long-term dynamic, therefore the word driving variables would be more appropriate with respect to the term driving trends.

Despite the standard interpretation attempting to estimate the fundamental trend in T, it must be noted that the multiplication $\alpha_{o\perp}\varepsilon$ does not always produce a set $\hat{T}$ with a cardinality aligning with that of T. Consider, for instance, the graph in Figure 3.9 paired with the model below:

$$\Delta \begin{bmatrix} A \\ B \\ C \\ D \\ E \end{bmatrix}_t = \begin{bmatrix} 0 & 0 & 0 \\ 0 & 0 & 0 \\ a & 0 & 0 \\ 0 & b & 0 \\ 0 & 0 & c \end{bmatrix} \begin{bmatrix} d & e & 1 & 0 & 0 \\ 0 & 0 & f & 1 & 0 \\ 0 & 0 & 0 & g & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \\ D \\ E \end{bmatrix}_{t-1} + \varepsilon_t$$

Without loss of generality, we set $\omega_o = I$, so that the matrix α is equal to $N_{o\perp}$. It follows

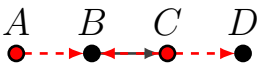

Figure 3.10: Causal Graph Generating a Misalignment Between Elements of $\aleph_T$ and Variables Included in The Standard Estimation of the Fundamental Trends

that one possible realization of $\alpha_\perp$ is:

$$\alpha_\perp = \begin{bmatrix} 1 \\ 0 \\ 0 \\ 0 \end{bmatrix}$$

and the driving trend is $\varepsilon_{o,C}$. Therefore, the standard definition of driving trends identifies just one driving force, despite the causal graph in Figure 3.9 indicates that the cardinality of T is 2.

It should also be noted that the term $\hat{T}$ is not always composed of linear combinations of the residuals corresponding to $\aleph_T$. To demonstrate this, consider the graph in Figure 3.10 together with its VECM representation below:

$$\Delta \begin{bmatrix} A \\ B \\ C \\ D \end{bmatrix}_t = \begin{bmatrix} 0 & 0 & 0 \\ a & 0 & 0 \\ 0 & b & 0 \\ 0 & 0 & c \end{bmatrix} \begin{bmatrix} d & 1 & e & 0 \\ 0 & f & 1 & 0 \\ 0 & 0 & g & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \\ D \\ E \end{bmatrix}_{t-1} + \varepsilon_t$$

By setting $X_o = \{B, D\}$, an admissible realization for $\alpha_{o\perp}$ is:

$$\alpha_{o\perp} = \begin{bmatrix} aeV_{CA} + adV_A \\ \\ cgV_{CA} \end{bmatrix}$$

where V_{CA} and V_A are both different from 0 and correspond, respectively, to the covariance between C and A and the variance of A conditioned to X_o.

We observe that here the trend resulting from the standard definition is a linear combination of the residuals corresponding to both B and D. Nevertheless, only B is a driving force according to the causal definition, since $D \notin \aleph_T$. In fact, by keeping D fixed, B will continue to be non-stationary due to the influence of the fundamental trend A. Conversely, it is sufficient to hold B constant to eliminate the long-term dynamics of C and, in turn, the one of D.

3.3.2 Non-Causality

The concept of non-causality is fundamental in causal inference. To provide a formal definition, we begin with a straightforward analysis involving two variables, A and B, connected by the directed edge $A \to B$. Here, B is designated as the caused variable, while A is considered the non-caused variable. Hence, in this context, non-causality is a property of the variable A.

The heuristic understanding of non-causality revolves around the absence of arrows pointing towards certain nodes in a graph. Therefore, a natural definition of non-causality

hinges on the lack of incoming edges. However, in the context of partially observed systems, non-causality must be defined relative to a subset of the nodes of the entire causal graph.

Let Y denote the set of variables corresponding to all nodes in the graph, and X_o represent the subset of variables under consideration. A variable $x_i \in X_o$ is deemed not-caused if there are no incoming edges from any other node within X_o to the node corresponding to x_i. Importantly, causal connections need not be direct; thus, non-causality has to be evaluated considering also the causal pathways mediated by latent components in Y (outside X_o).

Thus far, we have defined non-causality relative to a set X_o as a condition where there is a complete absence of influence from the other modeled components. However, variables within X_o may exhibit varying degrees of causal hierarchy based on their exogeneity. Thus, non-causality can also be defined with respect to a subset of X_o, for instance with respect to the subset of X_o not including the driving variables. This approach has also been used for other causal definitions. For example, in the causal heuristic definition of exogeneity proposed by Cohen et al. (2013), exogeneity implies that a variable is not caused by any other variable within the system that is not itself exogenous. This intuitive notion of exogeneity can be extended to the concept of non-causality, suggesting that a variable is not-caused if it is neither directly nor indirectly influenced by other components within X_o, except for those considered driving forces.

Building upon these principles, we can delineate a strong and a weak form of non-causality:

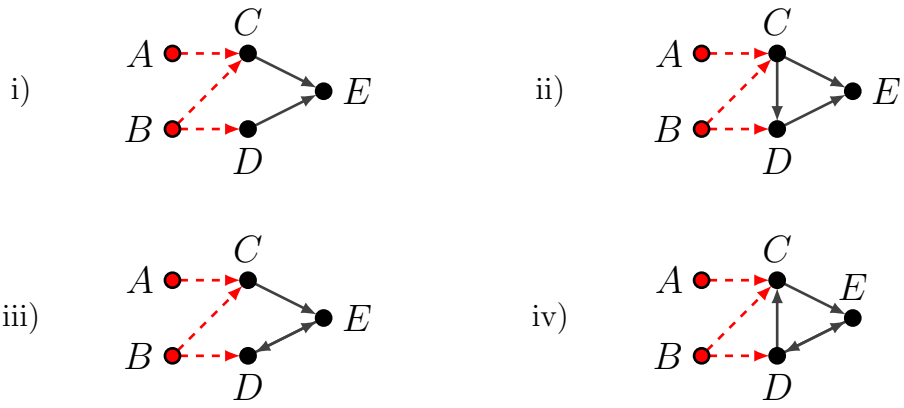

Figure 3.11: Causal Structures to Illustrate Non-Causality

Definition 13 (***Strong Non-Causality***)**.** *A variable $x_i \in X_o$ is said to be strongly not-caused with respect to X_o if there are no direct or indirect incoming edges starting from the nodes relative to X_o and arriving to x_i*

Definition 14 (***Weak Non-Causality***)**.** *A variable $x_i \in \aleph_T$ is said to be weakly not-caused with respect to X_o if there are not direct or indirect incoming edges starting from the nodes relative to $X_o \setminus \aleph_T$ and arriving to x_i*

To clarify the definitions, we examine the causal graphs depicted in Figure 3.11, where the set of interest is defined as $X_o = \{C, D, E\}$ in all the four graphs.

In graph *i*), both C and D lack incoming arrows from other components of X_o, thereby qualifying them as strongly not-caused with respect to X_o. Conversely, E does not meet the criteria for being strongly or weakly not-caused, as $E \notin \aleph_T$ and both B and C directly affect E.

Graph *ii*) introduces a causal link from C to D, resulting in D now having an incoming edge originating from an element in X_o. Consequently, D no longer qualifies as strongly

not-caused. However, both C and D are directly influenced by fundamental trends, placing them within $\aleph_T$. Thus, according to Definition 14, D is categorized as weakly not-caused.

In graph *iii*), D is caused by E. Given that $E \notin \aleph_T$, D can no longer be classified as weakly not-caused.

Finally, in graph *iv*), a causal link from D to C is introduced. Since $E \notin \aleph_T$ causes D, which subsequently affects C, there exists indirect causality from E to C. Consequently, none of the variables in X_o can be deemed not-caused, either strongly or weakly.

3.4 Driving Trends and Weak Exogeneity

This section examines the relationship between weak exogeneity and trend introduction by studying whether and how testing restrictions of the adjustment matrix can identify variables that introduce non-stationarity among observed variables.

As discussed in the previous section, we consider the variables in $\aleph_T$ as the components of X_o introducing non-stationarity coming from the latent fundamental trends, thereby forming the driving trends under analysis. The goal is to determine whether a zero row in the adjustment matrix can be associated only with variables belonging to $\aleph_T$.

Before presenting the main results, we introduce intermediate findings:

Lemma 3 (***Weak Exogeneity and Causal Endogeneity***)**.** *The rows in α_o corresponding to $\beth$ cannot be null vectors*

Proof of Lemma 3. This lemma follows directly from Corollary 2 in Chapter 1. Weak exogeneity is a sufficient condition for weak causal exogeneity; thus, variables corresponding to a null row in α_o cannot belong to ב. □

From Lemma 3, it follows that weak exogeneity cannot be a property of the components of ב. Consequently, weakly exogenous variables must be in א. However, not all variables in א are components of the driving trends. Thus, we need to further explore the connection between restrictions of α_o and the sets $א_T$ and $א_B$.

As described in previous chapters, the elements in א are influenced by components of T, ד, ב, and א itself. In contrast, ב is unaffected by ד and T. Additionally, the sub-matrix of V_{uT} corresponding to the rows of ל comprises solely null entries (see Lemma 1). Therefore, the structure of $M_{ou}V_{uT} + N_oV_T$ is:

$$\begin{array}{c} \\ |א| \\ |ב| \end{array}\begin{array}{c} \begin{array}{cc} |ד| & |ל| \end{array} \\ \begin{bmatrix} \boldsymbol{\mathcal{A}} & \mathbf{0} \\ \mathbf{0} & \boldsymbol{\mathcal{C}} \end{bmatrix} \end{array}\begin{array}{c} \begin{array}{c} |T| \end{array} \\ \begin{array}{c} |ד| \\ |ל| \end{array}\begin{bmatrix} \boldsymbol{\mathcal{E}} \\ \mathbf{0} \end{bmatrix} \end{array} + \begin{array}{c} \begin{array}{c} |T| \end{array} \\ \begin{array}{c} |א| \\ |ב| \end{array}\begin{bmatrix} \boldsymbol{\mathcal{D}} \\ \mathbf{0} \end{bmatrix} \end{array} |T|\begin{bmatrix} \boldsymbol{\mathcal{F}} \end{bmatrix}^{|T|} = \begin{bmatrix} \boldsymbol{\mathcal{AE}} + \boldsymbol{\mathcal{DF}} \\ \mathbf{0} \end{bmatrix} \tag{3.6}$$

The non-zero sub-matrix $\boldsymbol{\mathcal{AE}} + \boldsymbol{\mathcal{DF}}$ represents the product between the coefficients linking the elements in א to their parents and the covariance between them and the fundamental trends. Given that X_o does not interfere with fundamental trends or block paths between ד and T, each row of $\boldsymbol{\mathcal{E}}$ and $\boldsymbol{\mathcal{F}}$ will contain at least one non-zero entry. Hence, every א component is linked to at least one variable in ד or T, ensuring that $\begin{bmatrix} \boldsymbol{\mathcal{A}} & \boldsymbol{\mathcal{D}} \end{bmatrix}$ contains at least one non-zero entry per row, as well as $\boldsymbol{\mathcal{AE}} + \boldsymbol{\mathcal{DF}}$. This is naturally true since

every element in ℵ must be either directly or indirectly connected to T. Similarly, every element in T must be connected to at least one element in ℵ. If otherwise, ℵ and some T components would belong to disjoint graphs, implying different systems. Therefore, the columns of $\boldsymbol{\mathcal{AE}} + \boldsymbol{\mathcal{DF}}$ must also have at least one non-zero entry.

The values of $\boldsymbol{\mathcal{A}}$ are independent of $\boldsymbol{\mathcal{D}}$, so linear dependence between rows or columns of $\boldsymbol{\mathcal{AE}}+\boldsymbol{\mathcal{DF}}$ occurs only through parameter fine-tuning, which we exclude. Thus, $\boldsymbol{\mathcal{AE}}+\boldsymbol{\mathcal{DF}}$ is a full-rank matrix with rank $\rho = \min(|ℵ|, |T|)$.

These conclusions lead to the following lemma:

Lemma 4 (***Lower Bound for Cointegration Rank***)**.** *The cointegration rank r cannot be less than the cardinality of* ב.

Proof of Lemma 4. As proved in the first Chapter, the cointegration rank r equates the rank of the matrix γ. Given that $\gamma = \left(M_{ou}V_{uT} + N_oV_T\right)_\perp$, and $\rho = \text{Rank}(M_{ou}V_{uT} + N_oV_T)$, by definition γ is an $|X_o| \times \left(|X_o| - \rho\right)$ full rank matrix.

If $|ℵ| > |T|$, then $\rho = |T|$ and γ is an $|X_o| \times \left(|X_o| - |T|\right)$ of rank $|X_o| - |T|$. Since $|X_o| = |ℵ| + |ב|$, r exceeds $|ב|$.

Alternatively, if $|ℵ| \leq |T|$, then $\rho = |ℵ|$ and γ is an $|X_o| \times \left(|X_o| - |ℵ|\right)$ of rank $|X_o| - |ℵ|$. Since $|X_o| = |ℵ| + |ב|$, the rank r is equal to $|ב|$.

This proves the Lemma □

It is now possible to delineate a necessary condition for weak exogeneity:

Lemma 5 (***Necessary Condition for Weak Exogeneity***)**.** *A necessary condition for the variables in* $\aleph$ *to be weakly exogenous is that the cardinality of* $\beth$ *does not exceed the cointegration rank* r*.*

Proof of Lemma 5. According to equation (3.2), the adjustment matrix α_o is given by $\Sigma_o(M_{ou}V_{uT} + N_oV_T)_\perp$, represented in block form as:

$$\alpha_o = \begin{array}{c} \\ |\aleph| \\ |\beth| \end{array}\begin{array}{c} \begin{array}{cc} |\aleph| & |\beth| \end{array} \\ \begin{bmatrix} \boldsymbol{\mathcal{I}} & \boldsymbol{J} \\ \boldsymbol{J'} & \mathcal{K} \end{bmatrix} \end{array}\begin{array}{c} \begin{array}{c} |r| \end{array} \\ \begin{bmatrix} \boldsymbol{\mathcal{L}} \\ \boldsymbol{M} \end{bmatrix} \end{array} = \begin{bmatrix} \boldsymbol{\mathcal{I}}\boldsymbol{\mathcal{L}} + \boldsymbol{\mathcal{J}}\boldsymbol{\mathcal{M}} \\ \boldsymbol{\mathcal{J}'}\boldsymbol{\mathcal{L}} + \mathcal{K}\boldsymbol{\mathcal{M}} \end{bmatrix}$$

The matrix $\boldsymbol{\mathcal{I}}$ is the variance of the residuals corresponding to $\aleph$. Consequently it is a full rank square matrix, thus a necessary condition for weak exogeneity of $\aleph$ is that $\boldsymbol{\mathcal{L}} = 0$. This follows from the fact that for $\boldsymbol{\mathcal{I}}\boldsymbol{\mathcal{L}} \neq 0$, the equality $\boldsymbol{\mathcal{I}}\boldsymbol{\mathcal{L}} = -\boldsymbol{\mathcal{J}}\boldsymbol{\mathcal{M}}$ hold only for a fine tuning of the parameters, which we find reasonable to exclude.

To prove the lemma, it must be shown that if $|\beth| < r$, then $\boldsymbol{\mathcal{L}} \neq 0$.

The matrix $(M_{ou}V_{uT} + N_oV_T)_\perp$ finds the following representation:

$$(M_{ou}V_{uT} + N_oV_T)_\perp = \begin{array}{c} \\ |\aleph| \\ |\beth| \end{array}\begin{array}{c} \begin{array}{c} |T| \end{array} \\ \begin{bmatrix} \boldsymbol{\mathcal{A}}\boldsymbol{\mathcal{E}} + \boldsymbol{\mathcal{D}}\boldsymbol{\mathcal{F}} \\ \mathbf{0} \end{bmatrix}_\perp \end{array}$$

We will prove the lemma by contradiction, considering $(M_{ou}V_{uT} + N_oV_T)_\perp$ as:

$$(M_{ou}V_{uT} + N_oV_T)_\perp = \begin{matrix} \\ |\aleph| \\ |\beth| \end{matrix} \begin{matrix} r \\ \begin{bmatrix} \mathbf{0} \\ \boldsymbol{\mathcal{H}} \end{bmatrix} \end{matrix} \tag{3.7}$$

Under the scenario considered by the Lemma, the cointegration rank r exceeds $|\beth|$, the maximum rank of $\boldsymbol{\mathcal{H}}$ is $|\beth|$. It follows that the maximum rank of the block representation (3.7) is $|\beth|$, implying $(M_{ou}V_{uT}+N_oV_T)_\perp$ has reduced rank given that $\big(|\aleph|+|\beth|\big) > r > |\beth|$. This leads to a contradiction since the orthogonal complement is, by construction, a full-rank matrix.

□

To draw conclusions on $\aleph_T$ and $\aleph_B$, it is instructive to analyse the connection between the presence of $\aleph_B$ and the condition $r \leq \beth$:

Lemma 6 (***Relation Between Cointegration Rank and*** $|\beth|$ ***when*** $\aleph_T$ ***Diverges from*** $\aleph$)**.** *If* $\aleph_B$ *is not empty, it follows that* $r > |\beth|$.

Proof of Lemma 6*.* This lemma can be proved by analyzing the relation between $\aleph_B$ and the cointegration rank through Equation (3.3). The idea is to study the dimension of the cointegration matrix since the number of columns of β_o, thus of $(N_o - M_{ou}M_u^{-1}N_u)_\perp$, equals the cointegration rank.

Define $\gimel_B$ as the subset of $\gimel$ whose forward paths with T are blocked by $\aleph_T$, and $\gimel_T = \gimel \setminus \gimel_B$. By construction, the fundamental trends cannot directly cause $\aleph_B$ and $\gimel_B$, as well

as ל and ב. Therefore,

$$N_o = \begin{array}{c} \\ |\aleph_T| \\ |\aleph_B| \\ |\beth| \end{array} \begin{array}{c} |T| \\ \left[\begin{array}{c} \boldsymbol{\mathcal{N}} \\ \mathbf{0} \\ \mathbf{0} \end{array}\right] \end{array} \qquad N_u = \begin{array}{c} \\ |\daleth_T| \\ |\daleth_B| \\ |\gimel| \end{array} \begin{array}{c} |T| \\ \left[\begin{array}{c} \boldsymbol{\mathcal{O}} \\ \mathbf{0} \\ \mathbf{0} \end{array}\right] \end{array} \tag{3.8}$$

The unobserved ל, can exclusively cause ב, while $\daleth_B$ can cause, $\aleph_T$, $\aleph_B$ and $\daleth_T$. Conversely, $\daleth_T$ can have influence solely on $\aleph_T$. Consequently, the matrices M_{ou} and M_u are

$$M_{ou} = \begin{array}{c} \\ |\aleph_T| \\ |\aleph_B| \\ |\beth| \end{array} \begin{array}{c} |\daleth_T| \quad |\daleth_B| \quad |\gimel| \\ \left[\begin{array}{ccc} \boldsymbol{\mathcal{P}} & \boldsymbol{\mathcal{Q}} & \mathbf{0} \\ \mathbf{0} & \boldsymbol{\mathcal{R}} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \boldsymbol{\mathcal{S}} \end{array}\right] \end{array} \qquad M_u = \begin{array}{c} \\ |\daleth_T| \\ |\daleth_B| \\ |\gimel| \end{array} \begin{array}{c} |\daleth_T| \quad |\daleth_B| \quad |\gimel| \\ \left[\begin{array}{ccc} \boldsymbol{\mathcal{T}} & \boldsymbol{\mathcal{U}} & \mathbf{0} \\ \mathbf{0} & \boldsymbol{\mathcal{V}} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \boldsymbol{\mathcal{W}} \end{array}\right] \end{array} \tag{3.9}$$

It can be noted that the matrices $\boldsymbol{\mathcal{T}}$, $\boldsymbol{\mathcal{V}}$, and $\boldsymbol{\mathcal{W}}$ are full rank matrices since they related the causal sets with themselves. Therefore $\boldsymbol{\mathcal{T}}^{-1}$, $\boldsymbol{\mathcal{V}}^{-1}$, and $\boldsymbol{\mathcal{W}}^{-1}$ exist and the matrix M_u^{-1} is as follows:

$$M_u^{-1} = \begin{array}{c} \\ |\daleth_T| \\ |\daleth_B| \\ |\gimel| \end{array} \begin{array}{c} |\daleth_T| \qquad |\daleth_B| \qquad |\gimel| \\ \left[\begin{array}{ccc} \boldsymbol{\mathcal{T}}^{-1} & \boldsymbol{\mathcal{T}}^{-1}\boldsymbol{\mathcal{U}}\boldsymbol{\mathcal{V}}^{-1} & \mathbf{0} \\ \mathbf{0} & \boldsymbol{\mathcal{V}}^{-1} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \boldsymbol{\mathcal{W}}^{-1} \end{array}\right] \end{array} \tag{3.10}$$

Substituting (3.8)-(3.10) into Equation (3.3), we obtain:

$$N_o - M_{ou}M_u^{-1}N_u = \begin{array}{r} \\ |ך_T| \\ |ך_B| \\ |ל| \end{array} \begin{array}{c} |T| \\ \left[\begin{array}{c} \boldsymbol{\mathcal{X}} \\ \mathbf{0} \\ \mathbf{0} \end{array}\right. \end{array}$$

From the representation above, it is evident that the maximum rank of $\boldsymbol{\mathcal{X}}$ is the minimum between the cardinality of $\aleph_T$ and the cardinality of T. It follows that the number of columns of $(N_o - M_{ou}M^{-1}N_u)_\perp$, i.e. cointegration rank, is equal to $|\aleph_T| + |\aleph_B| + |ב| - \min(|T|, |\aleph_T|)$.

Suppose $|\aleph_T| > |T|$, thus $r = |\aleph_T| + |\aleph_B| + |ב| - |T|$. Given that $|\aleph_T| > |T|$, it must hold that $|\aleph| > |T|$. Therefore $|\aleph| + |ב| - |T| > |ב|$.

If $|\aleph_T| < |T|$, the cointegration rank is $r = |\aleph_T| + |\aleph_B| + |ב| - |\aleph_T|$. Thus, the only possibility for $|ב|$ to be equal to r is that $|\aleph_B| = 0$.

This proves the Lemma.

□

The role of weak exogeneity in the identification of $\aleph_T$ can now be analysed:

Theorem 4 (***Weak Exogeneity as Identifier of Driving Variables***)**.** *Weak exogeneity is a sufficient condition for a variable to be among the components introducing non-stationarity.*

Proof of Theorem 4. To prove this theorem, it is sufficient to show that the variables

in $\beth$ and $\aleph_B$ cannot be weakly exogenous.

From Lemma 3, we can exclude weak exogeneity for the variables in $\beth$. From Lemma 5, we can exclude weak exogeneity for systems where $r > |\beth|$, while Lemma 6 demonstrates that the cointegration rank cannot be lower or equal to $|\beth|$ if $\aleph_B$ is not empty. It follows that only the variables in $\aleph_T$ can be weakly exogenous.

This concludes the proof.

□

Corollary 8 (***Corollary to Theorem 4***)**.** *Weak exogeneity is not a necessary condition for a variable to be among the components introducing non-stationarity.*

The proof of this corollary is omitted, as the example in section 3.3.2 is sufficient to demonstrate that absence of weak exogeneity can coexist with the condition of belonging to the set $\aleph_T$.

Theorem 4 is fundamental as it clarifies that weak exogeneity can be utilized for the identification of driving forces. However, as stated in the corollary, not all the variables in $\aleph_T$ correspond to a zero row in α_o. Consequently, identifying the driving trends by means of tests on α_o requires caution. Failure to reject the null hypothesis of weak exogeneity aligns with failure to reject the hypothesis that the corresponding variable is a driving force. Conversely, rejection of weak exogeneity does not necessarily imply that a variable does not introduce non-stationarity within the system.

The following theorem clarifies the conditions ensuring that weak exogeneity corresponds

to the variables in א:

Theorem 5 (***Conditions for the Alignment Between Weak Exogeneity and Driving Trends***)**.** *The variables in* א *are jointly weakly exogenous if and only if* $r = |\text{ב}|$.

Proof of Theorem 5. According to lemma 4, the minimum value for r is $|\text{ב}|$, therefore we can focus only on $r \geq |\text{ב}|$.

As per Equation (3.2), the adjustment matrix is equal to $\Sigma_o(M_{ou}V_{uT} + N_oV_T)_\perp$. The proof starts with the analysis of the matrix Σ_o, retrieved as per Equation (3.1). By definition, ל does not affect א, while T and ד do not cause ב. Moreover, the covariance paths between ד$\cup T$ and ל are all blocked by X_o. In light of these considerations, the matrix Σ_o becomes

$$\Sigma_o = \begin{array}{c} \\ |\text{א}| \\ |\text{ב}| \end{array}\!\!\begin{array}{c} \begin{array}{ccc} |\text{ד}| & |\text{ל}| & |T| \end{array} \\ \begin{bmatrix} \boldsymbol{\mathcal{A}} & \mathbf{0} & \boldsymbol{\mathcal{D}} \\ \mathbf{0} & \boldsymbol{\mathcal{C}} & \mathbf{0} \end{bmatrix} \end{array} \begin{array}{c} \\ |\text{א}| \\ |\text{ב}| \\ |T| \end{array}\!\!\begin{array}{c} \begin{array}{ccc} |\text{ד}| & |\text{ל}| & |T| \end{array} \\ \begin{bmatrix} \boldsymbol{\mathcal{J}} & \mathbf{0} & \boldsymbol{\mathcal{E}} \\ \mathbf{0} & \boldsymbol{\mathcal{L}} & \mathbf{0} \\ \boldsymbol{\mathcal{E}}' & \mathbf{0} & \boldsymbol{\mathcal{F}} \end{bmatrix} \end{array} \begin{array}{c} \\ |\text{א}| \\ |\text{ב}| \\ |T| \end{array}\!\!\begin{array}{c} \begin{array}{cc} |\text{ד}| & |\text{ל}| \end{array} \\ \begin{bmatrix} \boldsymbol{\mathcal{A}}' & \mathbf{0} \\ \mathbf{0} & \boldsymbol{\mathcal{C}}' \\ \boldsymbol{\mathcal{D}}' & \mathbf{0} \end{bmatrix} \end{array} + \Omega_o$$

which equals a matrix with a diagonal block structure:

$$\Sigma_o = \begin{array}{c} \\ |\text{א}| \\ |\text{ב}| \end{array}\!\!\begin{array}{c} \begin{array}{cc} |\text{א}| & |\text{ב}| \end{array} \\ \begin{bmatrix} \boldsymbol{\mathcal{I}} & \mathbf{0} \\ \mathbf{0} & \boldsymbol{\mathcal{K}} \end{bmatrix} \end{array}$$

It follows that α_o is:

$$\alpha_o = \begin{matrix} \\ |\aleph| \\ |\beth| \end{matrix} \begin{matrix} \begin{matrix} |\aleph| & |\beth| \end{matrix} \\ \begin{bmatrix} \boldsymbol{\mathcal{I}} & \mathbf{0} \\ \mathbf{0} & \boldsymbol{\mathcal{K}} \end{bmatrix} \end{matrix} \begin{matrix} \begin{matrix} |r| \end{matrix} \\ \begin{matrix} |\aleph| \\ |\beth| \end{matrix} \begin{bmatrix} \boldsymbol{\mathcal{L}} \\ \boldsymbol{M} \end{bmatrix} \end{matrix} = \begin{bmatrix} \boldsymbol{\mathcal{I}}\boldsymbol{\mathcal{L}} \\ \boldsymbol{\mathcal{K}}\boldsymbol{\mathcal{M}} \end{bmatrix}$$

Given that $\boldsymbol{\mathcal{I}}$ cannot be zero, to prove the "if" part, it is necessary to show that when $r = |\beth|$, it follows that $\boldsymbol{\mathcal{L}} = 0$.

The $\left(|\aleph| + |\beth|\right) \times r$ full-rank matrix of interest is obtained by computing the orthogonal complement of the $\left(|\aleph| + |\beth|\right) \times |T|$ matrix $M_{ou}V_{uT} + N_oV_T$.

By construction, r is equal to $|\aleph| + |\beth| - \text{Rank}(M_{ou}V_{uT} + N_oV_T)$. Lemma 4 establishes that the minimum value for r is $|\beth|$, thereby focusing the Theorem on instances where $\text{Rank}(M_{ou}V_{uT} + N_oV_T) = |\aleph|$. Consequently, the non-null matrix $\boldsymbol{\mathcal{A}}\boldsymbol{\mathcal{E}} + \boldsymbol{\mathcal{D}}\boldsymbol{\mathcal{F}}$ in Equation (3.6) is a $|\aleph| \times |T|$ matrix with rank $|\aleph|$, where $|T|$ is necessarily greater than or equal to $|\aleph|$.

According to the Rank-Nullity Theorem, under this scenario, the nullity of $\left(\boldsymbol{\mathcal{A}}\boldsymbol{\mathcal{E}} + \boldsymbol{\mathcal{D}}\boldsymbol{\mathcal{F}}\right)'$ is 0, while both the number of columns and the rank of the matrix are $|\aleph|$.

As the nullity is 0, the null space of $\left(\boldsymbol{\mathcal{A}}\boldsymbol{\mathcal{E}} + \boldsymbol{\mathcal{D}}\boldsymbol{\mathcal{F}}\right)'$ is zero-dimensional, implying that the only vector $\boldsymbol{\delta}$ ensuring that $\left(\boldsymbol{\mathcal{A}}\boldsymbol{\mathcal{E}} + \boldsymbol{\mathcal{D}}\boldsymbol{\mathcal{F}}\right)'\boldsymbol{\delta} = \mathbf{0}$ is the trivial vector. Thus, the orthogonal complement has dimension 0, and the only solution for $\left(M_{ou}V_{uT} + N_oV_T\right)_\perp$ is

$$\begin{array}{c} \\ |\aleph| \\ |\beth| \end{array} \begin{array}{c} r = |\beth| \\ \left[\begin{array}{c} \mathbf{0} \\ \boldsymbol{\mathcal{H}} \end{array}\right] \end{array} \tag{3.11}$$

$$\left(M_{ou}V_{uT} + N_oV_T\right)_\perp =$$

where $\boldsymbol{\mathcal{H}}$ is any full-rank matrix.

This concludes the proof of the "if" part.

To prove the "only if" part, it is necessary to include the findings of Lemma 5.

This concludes the proof of the Theorem.

□

From Theorem 5, we can deduce that the components of $\aleph_T$ can be identified when the cointegration rank matches the cardinality of $\beth$. It follows that the weak exogeneity test can be used to identify $\aleph_T$ if the estimated rank is equal to the cardinality of $X_o \setminus \Upsilon$, where Υ is the arbitrarily selected set for which belonging to $\aleph_T$ is under testing.

3.5 Weak Exogeneity and Non Causality

This section explores the relationship between constraints on the adjustment matrix and non-causality. As defined in section 3.3, non-causality manifests in both weak and strong forms. Strong non-causality refers to variables without direct or indirect connections from other components in X_o. Conversely, weak non-causality pertains to the absence of direct and indirect influences from $\beth \cup \aleph_B$ to $\aleph_T$.

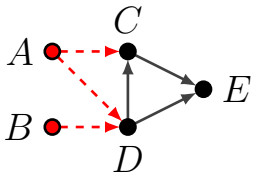

Figure 3.12: Causal Graph Illustrating How Variables That Are Not Strongly Not-Caused Can Be Weakly Exogenous

We commence by examining strong non-causality to ascertain any relationship with weak exogeneity. Consider the data generating process given by the VECM below, depicted in the causal graph in Figure 3.12:

$$\Delta \begin{bmatrix} A \\ B \\ C \\ D \\ E \end{bmatrix}_t = \begin{bmatrix} 0 & 0 & 0 \\ 0 & 0 & 0 \\ a & 0 & 0 \\ 0 & b & 0 \\ 0 & 0 & c \end{bmatrix} \begin{bmatrix} d & 0 & 1 & e & 0 \\ f & g & 0 & 1 & 0 \\ 0 & 0 & h & i & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \\ D \\ E \end{bmatrix}_{t-1} + \varepsilon_t$$

Setting $X_o = \{C, D, E\}$, we observe $T = Y_u = \{A, B\}$, $\aleph = \aleph_T = \{C, D\}$, and $\beth = \{E\}$. Notably, all forward paths from T to E are blocked by $\{C, D\}$, and causality from this group to E is direct.

Within X_o, the only node without incoming edges is the one relative to D. Despite $C \in \aleph_T$, there exists a direct edge from D to C.

To explore the relationship between weak exogeneity and strong non-causality, we compute the adjustment matrix α_o defined in Equation (3.2). Given that only the fundamental trends are unobserved, Equation (3.2) can be simplified as per Equation (3.4). Without

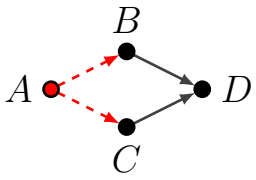

Figure 3.13: Causal Graph Illustrating How Variables That Are Strongly Not-Caused Are Not Necessarily Weakly Exogenous

loss of generality, we set Ω_o to the identity, so $\alpha_o = N_{o\perp}$:

$$\alpha_o = \begin{bmatrix} ad & 0 \\ bf & bg \\ 0 & 0 \end{bmatrix}_{\perp} = \begin{bmatrix} 0 \\ 0 \\ x \end{bmatrix}$$

where x is any non-null real number.

It follows from the representation of α_o that both C and D are weakly exogenous, though only D is strongly non-caused. This observation motivates the following lemma:

Lemma 7 (***Weak Exogneity and Strong Non-Causality (1)***)**.** *Weak exogeneity is not a sufficient condition for strong non-causality*

The lemma asserts that the association between weak exogeneity and strong non-causality is flawed, as absence of strong non-causality can coexist with weak exogeneity.

To ascertain whether weak exogeneity is a necessary condition for strong non-causality, consider the example illustrated in section 3.2, depicted in Figure 3.13 together with its VECM representation below:

$$\Delta \begin{bmatrix} A \\ B \\ C \\ D \end{bmatrix}_t = \begin{bmatrix} 0 & 0 & 0 \\ a & 0 & 0 \\ 0 & b & 0 \\ 0 & 0 & c \end{bmatrix} \begin{bmatrix} d & 1 & 0 & 0 \\ e & 0 & 1 & 0 \\ 0 & f & g & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \\ D \end{bmatrix}_{t-1} + \varepsilon_t$$

By setting $X_o = \{B, C, D\}$, we identify $\aleph = \aleph_T = \{B, C\}$ and $\beth = \{D\}$. Both B and D are strongly not-caused by the components of X_o. In fact, none of the elements in X_o directly or indirectly influence B and C.

As for the previous example, we set $\Omega_o = I$ so that $\alpha_o = N_{o\perp}$. The resulting adjustment matrix is

$$\alpha_o = \begin{bmatrix} ad \\ be \\ 0 \end{bmatrix}_\perp = \begin{bmatrix} -xbe & 0 \\ xad & 0 \\ 0 & y \end{bmatrix}$$

where both x and y are any non-null real number.

In this scenario, none of the components in X_o exhibit weak exogeneity, although both B and C are strongly not-caused. This observation leads to the following lemma:

Lemma 8 (***Weak Exogneity and Strong Non-Causality (2)***)**.** *Weak exogeneity is not a necessary condition for strong non-causality*

This lemma highlights that the absence of weak exogeneity does not imply the absence of strong non-causality, as instances of strong non-causality may still coexist with non-zero rows in α_o.

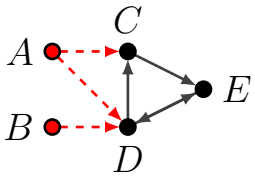

Figure 3.14: Causal Graph Illustrating How Variables That Are Not Weakly Not-Caused Can Be Weakly Exogenous

So far, we demonstrated that testing restrictions on α_o is completely uninformative with respect to strong non-causality. We now study whether weak exogeneity can be associated with weak non-causality.

From Lemma 8, we deduce:

Lemma 9 (***Weak Exogneity and Weak Non-Causality (1)***)**.** *Weak exogeneity is not a necessary condition for weak non-causality*

Weak non-causality represents a particular instance of strong non-causality, thereby implying that the coexistence of strong non-causality with the absence of weak exogeneity generates a parallel coexistence with weak non-causality.

To investigate if weak exogeneity suffices as a condition for weak non-causality, consider the causal graph in Figure 3.14 and its corresponding VECM representation:

$$\Delta \begin{bmatrix} A \\ B \\ C \\ D \\ E \end{bmatrix}_t = \begin{bmatrix} 0 & 0 & 0 \\ 0 & 0 & 0 \\ a & 0 & 0 \\ 0 & b & 0 \\ 0 & 0 & c \end{bmatrix} \begin{bmatrix} d & 0 & 1 & e & 0 \\ f & g & 0 & 1 & h \\ 0 & 0 & i & j & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \\ D \\ E \end{bmatrix}_{t-1} + \varepsilon_t$$

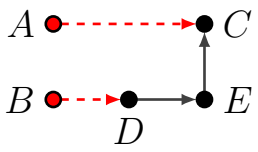

Figure 3.15: Causal Graph Illustrating How Variables That Are Not Weakly Not-Caused Can Be Weakly Exogenous Even in Absence of Cyclicality

In this system, the causal structure resembles that of Figure 3.12, with $T = \{A, B\}$, $\aleph = \aleph_T = \{C, D\}$, and $\beth = \{E\}$. The difference stand on the introduction of a causal feedback between E and D. Since $E \in \beth \to D$ which in turn causes C, no variables in X_o exhibit non-causality in both weak and strong form.

With $\Omega_o = I$, the resulting adjustment matrix is:

$$\alpha_o = \begin{bmatrix} ad & 0 \\ bf & bg \\ 0 & 0 \end{bmatrix}_{\perp} = \begin{bmatrix} 0 \\ 0 \\ x \end{bmatrix}$$

Thus, both C and D are classified as weakly exogenous, despite none of the components of X_o being weakly not-caused. This observation leads to the following lemma:

Lemma 10 (***Weak Exogneity and Weak Non-Causality (2)***)**.** *Weak exogeneity is not a sufficient condition for weak non-causality*

This lemma emphasizes that the relationship between weak exogeneity and weak non-causality is flawed, as the rows in α_o corresponding to weakly not-caused variables are not necessarily null vectors.

Lemma 10 holds even under the assumption of acyclical behaviour. Consider the acyclical graph depicted in Figure 3.15, with its corresponding VECM representation:

$$\Delta \begin{bmatrix} A \\ B \\ C \\ D \\ E \end{bmatrix}_t = \begin{bmatrix} 0 & 0 & 0 \\ 0 & 0 & 0 \\ a & 0 & 0 \\ 0 & b & 0 \\ 0 & 0 & c \end{bmatrix} \begin{bmatrix} d & 0 & 1 & 0 & e \\ 0 & f & 0 & 1 & 0 \\ 0 & 0 & 0 & g & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \\ D \\ E \end{bmatrix}_{t-1} + \varepsilon_t$$

By setting $X_o = \{C, D, E\}$, it results $T = Y_u = \{A, B\}$, $\aleph = \aleph_T = \{C, D\}$, and $\beth = \{E\}$. Notably, the causal graph does not depict any cyclical behaviour, so it is a directed acyclic graph (DAG). Under this setting, $C \in \aleph_T$ is influenced by $E \in \beth$, consequently C it is not weakly not caused.

Upon computing the adjustment matrix, we obtain:

$$\alpha_o = \begin{bmatrix} ad & 0 \\ bf & bg \\ 0 & 0 \end{bmatrix}_\perp = \begin{bmatrix} 0 \\ 0 \\ x \end{bmatrix}$$

Here, both C and D are weakly exogenous, despite C not being weakly not-caused.

In conclusion, this subsection illustrates that weak exogeneity fails to provide informative insights into non-causality. Consequently, employing weak exogeneity tests to infer the

absence of influences can lead to misleading conclusions, even under the assumption of acyclical behaviour, which is a hypothesis commonly employed in the context of causal inference.

3.6 Simulations

This section analyses the practical application of the results discussed in sections 3.4 and 3.5. Specifically, it investigates the test performance under various causal structures, following the implications of the Lemmas and Theorems. The aim is to determine whether specific forms of non-causality or structures of the set $\aleph_T$ impact the test performance both in terms of power and size for a given sample size.

Subsection 3.6.1 lists the causal graphs used for the simulations and describes their causal properties. Subsection 3.6.2 then reports and comments on the simulation outcomes.

3.6.1 Data Generating Processes

Figure 3.16 illustrates the causal structures employed in the data-generating process of the simulated time-series. Red nodes and red dotted edges denote unobserved variables and latent causal paths, respectively, while observed variables are depicted in black.

Structures *i*) and *ii*) represent graphs with behaviours similar to causal chains. The primary distinction between *i*) and *ii*) is the introduction of a direct causal relationship between A and D. In both graphs, the original source of non-stationarity is the variable

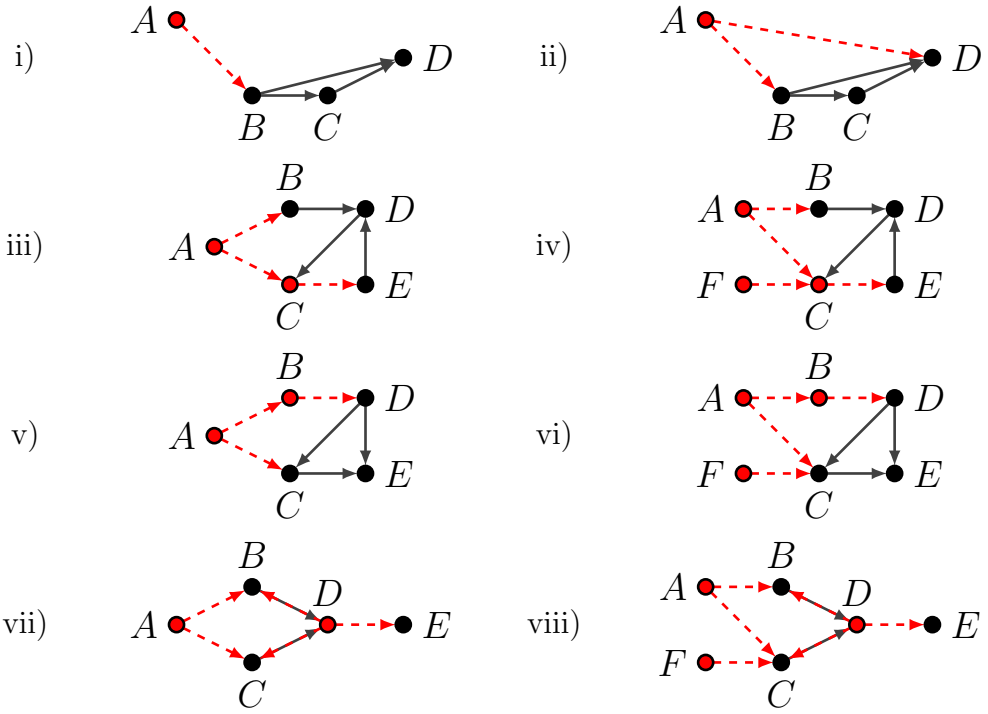

Figure 3.16: Causal Structures for Simulations

A. In i), A is directly connected to B, while all other nodes are only indirectly influenced by A through B. Thus, if B is kept fixed, both C and D become stationary, whereas the converse is not true. Therefore, $B \in \aleph_T$ is the sole driving causal trend with respect to $X_o = \{B, C, D\}$.

In graph ii), the direct edge between A and D alters the set of driving trends. In this configuration, even if B is held constant, D maintains its long-term dynamics as it continues to follow A, which exhibits non-stationary behaviour. Consequently, the driving trends are B and D.

Regarding non-causality, B lacks both direct and indirect incoming arrows from the elements of X_o, hence it is considered strongly not-caused in both graphs. Conversely, D is

influenced by C, which in both cases is an element of $\beth$. Thus, neither C nor D is strongly or weakly not-caused with respect to X_o.

Graphs i) and ii) can be represented using vector error correction models as follows:

$$\Delta \begin{bmatrix} A \\ B \\ C \\ D \end{bmatrix}_t = \begin{bmatrix} 0 & 0 & 0 \\ a & 0 & 0 \\ 0 & b & 0 \\ 0 & 0 & c \end{bmatrix} \begin{bmatrix} d & 1 & 0 & 0 \\ 0 & e & 1 & 0 \\ 0 & f & g & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \\ D \end{bmatrix}_{t-1} + \varepsilon_t \tag{3.12}$$

$$\Delta \begin{bmatrix} A \\ B \\ C \\ D \end{bmatrix}_t = \begin{bmatrix} 0 & 0 & 0 \\ a & 0 & 0 \\ 0 & b & 0 \\ 0 & 0 & c \end{bmatrix} \begin{bmatrix} d & 1 & 0 & 0 \\ 0 & e & 1 & 0 \\ f & g & h & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \\ D \end{bmatrix}_{t-1} + \varepsilon_t \tag{3.13}$$

The difference in their representation lies in the last row of β, where the term f in model (3.13) captures the influence of A on D.

Since A is treated as unobserved, the focus is on the adjustment matrix extracted from the model estimated by considering $X_o = \{B, C, D\}$ only. Given that the only latent variable is in T, the adjustment matrix can be computed using the reduced form in equation (3.4). Without loss of generality, we set Ω_o equal to the identity, so that α_o is simply $N_{o\perp}$. For

graph i), the resulting α_o is:

$$\alpha_o = \begin{bmatrix} ad \\ 0 \\ 0 \end{bmatrix}_\perp = \begin{bmatrix} 0 & 0 \\ \alpha_{o,1} & 0 \\ 0 & \alpha_{o,2} \end{bmatrix}$$

In graph ii), the matrix α_o assumes a different structure since N_o contains a non-null entry in the row corresponding to D. This results in a linear combination of the non-zero entries of N_o that sums to zero:

$$\alpha_o = \begin{bmatrix} ad \\ 0 \\ cf \end{bmatrix}_\perp = \begin{bmatrix} -xcf & 0 \\ 0 & \alpha_{o,1} \\ xad & 0 \end{bmatrix}$$

where x is any non-zero real number.

As illustrated in section 3.5, weak exogeneity is not informative for what concerns non-causality. In both cases, B is strongly not-caused, but the corresponding row in α_o is null only in graph i). Weak exogeneity is a property of the driving variable B only in graph i). This is consistent with the findings of section 3.4, given that in i) the set $\beth$ is $\{C, D\}$, while in ii), $\beth$ is the singleton set containing C. Thus, the cardinality of $\beth$ is 2 in the first case and 1 in the second. Since the cointegration rank is 2 in both cases, only in i) does r not exceed $|\beth|$, meeting the condition for the correspondence between weak exogeneity and driving trends, as per Theorem 5.

Graphs *iii*) and *iv*) have similar structures, differing only by the inclusion of a second fundamental trend in *iv*). Specifically, the set T for *iii*) is $\{A\}$, while for *iv*) it is $\{A, F\}$. In both cases, causality from T to D is mediated by the set $\{B, E\}$. Additionally, causality from $\{B, E\}$ to D is direct, thus $\aleph = \{B, E\}$ and $\beth = \{D\}$. No forward paths from T to $\aleph$ are blocked by elements of X_o. Consequently, $\aleph = \aleph_T$, and B and E introduce non-stationarity within X_o.

In both cases, the only variable not caused by any other element of X_o is B. Although E has no direct arrow from X_o, it is indirectly caused by D through C. Since $D \in \beth$, both D and E are neither strongly nor weakly not caused, whereas B is strongly not caused.

Graphs *iii*) and *iv*) are translated into models as follows:

$$\Delta \begin{bmatrix} A \\ B \\ C \\ D \\ E \end{bmatrix}_t = \begin{bmatrix} 0 & 0 & 0 & 0 \\ a & 0 & 0 & 0 \\ 0 & b & 0 & 0 \\ 0 & 0 & c & 0 \\ 0 & 0 & 0 & d \end{bmatrix} \begin{bmatrix} e & 1 & 0 & 0 & 0 \\ f & 0 & 1 & g & 0 \\ 0 & h & 0 & 1 & i \\ 0 & 0 & j & 0 & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \\ D \\ E \end{bmatrix}_{t-1} + \varepsilon_t \tag{3.14}$$

$$\Delta \begin{bmatrix} A \\ B \\ C \\ D \\ E \\ F \end{bmatrix}_t = \begin{bmatrix} 0 & 0 & 0 & 0 \\ a & 0 & 0 & 0 \\ 0 & b & 0 & 0 \\ 0 & 0 & c & 0 \\ 0 & 0 & 0 & d \\ 0 & 0 & 0 & 0 \end{bmatrix} \begin{bmatrix} e & 1 & 0 & 0 & 0 & 0 \\ f & 0 & 1 & g & 0 & h \\ 0 & i & 0 & 1 & j & 0 \\ 0 & 0 & k & 0 & 1 & 0 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \\ D \\ E \\ F \end{bmatrix}_{t-1} + \varepsilon_t \tag{3.15}$$

The adjustment matrix estimated by observing $X_o = \{C, D, E\}$ is derived using (3.2). For graph *iii*), it is:

$$\alpha_o = \Sigma_o(M_{ou}V_{uT} + N_oV_T) = \begin{bmatrix} \Sigma_{o1} & 0 & \Sigma_{o2} \\ 0 & \Sigma_{o3} & 0 \\ \Sigma_{o2} & 0 & \Sigma_{o4} \end{bmatrix} \begin{bmatrix} z \\ 0 \\ y \end{bmatrix}_\perp = \begin{bmatrix} \alpha_{o1} & 0 \\ 0 & \alpha_{o2} \\ \alpha_{o3} & 0 \end{bmatrix}$$

while for graph *iv*), it is

$$\alpha_o = \Sigma_o(M_{ou}V_{uT} + N_oV_T) = \begin{bmatrix} \Sigma_{o1} & 0 & \Sigma_{o2} \\ 0 & \Sigma_{o3} & 0 \\ \Sigma_{o2} & 0 & \Sigma_{o4} \end{bmatrix} \begin{bmatrix} z & y \\ 0 & 0 \\ x & w \end{bmatrix}_\perp = \begin{bmatrix} 0 \\ \alpha_{o1} \\ 0 \end{bmatrix}$$

As with graphs *i*) and *ii*), weak exogeneity is not associated with non-causality. Although B is strongly not-caused in both graphs, it is weakly exogenous in *iv*) but not in *iii*). Weak exogeneity is related to the driving trends in *iv*) but not in *iii*). This follows from

the findings of Theorem 5, given that in both cases $\beth = \{D\}$, but in *iii*) the cointegration rank is 2, while in *iv*) it is 1. Thus, only in *iv*) is the condition $r \leq |\beth|$ met, making the variables in $\aleph_T$ weakly exogenous.

Graphs *v*) and *vi*) are similar to graphs *iii*) and *iv*), with key differences in the causality from D to E and the group of variables treated as observed. The fundamental trends are A in *v*) and $\{A, F\}$ in *vi*). Unlike the previous graphs, D is now among the causal driving trends since B, which blocks its forward path from A, is no longer among the modeled variables. Conversely, E is now outside $\aleph_T$ since C, which transmits causality from T to E, is inside X_o in *v*) and *vi*). Consequently, the driving trends in both graphs are D and C.

The analysis of graphs *v*) and *vi*) differs from that of *iii*) and *iv*) due to the presence of weak non-causality. Specifically, C is caused by D, with no influence from $E \notin \aleph_T$. Conversely, D has no influence from any variable in X_o. Therefore, both C and D are weakly not caused, but only D is strongly not caused.

The VECM representation for *v*) and *vi*) is as follows:

$$\Delta \begin{bmatrix} A \\ B \\ C \\ D \\ E \end{bmatrix}_t = \begin{bmatrix} 0 & 0 & 0 & 0 \\ a & 0 & 0 & 0 \\ 0 & b & 0 & 0 \\ 0 & 0 & c & 0 \\ 0 & 0 & 0 & d \end{bmatrix} \begin{bmatrix} e & 1 & 0 & 0 & 0 \\ f & 0 & 1 & g & 0 \\ 0 & h & 0 & 1 & 0 \\ 0 & 0 & i & j & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \\ D \\ E \end{bmatrix}_{t-1} + \varepsilon_t \tag{3.16}$$

$$\Delta \begin{bmatrix} A \\ B \\ C \\ D \\ E \\ F \end{bmatrix}_t = \begin{bmatrix} 0 & 0 & 0 & 0 \\ a & 0 & 0 & 0 \\ 0 & b & 0 & 0 \\ 0 & 0 & c & 0 \\ 0 & 0 & 0 & d \\ 0 & 0 & 0 & 0 \end{bmatrix} \begin{bmatrix} e & 1 & 0 & 0 & 0 & 0 \\ f & 0 & 1 & g & 0 & h \\ 0 & i & 0 & 1 & 0 & 0 \\ 0 & 0 & j & k & 1 & 0 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \\ D \\ E \\ F \end{bmatrix}_{t-1} + \varepsilon_t \tag{3.17}$$

The adjustment matrix estimated by observing $X_o = \{B, C, D\}$ is derived using (3.2). For graph v), it is:

$$\alpha_o = \Sigma_o(M_{ou}V_{uT} + N_oV_T) = \begin{bmatrix} \Sigma_{o1} & \Sigma_{o2} & 0 \\ \Sigma_{o2} & \Sigma_{o3} & 0 \\ 0 & 0 & \Sigma_{o4} \end{bmatrix} \begin{bmatrix} z \\ 0 \\ 0 \end{bmatrix}_\perp = \begin{bmatrix} \alpha_{o1} & 0 \\ \alpha_{o2} & 0 \\ 0 & \alpha_{o3} \end{bmatrix}$$

while for graph vi), it is

$$\alpha_o = \Sigma_o(M_{ou}V_{uT} + N_oV_T) = \begin{bmatrix} \Sigma_{o1} & \Sigma_{o2} & 0 \\ \Sigma_{o2} & \Sigma_{o3} & 0 \\ 0 & 0 & \Sigma_{o4} \end{bmatrix} \begin{bmatrix} z & y \\ x & w \\ 0 & 0 \end{bmatrix}_\perp = \begin{bmatrix} 0 \\ 0 \\ \alpha_{o1} \end{bmatrix}$$

These matrices are equivalent to those of graphs iii) and iv).

As with strong non-causality, weak non-causality is not linked to the restrictions on α_o. Specifically, C, which is weakly not caused in both graphs, is weakly exogenous in v) but

not in *vi*). Regarding the driving forces, the components in $\aleph_T$ are weakly exogenous in *v*) but not in *vi*). This aligns with Theorem 5, which states that $\aleph_T$ are related to zero rows in α_o if $r \leq |\beth|$. Since the cardinality of $\beth$ is 1 and the rank of the partially observed model is 2 in *v*) and 1 in *vi*), only in the latter scenario is weak exogeneity connected with the causal driving trends.

The analysis of graphs *vii*) and *viii*) is particularly interesting because the set of driving forces does not correspond to $\aleph$. Despite B and C blocking the forward paths between the fundamental trends and E, the causal transmission between $\{B, C\}$ and E is indirect, passing through the unobserved D, which in turn causes B and C. Consequently, the set $\beth$ is empty while $\aleph$ equals X_o. Fixing B and C causes D and subsequently E to lose their non-stationary behaviour. Therefore, the driving trends are the variables B and C. Both B and C are weakly not caused since the component of $\beth$ does not influence them. Nevertheless, they are both influenced by D which in turn is caused by B and C themselves. It follows that $B \in \aleph_T$ indirectly causes $C \in \aleph_T$ and vice-versa. Consequently, neither B nor C are strongly not-caused.

Graphs *vii*) and *viii*) are represented by the following models:

$$\Delta \begin{bmatrix} A \\ B \\ C \\ D \\ E \end{bmatrix}_t = \begin{bmatrix} 0 & 0 & 0 & 0 \\ a & 0 & 0 & 0 \\ 0 & b & 0 & 0 \\ 0 & 0 & c & 0 \\ 0 & 0 & 0 & d \end{bmatrix} \begin{bmatrix} e & 1 & 0 & f & 0 \\ g & 0 & 1 & h & 0 \\ 0 & i & j & 1 & 0 \\ 0 & 0 & 0 & k & 1 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \\ D \\ E \end{bmatrix}_{t-1} + \varepsilon_t \tag{3.18}$$

$$\Delta \begin{bmatrix} A \\ B \\ C \\ D \\ E \\ F \end{bmatrix}_t = \begin{bmatrix} 0 & 0 & 0 & 0 \\ a & 0 & 0 & 0 \\ 0 & b & 0 & 0 \\ 0 & 0 & c & 0 \\ 0 & 0 & 0 & d \\ 0 & 0 & 0 & 0 \end{bmatrix} \begin{bmatrix} e & 1 & 0 & f & 0 & 0 \\ g & 0 & 1 & h & 0 & i \\ 0 & j & k & 1 & 0 & 0 \\ 0 & 0 & 0 & l & 1 & 0 \end{bmatrix} \begin{bmatrix} A \\ B \\ C \\ D \\ E \\ F \end{bmatrix}_{t-1} + \varepsilon_t \tag{3.19}$$

The adjustment matrix α_o for the VECM modeling $X_o = \{C, D, E\}$ is derived using (3.2).

For graph *vii*), it is:

$$\alpha_o = \Sigma_o(M_{ou}V_{uT} + N_oV_T) = \begin{bmatrix} \Sigma_{o1} & \Sigma_{o2} & \Sigma_{o3} \\ \Sigma_{o2} & \Sigma_{o4} & \Sigma_{o5} \\ \Sigma_{o3} & \Sigma_{o5} & \Sigma_{o6} \end{bmatrix} \begin{bmatrix} z \\ y \\ x \end{bmatrix}_{\perp} = \begin{bmatrix} \alpha_{o1} & \alpha_{o2} \\ \alpha_{o3} & \alpha_{o4} \\ \alpha_{o5} & \alpha_{o6} \end{bmatrix}$$

while for graph *iv*), it is

$$\alpha_o = \Sigma_o(M_{ou}V_{uT} + N_oV_T) = \begin{bmatrix} \Sigma_{o1} & \Sigma_{o2} & \Sigma_{o3} \\ \Sigma_{o2} & \Sigma_{o4} & \Sigma_{o5} \\ \Sigma_{o3} & \Sigma_{o5} & \Sigma_{o6} \end{bmatrix} \begin{bmatrix} z & y \\ x & w \\ v & u \end{bmatrix}_{\perp} = \begin{bmatrix} \alpha_{o1} \\ \alpha_{o3} \\ \alpha_{o5} \end{bmatrix}$$

Similar to previous cases, non-causality does not correspond to restrictions on the adjustment matrix. According to Theorem 4, when $\aleph_B$ is not empty, the variables in $\aleph$ are not weakly exogenous. In fact, as demonstrated in section 3.4, when $\aleph_B \neq \emptyset$, the condition $r \leq |\beth|$ cannot be met. In graphs *vii*) and *viii*), $\aleph_B = \{E\}$ and the cardinality of $\beth$ is zero. The cointegration rank is 2 in the model relative to *viii*) and 1 in the model corresponding to *vii*). Therefore, in both cases, the rank exceeds $|\beth|$, as expected from the findings of section 3.4.

In the next subsection, we simulate time series generated from the causal structures illustrated thus far and test for weak exogeneity. We will evaluate the test performance under the different scenarios discussed in this section.

3.6.2 Results of Simulations

The observations are simulated following the methodology outlined in section 5 of Chapter 2. The pseudo-code detailing the generation of the time series is provided in Algorithm 1. To test the restrictions on the adjustment matrix, standard statistical software packages offer built-in functions. In this study, the Matlab function *jcontest* was utilized for con-

Graph	Variable	N=100	N=500	N=1000	N=10000
i)	B	9.2%	8.1%	6.6%	6.6%
iv)	B	13.1%	7.2%	6.7%	7%
	E	21.5%	12.4%	9.6%	7.3%
vi)	C	12.5%	8.1%	6.9%	5.5%
	D	12.3%	7.1%	5.1%	6%

Table 3.1: Rejection Rates for Tests Implemented Under the Null Hypothesis. Significance Level 5%. N indicates the Sample Size.

ducting these simulations. Given that the order of the partially observed C-VAR model is infinite, it was approximated by a VECM of order $\lceil \ln(N) \rceil$, where N denotes the sample size. This approximation was chosen due to the exponentially decreasing coefficients associated with short-term dynamics, ensuring minimal information loss.

We begin by examining the outcomes of the test under the null hypothesis, i.e. the test applied to the variables B in graphs i) and iv), E in graph iv), and C and D in graph vi). The test is conducted across four different sample sizes: $N = 100$, $N = 500$, $N = 1,000$, and $N = 10,000$. Table 3.2 presents a summary of the simulation results.

The rejection rates approach the designated significance level as the sample size increases, aligning with expectations based on the theory of the log-likelihood ratio test. This behaviour stems from the fact that the statistic asymptotically follows a chi-squared distribution as the sample size tends to infinity. Across all cases considered, the rejection rates generally decrease as the sample size, denoted as N, increases. An exception to this trend is observed in the rejection rates for variables D and B in graphs vi) and iv), which increase from 5.1% and 6.7% at $N = 1,000$ to 6% and 7.3% at $N = 10,000$. The reason for this increase is not entirely clear and may be attributed to the order approximation or

Graph	Variable	N=100	N=500	N=1000	N=10000
i)	C	97.3%	99.7%	99.9%	100%
	D	98.6%	99.7%	99.8%	100%
ii)	B	33.9%	52%	52.1%	70.2%
	C	97.9%	99.9%	100%	100%
	D	36.6%	46.1%	57.2%	72.6%
iii)	B	17.7%	23.3%	25.5%	32%
	D	96.8%	99.7%	99.8%	100%
	E	90.4%	93.9%	95.5%	98.2%
iv)	D	82.2%	95.8%	97.9%	99.4%
v)	C	28.3%	30.6%	31.3%	39.9%
	D	81.7%	93.7%	94%	98.1%
	E	98.8%	100%	99.9%	100%
vi)	E	92.4%	99%	99.9%	100%
vii)	B	56.6%	80.8%	84%	94.2%
	C	58.6%	78.1%	82.8%	95.3%
	E	95.4%	99.7%	99.9%	100%
viii)	B	42%	62.6%	69.5%	85.2%
	C	35.3%	53.8%	63.4%	80%
	E	85.3%	94.6%	97.2%	99.4%

Table 3.2: Rejection Rates for Tests Implemented Under the Alternative Hypothesis. Significance Level 5%. N Indicates the Sample Size.

random variability, given that these percentages are not significantly divergent from 5%.

An important observation pertains to the heterogeneity among rejection rates, particularly evident at smaller sample sizes. It is notable that the test's performance varies considerably across different scenarios. For instance, the variable E in graph *iv*) exhibits the poorest performance across all sample sizes. This variability can be attributed to the interplay between weak exogeneity and non-causality. Specifically, E in graph *iv*) is unique among the simulated variables as it is weakly exogenous while neither strongly nor weakly not-caused. This suggests that while weak exogeneity is distinct from non-causality, its absence can impact the accuracy of tests, especially evident with smaller sample sizes.

We now analyse the test performance under the alternative hypothesis. The test outcomes are reported in Table 3.2. As expected, the rejection rates increase as the sample size increases. This is coherent with the expectation, as the power approaches 100% as the sample size tends to infinity. In all the cases, the rejection rate grows as N increases without any significant exception. Nevertheless, the difference in the test's performance across the several scenarios is immediately evident. There are in fact cases where the empirical power exceeds 97% with only 100 observations while other cases where the power is below 35% even with 10,000 observations.

The test exhibits robust performance when applied to variables within $\beth$ or $\aleph_B$, evidenced by the results for C and D in i), C in ii), D in iii) and iv), and E in v)-$viii$). Slightly inferior performance is noted for D in iv) and v), as well as E in vi) and $viii$), where the cardinality of $\aleph_T$ matches the number of fundamental trends. However, the differences in accuracy are relatively minor and diminish as the sample size increases.

In general, the test accuracy is lower when the tested variable belongs to $\aleph_T$. The poorest performances are observed in scenarios where some of the variables in $\aleph_T$ are directly connected to T, while causality from T to the other variables in $\aleph_T$ is mediated by unobserved components. In such scenarios, the test power is consistently low for the variables directly connected to T, while it reaches reasonable levels for the variables indirectly connected via latent components. For example, consider the variables B and C in graphs iii) and v), respectively. In these cases, B and C are directly influenced by fundamental trends, while causality from T to other components of $\aleph_T$ (E in iii) and D in v)) is mediated through

latent variables. The test power for the directly connected group is below 40%, even with a sample size of 10,000, while it approaches 100% for the indirectly connected group.

In the other cases where the tested variable is not weakly exogenous but is among the driving forces, test performance is poor for small sample sizes, with rejection rates ranging around 30% and 50%, improving markedly to between 80% and 90% in larger samples.

In summary, this section has demonstrated that the causal characteristics of tested variables significantly influence test performance on restrictions of the partially observed adjustment matrix. Notably, while some driving trends are not weakly exogenous, their rejection rates in small samples remain low. Conversely, tests on components of $\aleph_B$ and $\beth$ frequently reject the hypothesis of weak exogeneity. It has also been observed that the weakest performance occurs when one driving variable is directly influenced by T, while others in $\aleph_T$ are indirectly linked to fundamental trends. In these cases, rejection rates for the former remain consistently low, while those for the latter reach levels akin to tests on elements of $\beth \cup \aleph_B$.

Although non-causality is distinct from weak exogeneity, this property impacts the test performance in terms of size for finite sample sizes. Specifically, if a weakly exogenous variable is caused by elements outside $\aleph_T$, rejection rates approach the significance level more gradually compared to non-caused variables.

3.7 Empirical Illustration

In this section, we demonstrate the practical application of the results presented in this chapter through an empirical illustration related to business cycles.

The results should be interpreted as illustrative due to the preliminary nature of the analysis. Critical aspects, such as the presence of deterministic components, are not addressed because the theoretical framework on which this chapter is grounded does not cover general vector error correction models with constant or deterministic trends. To mitigate this issue, we will detrend the data to remove deterministic trends. However, this may result in a loss of informative content. Additionally, diagnostic checks for model specifications and tests for other specification issues, such as coefficient constancy, are not conducted.

This section is structured as follows: the next subsection briefly outlines the main business cycle theories and the expected empirical results associated with each. Subsection 3.7.2 describes the dataset, including the data sources and plots of the raw and the modified time-series employed for the empirical analysis. Finally, subsection 3.7.3 presents and discusses the empirical results.

3.7.1 Business Cycle Theories

Business cycles, also known as economic cycles, refer to the fluctuations in economic activity that an economy experiences over time. These cycles consist of periods of economic

expansion, peak, contraction, and trough. Understanding the causes and dynamics of these cycles is essential for policymakers, in order to mitigate the crisis periods produced by the cyclical behaviour.

The study of business cycles recognizes significant long-standing regularities. Although the observed fluctuations vary greatly in amplitude, scope, and duration, they also share common features. Business cycles are persistent, typically lasting several years, allowing for cumulative movements in both downward and upward directions. Despite their differences, business expansions and contractions exhibit recurrent, serially correlated, and cross-correlated movements in many economic and other activities (see Zarnowitz (1992)).

The systematic study of business cycles began in the early 19th century, when economists started exploring the causes of economic downturns and expansions and the driving forces behind this wave-like behaviour in economic output. Early theories identified diverse driving forces, ranging from financial reasons to natural cycles, and even astronomical phenomena such as planetary movements and solar spots. More recent developments focus on economic variables, emphasizing the role of macroeconomic factors in the cyclical behaviour of the general economy. Consensus on the origin of cycles has not yet been achieved, and contrasting schools of thought propose various reasons for the stylized facts concerning economic trends.

The analysis of business cycles is closely related to identifying driving forces in non-stationary systems. Identifying variables that introduce non-stationarity into the system is equivalent to pinpointing which variables drive the cyclical behaviour of economic vari-

ables. In essence, the driving forces in economic systems are those variables that, if kept fixed, would cause the other analysed variables to lose their non-stationary, and thus their cyclical behaviour.

There is no consensus on which variables drive cyclical paths. This section compares three different theories: Keynesian, Monetarist, and Real Business Cycle (RBC) Theory.

Central to the Keynesian theory of business cycles is the concept of aggregate demand. Keynes argued that changes in aggregate demand are the primary drivers of economic expansions and contractions. In Keynesian economics, economic output is driven by the level of aggregate demand. When aggregate demand is high, businesses produce more goods and services, leading to economic expansion and higher employment. Conversely, when aggregate demand falls, businesses cut back on production, leading to economic contraction and higher unemployment. Keynes also emphasized the "sticky" nature of prices and wages, which do not adjust immediately to changes in economic conditions, leading to prolonged periods of disequilibrium where supply does not match demand.

In the Keynesian theory, interest rates are seen as the key variable causing the repetition of the cyclical fluctuations. If the interest rates rise, investment is eventually reduced. If this depressing effect is strong enough, a recession is induced by the corresponding fall in aggregate demand; unemployment and excess capacity increase, the transaction demand for money decreases, and the interest rate falls. If the latter influence is sharp enough investment may overcome the depressing influence of excess capacity and low profits.

Recovery sets in and the stage is set for a repetition of the story.[1]

It should be emphasized that, despite the Keynesian view positing the interest rate as the variable generating cyclical behaviour, this behaviour is transmitted and introduced into the system, and thus to the economic output, by aggregate demand. Therefore, the analysis of the driving variables should focus on aggregate demand rather than on the interest rate.

The monetarist view attributes economic fluctuations primarily to changes in the money supply. According to monetarists, the main driver of business cycles is the amount of money circulating in the economy, which directly influences levels of economic activity. Monetarists argue that stable growth in the money supply is crucial for maintaining economic stability. When the money supply grows at a consistent, moderate rate, it fosters steady economic growth and low inflation. Conversely, if the money supply contracts or grows too slowly, it can lead to economic stagnation or recession, as there is insufficient money to support economic activities.

The real business cycle (RBC) theory offers a distinctive approach by focusing on real, rather than monetary, factors as the primary drivers of economic fluctuations. Developed in the 1980s following the seminal work by Kydland and Prescott (1982), RBC theory posits that changes in productivity and external shocks to the economy are the main causes of economic expansions and contractions. Business cycles result from variations in the rate of technological innovation and changes in factors that affect productivity, such as

[1]For further discussion on keynesian business cycle, refer to Day and Lin (1991), and Onwumere et al. (2011)

advancements in technology, availability of natural resources, and changes in government regulations. These real shocks influence the production capacity of the economy.

RBC theory assumes rational expectations and market efficiency, suggesting that individuals and firms make decisions based on all available information and adjust their behaviour optimally in response to economic changes. Fluctuations in output and employment are seen as efficient responses to real changes rather than deviations from a potential output level. Economic agents adjust their labor supply, and consumption patterns in response to productivity changes, leading to economic expansion during high productivity and contraction during low productivity periods.

Understanding which business cycle theory is correct is crucial as each leads to different policy solutions for mitigating periods of downturn.

Keynesians advocate for government intervention to stabilize the economy. During low aggregate demand periods, the government should increase spending and reduce taxes to boost demand, encouraging businesses to produce more and hire more workers. During excessive demand periods, reducing government spending or increasing taxes is recommended to cool the economy.

Monetarists criticize active fiscal policy and government intervention, arguing that such measures often cause more harm than good due to delays and inaccuracies in implementation. Instead, they advocate for a rules-based monetary policy, such as targeting steady money supply growth to reduce uncertainty and enable better economic decision-making by businesses and consumers.

RBC theory downplays the role of monetary policy and government intervention, viewing fluctuations as optimal responses to real changes. Stabilization attempts through monetary or fiscal policy are often seen as unnecessary or harmful. RBC proponents argue that policy should support efficient market operations by ensuring property rights and reducing market distortions.

In this empirical application, we aim to determine which of the three theories finds more support in the data. If the Keynesian view is correct, aggregate demand components are the driving forces, and we expect to find weak exogeneity patterns among those variables. In the monetarist view, output is driven by the money supply, so weak exogeneity should characterize the money supply. Finally, if RBC theory is correct, weak exogeneity is a characteristic of factors efficiency measures, such as labor productivity. In the following sections, we analyse the data by testing restrictions on the adjustment matrix and assessing which theory aligns with the results.

3.7.2 Data

In this subsection, we describe the dataset employed for the empirical analysis. To study which of the three business cycle theories described in subsection 3.6.1 finds evidence in the data, we used data related to aggregate demand, labor productivity, money aggregate, and economic output. Aggregate demand components include consumption, investment, public spending, and net exports, computed as the difference between total exports and total imports.

To capture the long-term aspects of business cycle analysis, we decided to use a long dataset spanning nearly a century and a half, from 1870 to 2016. The choice of 2016 as the endpoint is due to data availability for labor productivity. The country chosen for analysis is the United Kingdom.

The data are sourced from two primary databases. Labor productivity data are obtained from the website "Our World in Data," based on the work of Bergeaud et al. (2016). Labor productivity is measured as the ratio of GDP to total hours worked, adjusted for inflation using 2010 prices. Other data are sourced from the "Jordà-Schularick-Taylor Macrohistory" Database (JST Database), which compiles several works to create a comprehensive economic dataset for the period 1870-2020 (see Jordà et al. (2017)). Appendix B provides the data sources along with the corresponding web links.

Economic output is measured using real GDP. While the JST Database does not provide real GDP directly, it includes an index of real GDP per capita (base year 2005). This index, sourced from Barro and Ursúa (2010) and World Bank (2021), is multiplied by the annual population divided by the population in 2005 to obtain the real GDP index, denoted as Y. Population data are taken from Angus Maddison Database (2008) and International Monetary Fund (2019). Similarly, real consumption data are provided as an index of real consumption per capita with 2006 as the base year. To derive the total real consumption index (denoted C), each observation is multiplied by the ratio of the annual population to the population in 2006. Investment data (I) are given as the ratio between investment and nominal GDP, sourced from Hills et al. (2015). To obtain total investment

time series, these observations are multiplied by nominal GDP, sourced from Hills et al. (2015) and Office for National Statistics (2021). Data for other components of aggregate demand, such as government spending (G), imports (X), and exports (E), are provided in billions of GBP. Government spending data are sourced from Mitchell (1988), and the database of the Office for National Statistics, while import and export data are sourced from Hills et al. (2015) and the IMF International Monetary Fund (2021).

Money supply (M) is measured using a broad aggregate collected in billions of pounds from Dimsdale and Thomas (2016). A broad aggregate is used because the total amount of money in the economy is more relevant for economic dynamics than the quantity directly supplied by central banks.

The raw data are depicted in Figures 3.17-3.24. Since some variables are indicated as indices and others in monetary terms, all magnitudes are expressed as indices, with 2006 as the base year (2006=100). Figure 3.25 shows the variables in index form.

To account for exponential growth, we transformed the data using logarithms and then de-trended it to eliminate deterministic components. To verify the correctness of the de-trending, a Dickey-Fuller test was conducted to determine if the variables remain $I(1)$ after de-trending. Table 3.3 presents the test results. The hypothesis of a unit root can be rejected only for G. Therefore, the variable for public spending is not de-trended but used solely in its log-value. The result of the unit root test on the log of G is reported in Table 3.4.

The unit root test was then applied to the first differences of the selected variables to

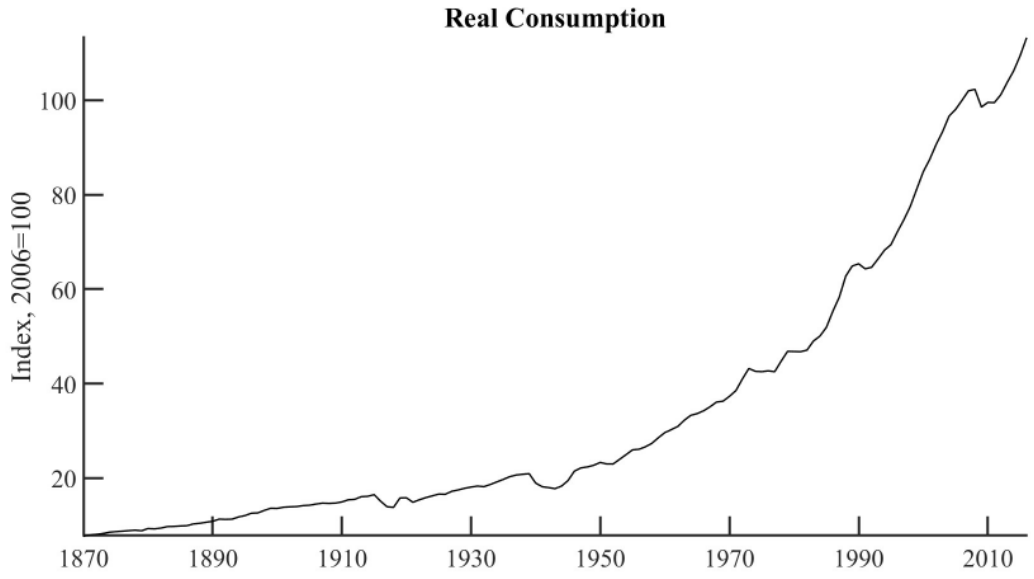

Figure 3.17: Real Consumption Index, Retrieved from the "JST Database"

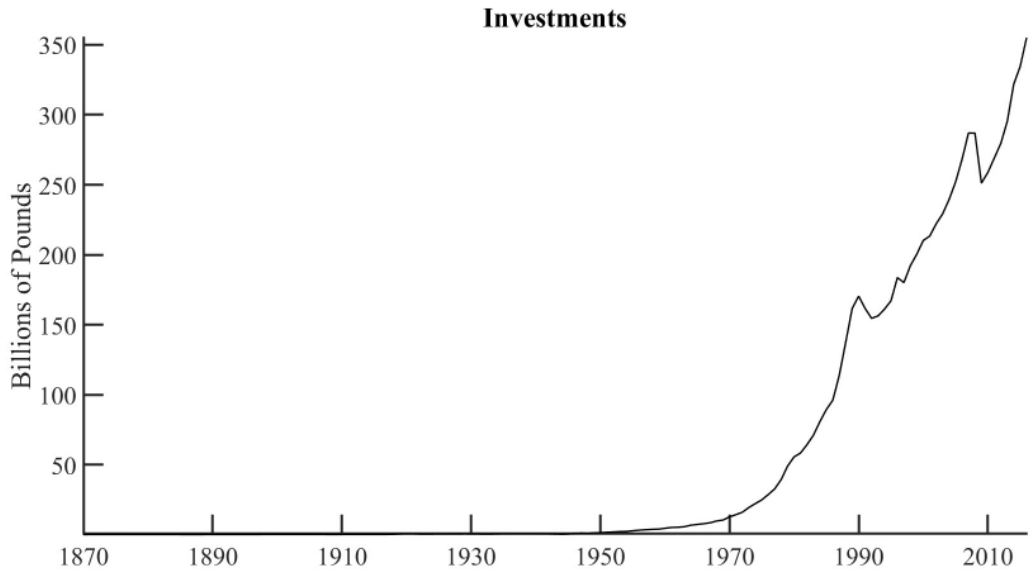

Figure 3.18: Investments, Retrieved from the "JST Database"

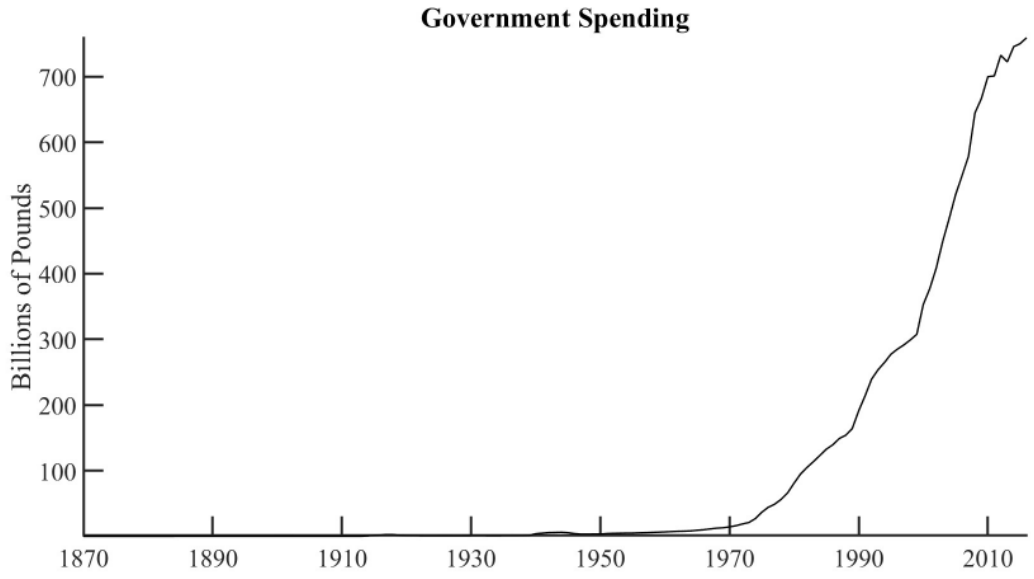

Figure 3.19: Total Government Spending, Retrieved from the "JST Database"

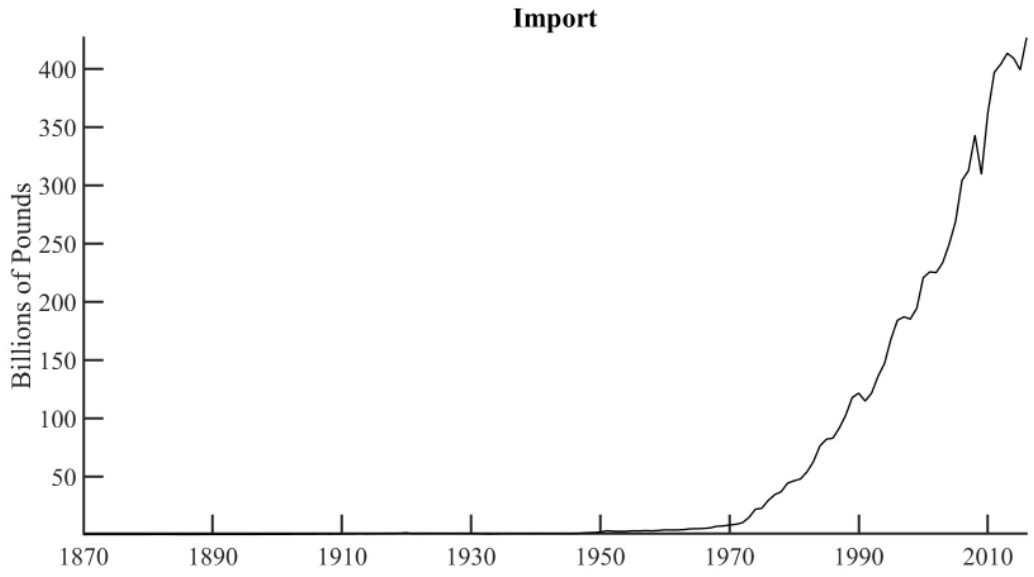

Figure 3.20: Import, Retrieved from the "JST Database"

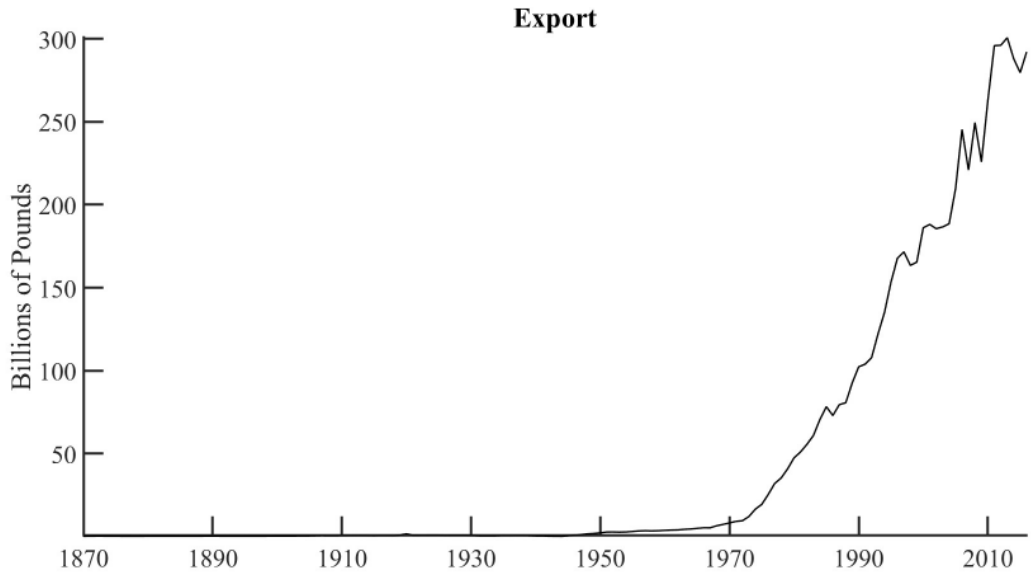

Figure 3.21: Export, Retrieved from the "JST Database"

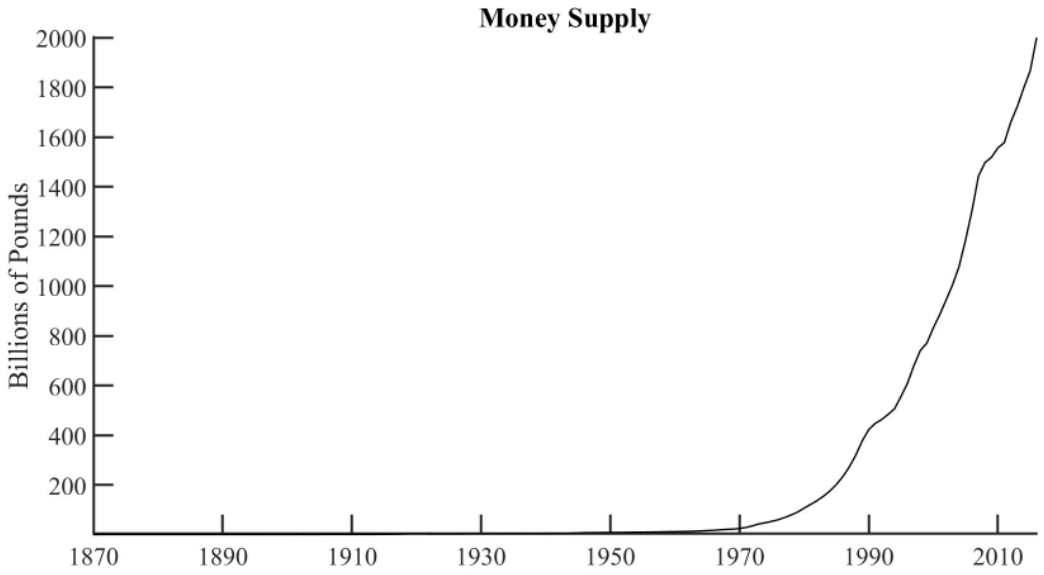

Figure 3.22: Money Supply, Retrieved from the "JST Database"

Figure 3.23: Labour Productivity, Retrieved from the Database "Our World in Data"

Figure 3.24: Real GDP Index, Retrieved from the "JST Database"

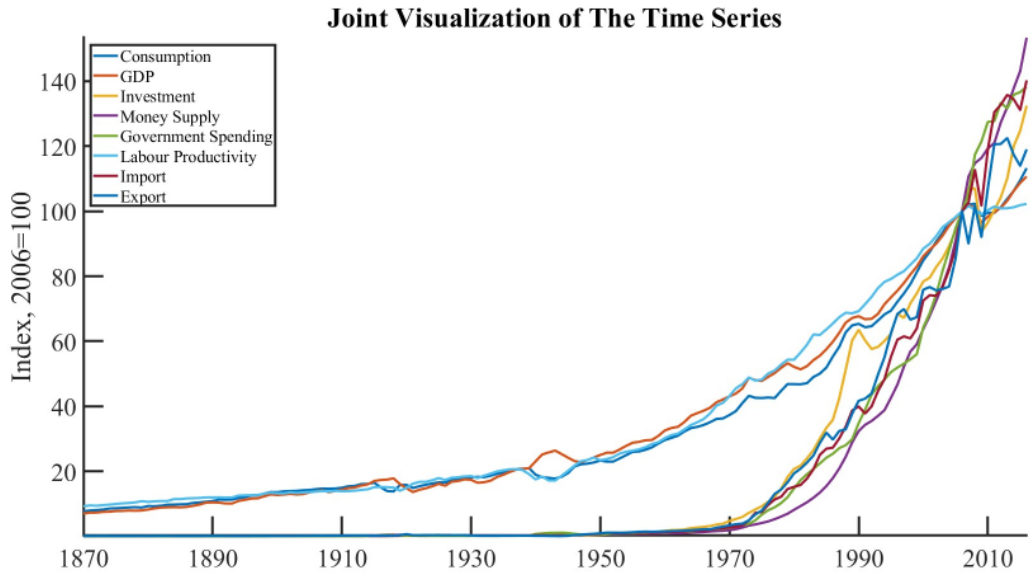

Figure 3.25: Combined visualization of all individual time series.

	P-Value	Statistic	C-Value
C	0.3308	-0.8728	-1.9427
Y	0.0724	-1.7732	-1.9427
I	0.0695	-1.7926	-1.9427
M	0.0635	-1.8348	-1.9427
G	**0.0201**	**-2.3197**	-1.9427
L	0.1352	-1.4582	-1.9427
X	0.1639	-1.3497	-1.9427
E	0.1089	-1.5740	-1.9427

Table 3.3: Results for the Dickey-Fuller Test. Rejections are depicted in bold.

	P-Value	Statistic	C-Value
G	0.6480	-0.0061	-1.9427

Table 3.4: Results for the Dickey-Fuller Test for the logarithm of G

	P-Value	Statistic	C-Value
C	**1.0000e-03**	**-8.7098**	-1.9427
Y	**1.0000e-03**	**-8.2740**	-1.9427
I	**1.0000e-03**	**-7.2396**	-1.9427
M	**1.0000e-03**	**-4.4183**	-1.9427
G	**0.0032**	**-3.0433**	-1.9427
L	**1.0000e-03**	**-9.3929**	-1.9427
X	**1.0000e-03**	**-10.0103**	-1.9427
E	**1.0000e-03**	**-8.7424**	-1.9427

Table 3.5: Results for the Dickey-Fuller Test for the first difference of the variables

exclude the presence of $I(2)$ time-series. The results are reported in Table 3.5. As observed, the hypothesis of a unit root presence can be rejected for all the first differences of the variables. Consequently, we can conclude that their first differences are stationary, and the variables are $I(1)$.

To summarise, the dataset comprises time series from 1870 to 2016 for the United Kingdom, including aggregate demand components (consumption, investments, government spending, imports, and exports), real GDP, money supply, and labor productivity. To ensure uniformity, all data are expressed in index terms with 2006 as the base year. Given the exponential growth observed in the series, the data are logarithmically transformed. To eliminate deterministic components, the data are de-trended, except for public spending, given that de-trending G resulted in a process in which the unit root hypothesis could be rejected. The final dataset is illustrated in Figure 3.26.

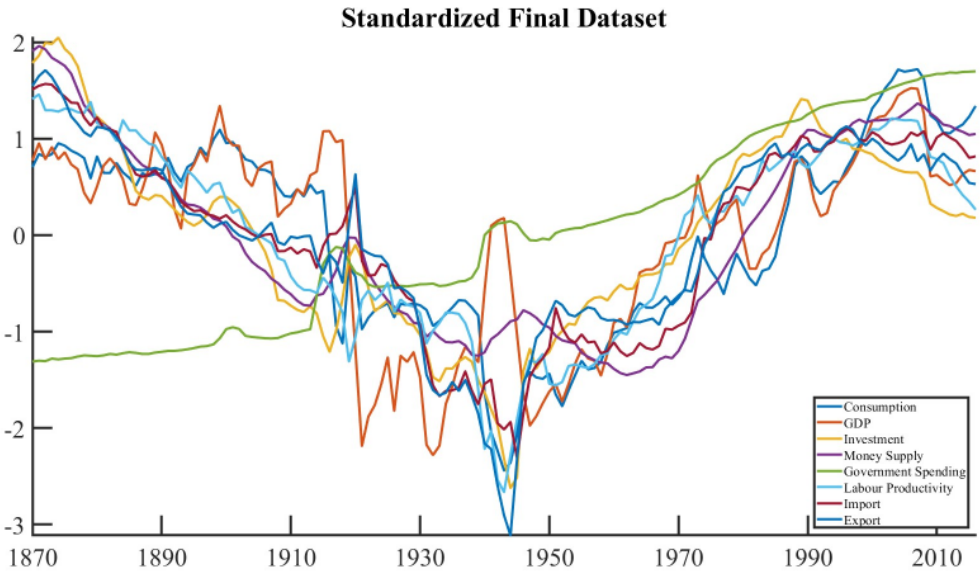

Figure 3.26: Final Dataset.

3.7.3 Results

The empirical analysis begins with determining the optimal order of the model. The Akaike Information Criterion (AIC) values for different model orders k of a simple VAR fitted to the data in levels are presented in Table 3.6. The AIC is minimized for $k = 10$. This high order aligns with Johansen and Juselius (2014), who found that a partially observed C-VAR(1) model behaves like a VECM of infinite order with exponentially decreasing short-term coefficients. To approximate the infinite order, we use the nearest integer to the logarithm of the sample size. With 147 observations, $\log(147) \approx 4.99$. Thus, the VECM(∞) is approximated by a VECM of order 5.

To determine the cointegration rank r, we employ the Johansen trace test. The test outcomes are recorded in Table 3.7. The estimated rank is found to be 6, as the hypothesis

k	AIC
1	-3.4242e+03
2	-3.6841e+03
3	-3.7115e+03
4	-3.6805e+03
5	-3.7063e+03
6	-3.7315e+03
7	-3.7327e+03
8	-3.7335e+03
9	-3.7843e+03
10	**-3.8626e+03**

Table 3.6: Akaike Information Criterion, Results for Different Lag k for the Simple VAR Fitted to the Levels. Bold Number Indicates the Minimal Value

	Statistic	C-Value	P-Value
$r = 0$	**219.2210**	143.6711	**0.0010**
$r \leq 1$	**162.2812**	111.7810	**0.0010**
$r \leq 2$	**112.7575**	83.9402	**0.0010**
$r \leq 3$	**72.8989**	60.0623	**0.0035**
$r \leq 4$	**46.3440**	40.1751	**0.0108**
$r \leq 5$	**24.4711**	24.2747	**0.0473**
$r \leq 6$	9.8101	12.3206	0.1273
$r \leq 7$	0.4137	4.1302	0.6931

Table 3.7: Statistics of the Trace Test for the Identification of Cointegration Rank and Critical Values for a 5% Significance Level. Bold Numbers Represent Rejection of the Null

$r \leq 6$ cannot be rejected, while there is evidence against the hypothesis $r \leq 5$.

The weak exogeneity test was implemented for each row of the adjustment matrix. The test results are presented in Table 3.8. The hypothesis of weak exogeneity cannot be rejected for aggregate consumption and government spending at the 5% significance level. However, the result for government spending is less robust, with a p-value of 0.0532, indicating the hypothesis would be rejected at the 10% significance level. Conversely, the p-value for aggregate consumption is below 1%, meaning the hypothesis would be rejected

	P-Value	Statistic	C-Value
C	0.5029	5.3244	12.5916
Y	**0.0040**	**19.0703**	12.5916
I	**0.0083**	**17.2758**	12.5916
M	**0.0027**	**20.0291**	12.5916
G	0.0532	12.4194	12.5916
L	**9.1366e-04**	**22.6732**	12.5916
X	**2.7688e-04**	**25.4910**	12.5916
E	**8.0979e-04**	**22.9606**	12.5916

Table 3.8: P-Value, Statistics and Critical Values for Weak Exogeneity Test. Bold Numbers Represent Rejection of the Null for a Significance Level of 5%

even at a 99% confidence level. On the other hand, the p-value for the test applied to the other variables is consistently below 1%, indicating that the hypothesis of weak exogeneity would be rejected at both the 95% and 99% confidence levels.

The test results indicate no evidence against the hypothesis that G and C are driving trends. This suggests that the demand components drive economic dynamics and, consequently, the business cycle. These findings align with the Keynesian view, which posits that fluctuations in economic activity are generated by changes in aggregate demand. However, it should be noted that the rejection of the weak exogeneity hypothesis for other components does not necessarily imply that monetary and real factors are not driving trends, given that weak exogeneity is a sufficient but not necessary condition for a variable to be a driving trend. This also applies to other components of aggregate demand, such as the net difference between imports and exports, and investments.

It is important to emphasize that even if demand factors are the sole driving forces, this does not preclude causality from money supply and real factors to the demand compo-

nents. This follows from the findings in section 3.5, which demonstrated that non-causality is not related to weak exogeneity. Consequently, the data suggests that demand drives the fluctuations of the economic system, with other components following demand. However, it remains possible that money supply and labor productivity exert causality on consumption, investment, and net exports.

3.8 Conclusions

In the literature, weak exogeneity has often been linked to structural characteristics such as non-causality and driving forces. Building on recent studies on partially observed cointegrated models, we investigated to what extent constraints on the adjustment matrix convey information regarding these causal features.

We confirmed the existence of a one-to-one mapping between weak exogeneity and driving trends, as well as between weak exogeneity and non-causality, when modeling the complete set of variables influencing a system. However, through specific examples, we illustrated how this correspondence breaks down when only a subset of variables is modeled.

Our analysis studied under what circumstances weak exogeneity is informative for identifying driving forces even within partially observed systems. We demonstrated that weak exogeneity serves as a sufficient but not necessary condition for a variable to act as a driving causal trend, i.e. those variables inducing non-stationarity in the system, such that holding them constant would render the other observed variables stationary.

In contrast, we showed that weak exogeneity does not provide information about non-causality. Indeed, our examples illustrated that weak exogeneity can apply to variables influenced directly or indirectly by other modeled variables, as well as to those unaffected by the other components of X_o.

In conclusion, this chapter contributes to econometric literature by highlighting that causal interpretations based on constraints of the adjustment matrix can be misleading, and clarifies the types of causal insights that can be gleaned from testing these restrictions.

Conclusions

In this final chapter, we present our conclusions, emphasizing the novelty of the techniques employed throughout the thesis, which form the basis of the econometric methods used in this research. We summarise the logical structure and reasoning to facilitate replication in future research. Additionally, we briefly outline our findings, highlighting their practical utility in addressing economic problems and providing empirical evaluations of contrasting macroeconomic theories. We also identify potential areas for further development and propose initial ideas for future research. Finally, we offer some concluding remarks.

The three chapters of the thesis addressed the complex problem of interpreting causality analysing restrictions of cointegrated systems focusing on their vector error correction representation. Specifically, we examined the interpretation of restrictions on the adjustment matrix. We demonstrated that the duality between null rows in the adjustment matrix and non-causality breaks down when not all relevant factors are modeled, proving that causal interpretations necessitate a departure from standard statistical analysis.

To address these challenges, we introduced novel definitions and developed a new analytical procedure. Building on the causal analysis framework of Hoover (2020) and utilizing

the tools from Johansen (2019), which propose methods to recover the adjustment and cointegration matrices of misspecified models, we explored how causality manifests in partially observed systems.

The primary objective of the proposed approach is to establish a general framework for causal analysis that does not depend on the unobserved causal relations among the analysed variables. Instead, our procedure aims to recover causal features regardless of the true shape of the latent graph depicting the causal connections within the entire system's data-generating process. In other words, our goal was to develop inferential procedures that do not rely on assumptions about the structure of the causal graph or the relationships between observed and unobserved components, such as acyclicity and causal sufficiency.

For example, our approach does not require absence of cyclicality. This is significant since mutual causality cannot be definitively ruled out, particularly in contexts involving long-term dynamics. Firstly, market adjustments often involve reciprocal interactions among economic forces. For example, in demand-supply models, quantities and prices influence each other to achieve equilibrium. Secondly, in the realm of long-term causality, the causal impact on the dependent variable is not directly initiated by its parents. Instead, movements are triggered by conditions of disequilibrium. For instance, in a causal graph represented as $x \to y$, the long-term dynamic of y is actually driven by the disequilibrium status, which can in turn be induced by movements of x. Therefore, the causal structure can be better depicted as $x \to$ Equilibrium Violation $\leftrightarrow y$. This concept similarly applies to the relationship between quantities and prices in demand-supply models. Indeed, the

causal link $p \leftrightarrow q$ represents a simplified illustration of $p \leftrightarrow$ Equilibrium Violation $\leftrightarrow q$, where p and q denote price and quantity, respectively. This observation is crucial as it challenges the a priori exclusion of cyclical causal behaviour. While it is true that if A generates B, then A cannot have reciprocally been generated by B, in this scenario, a third factor—specifically, a state of disequilibrium—drives the long-term dynamics. It follows that the actual source of generation of B is not A, therefore $A \leftrightarrow B$ cannot be ruled out beforehand.

Another important assumption not required by our analysis is causal sufficiency, which stipulates that any variable excluded from the model directly causes at most one variable within the set of modeled variables. This assumption proves overly restrictive in complex systems such as the economic one, where variables are intricately interconnected and the causal relationships are multifaceted. Consider, for instance, how external factors like climate change impact more than one critical economic variable. Climate change directly affects supply through disasters and damages to companies. It also indirectly influences demand by altering consumer awareness and preferences towards sustainable consumption and investment choices. This example illustrates the inadequacy of the causal sufficiency assumption in capturing the intricate interdependencies inherent in economic systems.

Our approach emphasizes and commences with the data generating process of a set of variables Y encompassing all variables influencing a system, and the causal graph $\mathcal{G}$ that delineates the causal connections among its components. From Y, we extract a subset X_o

comprising the variables observed and explicitly modeled by the user. The fundamental challenge lies in deciphering properties of $\mathcal{G}$ solely from $X_o \subseteq Y$. The set $Y_u = Y \setminus X_o$ should be disregarded because the model user may not have any knowledge over its components. To construct an approach that is entirely independent of Y_u it is crucial to avoid making assumptions about the causal relationships between Y_u and X_o. This ensures that the methodology remains robust and applicable regardless of the unobserved, and so unmanageable variables.

The initial step involves establishing and defining causal properties among the variables within X_o. These causal properties naturally needs to be associated with the nodes corresponding to X_o within the framework of the graph $\mathcal{G}$. The properties chosen in the thesis are new definitions of exogeneity that depart from their traditional statistical interpretation, and are instead grounded in the structure defined by $\mathcal{G}$.

The second step of our approach consists in understanding whether and how these properties manifest within the Vector Error Correction Model when modeling X_o only. Specifically, we examined their influence on the coefficients α_o and β_o of model

$$\Delta X_{o,t} = \alpha_o \beta_o' X_{o,t-1} + \sum \Gamma_i \Delta X_{o,t-i} + \varepsilon_{o,t}$$

To ensure the general nature of the analysis, it was necessary to establish that the relationship between the defined causal properties and the model coefficients remains consistent irrespective of the particular selection of X_o. In essence, the approach aims to demon-

strate a mapping between certain characteristics of $\mathcal{G}$ relative to the nodes in X_o and the coefficients α_o and β_o (or some transformation of them) for every arbitrary choice of X_o.

The approach outlined was introduced in the first chapter and further developed in the second and third chapters. The first chapter introduced the key concepts and initial findings that serve as the foundation for the entire thesis. The second chapter expanded on these ideas by proposing alternative inferential procedures and evaluating the test performance using simulated data. Chapter three focused on the causal interpretation of standard statistical tests, illustrating how drawing causal conclusions based on weak exogeneity tests could be misleading and providing tools for correctly interpreting the results of tests on the adjustment matrix restrictions.

More specifically, chapter one defined causal exogeneity, which manifests in both strong and weak forms. These definitions focus on the etymological roots of the terms exogeneity and endogeneity. The word exogenous derives from the Greek words "Exo-" meaning "outside" and "-genous" meaning "produced by" or "originating from," whereas the prefix "Endo-" in endogenous means "inside" or "within." These definitions exploit this primitive idea by concentrating on the source of generation of the variables in X_o. If all the parents of $x_i \in X_o$ are within X_o, then x_i can be considered internally generated, thus endogenous. Conversely, if some of the parents are outside the set of modeled variables, x_i is considered externally generated, thus exogenous.

There are cases where the parents of x_i are outside X_o, but the variables in X_o can perfectly control the nominal values of x_i. This occurs when causality is transmitted from some

components of X_o to x_i through unobserved components that can be fully governed by X_o.[2] Under this scenario, despite being caused by latent factors, it seems reasonable to treat x_i as endogenous, given that the generation of their long-term dynamic is totally dependent on the observed variables.

Since the novel definition of exogeneity is related to the causal structure, we call it causal exogeneity/endogeneity. The variables labeled as causally exogenous possess the ability to control the nominal limiting value of the causally endogenous components of X_o. In other words, if $X_o = \{A, B, C\}$, and C is causally endogenous, while A and B are causally exogenous, given an arbitrary value C_o for C, there always exists a combination of values for A and B resulting in C_o as the limiting value for C. This scenario is hypothetical since it implies that A and B can be modified at will, which can obviously not be the case.

Causal exogeneity, as described so far, is in its weak form. Strong causal exogeneity adds the further requirement that the causally exogenous variables are not in turn caused by the causally endogenous ones. Therefore, if $\aleph$ is the set containing the weakly causally exogenous variables and $\beth$ is the set containing the causally endogenous variables, the set $\aleph$ is also strongly causally exogenous if $\beth$ does not either directly or indirectly cause $\aleph$.

Naturally, the exogeneity/endogeneity status varies according to the composition of X_o. For instance, x_i may be causally exogenous in X_{o1} but causally endogenous in $X_{o2} \neq X_{o1}$. This variation arises because X_{o2} could include variables outside X_{o1} that can govern the nominal value of x_i.

[2] See the discussion in section 1.3

Causal exogeneity, in both its weak and strong forms, is a property based on the causal patterns in the graph $\mathcal{G}$. In fact, to determine whether the parents are within or outside X_o, as well as to ascertain whether the causal paths among components of X_o are direct or not, one must examine the causal graph depicting the causal relations of the whole set Y. What matters, is that those properties of the graph related to Y can be discovered by extracting information from the VECM fitted ignoring the latent variables.

Chapter 1 and Chapter 2 demonstrated that certain estimable transformations of the coefficients α_o and β_o possess properties that can be used to identify causal exogeneity and endogeneity, in both weak and strong forms. Specifically, Chapter 1 proved that the rows of $\Sigma_o^{-1}\alpha_o$ corresponding to $\aleph$ are linearly dependent, while the rows corresponding to $\beth$ are linearly independent. This duality was then exploited to construct an inferential procedure based on analyzing the effect of a row removal on the cointegration rank of the model: by dropping a linearly independent row, the rank of the resulting matrix decreases, whereas it remains unchanged when removing a linearly dependent row.

Chapter 2 extended the discourse over weak causal exogeneity by identifying a further duality between estimable parameters and the properties of the latent graph $\mathcal{G}$. It demonstrated that the rows of $(\Sigma_o^{-1}\alpha_o)_\perp$ are null vectors if they correspond to the variables in $\beth$. This duality was then used to construct an inferential procedure based on testing the restrictions of the orthogonal complements of $\Sigma_o^{-1}\alpha_o$. Chapter 2 also provides simulation results demonstrating the performance of the test. Additionally, it illustrates the practical application of these concepts by using the causal tools proposed in the first two chapters

to test for money endogeneity/exogeneity.

For what concerns strong causal exogeneity, Chapter 1 found that it can be tested by comparing the covariance matrix of the VEC model fitted exclusively to ℵ with the covariance matrix of the VEC model fitted to the entire set X_o. It has been shown that if the two covariances match, the variables in ℵ are also strongly causally exogenous.

Despite its connection to Chapters 1 and 2 through the analytical approach, Chapter 3 presents an independent study focused on the causal information content of the standard test of weak exogeneity. We reviewed examples from the literature to illustrate how this concept has been associated with driving trends and non-causality. By defining these concepts with an emphasis on their causal meanings, we examined the relationship between weak exogeneity and these notions. Our findings revealed that the informative content of weak exogeneity is limited in relation to driving trends and non-causality. Specifically, weak exogeneity serves only as a sufficient condition for a variable to be a driving force, while being uninformative for non-causality. Consequently, we clarified the correct approach that should follow from a causal interpretation of the outcome of weak exogeneity tests.

Identifying properties related to the causal interpretation of exogeneity and endogeneity is crucial for empirically evaluating many debates within the macroeconomic context. The macroeconomic debate concerning the causal relationships among economic variables and the distinctions between endogenous and exogenous factors is extensive and multifaceted, involving various schools of thought and theoretical perspectives. This debate has signif-

icant implications for economic modeling, and our understanding of economic dynamics. Economists often disagree on the direction of causality between key economic variables. For instance, there is ongoing debate about whether investment drives economic growth or if economic growth spurs investment. Similarly, there is contention over whether inflation results from an increased money supply or if other factors play a more significant role. Addressing these questions requires a robust empirical framework capable of discerning the causal relationships underlying the modeled economic phenomena.

Moreover, disagreements about the endogenous or exogenous nature of key variables, as well as their causal connections, significantly influence perspectives on the effectiveness of fiscal and monetary policies. For instance, Keynesians advocate for active policy interventions, whereas some modern approaches emphasize market self-regulation and caution against policy-induced distortions. Consequently, the study of causality in macroeconometrics is not just a tool to evaluate the validity of contrasting theories, but it also has a strong impact for practical pollicy aspects.

Despite the three chapters provide a comprehensive account of the causal analysis of cointegrated systems, the analysis is far from being exhaustive, and there is considerable scope for further development.

We identify three major areas where future studies should focus. The first area pertains to relaxing model assumptions. Further developments in this direction necessitate extending the work on partially observed models as discussed in Johansen (2019). Notably, the existing literature primarily addresses simple cases, such as C-VAR of order 1 without

deterministic components. This simple model does not allow to capture in full the complexity of the economic system. The second area of development involves the refinement of inferential procedures. There remains much to explore regarding how causal structures influence test performance and whether alternative procedures can be devised. Additionally, the asymptotic behaviour of tests for causal endogeneity requires investigation, especially given that they deviate from standard log-likelihood tests due to the estimation of unobserved left-hand sides of the regressions. The third area of development entails exploring the potential of these techniques to recover either the complete or a portion of the causal graph $\mathcal{G}$. This exploration aims to construct algorithms for discovering the architecture of the long-run causal structure.

We now turn our attention to the first area of development, focusing on relaxing model assumptions. The analysis is predicated on the assumption that the data generating process of the fully observed model (i.e., the process for Y) follows a simple C-VAR(1):

$$\Delta Y_t = \alpha\beta' Y_{t-1} + \varepsilon_t$$

The rationale behind selecting this simplified version of the data generating process lies in its alignment with the framework established by Johansen for analyzing partially observed models. Specifically, Johansen's methodology assumes that the fully observed model lacks short-term dynamics and does not incorporate any deterministic components, whether internal or external to the cointegration relationships.

One can find economic justification for selecting the simplest model, as the majority of macroeconomic theory emphasizes equilibrium adjustments, with less emphasis on short-term dynamics in economic literature. Thus, the straightforward C-VAR(1) model suffices to encapsulate the primary behaviours of economic systems, rendering an extension to a generic C-VAR(k) model merely a mathematical exercise. However, this viewpoint is only partially accurate, as dynamics based on equilibrium adjustments presuppose rational behaviour. This implies that when there is disequilibrium, agents react by adjusting prices, wages, and other economic factors rationally to maximize their utility, eventually reaching a new equilibrium level. Nevertheless, it is widely recognized that this adjustment mechanism encounters challenges when applied to real-world scenarios.

Firstly, prices and wages are often sticky and do not adjust immediately to restore equilibrium. However, this limitation primarily affects the speed of equilibrium adjustment rather than the overall structure of economic dynamics. Secondly, economic literature has highlighted the prevalence of irrational behaviour among agents. Cognitive biases, emotional and psychological factors, and social influences can all distort rational decision-making by agents. Moreover, the concept of bounded rationality is widely acknowledged, where individuals face constraints such as limited information, cognitive limitations, and time pressures, leading them to use heuristics or rules of thumb. These cognitive shortcuts, will generally result in irrational outcomes.[3]

[3]For an in-depth exploration of irrational behaviour and its implications in economics, seminal works such as Kahneman and Tversky (1979) introduce concepts like prospect theory and delve into decision-making under risk and uncertainty, departing from expected utility theory. Further studies on specific irrational behaviours can be found in Tversky et al. (1982).

The literature has also extensively documented numerous instances of market failures that necessitate government intervention, potentially affecting the equilibrium adjustment dynamics typical of the simple model employed in this analysis. A prominent example of market failure is the provision of public goods. Public goods are characterized by non-excludability, making it challenging to prevent their use, and non-rivalry, meaning one person's consumption does not diminish availability to others. National defense, clean air, and public parks exemplify public goods that markets often fail to provide efficiently. Another form of market failure arises from costs or benefits of economic activities that affect third parties not directly involved in transactions, known as externalities. Information asymmetry, monopoly power, incomplete markets, and factor immobility are additional phenomena contributing to market inefficiency, necessitating policy interventions that modify the adjustment behaviours assumed in the dynamic of the simple C-VAR(1) model.[4]

Short-term dynamics also manifest by considering certain mechanisms related to the formation of the expectations concerning future economic conditions, such as prices, incomes, interest rates, or overall economic growth. These expectations are often mathematically modeled with a dynamic proper of the short term model, due to the presence of anchoring on previous observations and beliefs. For example, extrapolative expectation theory posits that economic agents update their expectations by simply extrapolating from past

[4]For a comprehensive examination of market failures, their causes, and implications, consult Mas-Colell et al. (1995). For seminal works on specific market failures, see Akerlof (1978) for asymmetric information, Ostrom (1990) for public goods, and Coase (2013) for insights into externalities and the role of property rights in reducing social costs and enhancing economic efficiency.

trends or observations. This approach assumes that people expect future values of economic variables to follow the same pattern observed in the recent past. Mathematically, extrapolative expectations can be expressed as:

$$E_t = E_{t-1} + a(E_{t-1} - E_{t-2})$$

or in autoregressive form as

$$\Delta E_t = a\Delta E_{t-1}$$

Here, E_t denotes the expectation at time t, and a is a parameter reflecting the influence of ΔE_{t-1} on ΔE_t.

The concept of extrapolative expectations has been used to explain various economic phenomena, such as speculative bubbles, herd behaviour in financial markets, and short-term fluctuations in economic activity. It underscores the role of psychology and behavioural biases in shaping economic decisions and outcomes, highlighting the limitations of purely rational expectations models in capturing real-world economic dynamics. If this framework holds true, it suggests that the assumption of the fully observed model being a C-VAR(1) might be overly restrictive.

However, other expectation theories do not necessarily undermine the exclusive focus on long-term dynamics implied by the C-VAR(1) model. For instance, adaptive expectation are grounded on the concept of adjustment (Cagan (1956)). Moreover, as introduced in Muth (1961), expectations are often considered to be rationally formed, challenging

the simplicity of extrapolative models by proposing that economic agents incorporate all available information into their predictions. This perspective suggests that extrapolative expectations may be inadequate for explaining economic behaviour across all scenarios. Lucas (1976) further elaborated on this critique, arguing in his eponymous critique that policies based on models using adaptive expectations could fail, as agents adjust their behaviour in response to policy changes. Rational expectations theory asserts that economic agents optimize their forecasts given the available information, aiming to maximize their utility or profits. According to this theory, these optimizing behaviours align with the dynamic processes of a C-VAR(1) model, thereby not challenging the assumption of the data generating process (DGP) implied by the analysis in this thesis.

The ongoing debate among economists regarding rational, extrapolative, and adaptive expectations remains unresolved and continues to evolve with advancements in modeling techniques and insights from behavioural economics and empirical research. Each approach has distinct strengths and weaknesses, and the selection of expectation formation depends heavily on the particular context and behaviour under investigation. Consequently, assuming the absence of short-term dynamics may limit the generalizability of the analysis. If economic agents adjust their expectations according to extrapolative theory, and their behaviour deviates from strict rationality, this assumption could potentially yield misleading results. Thus, considering the complexities introduced by different expectation formations is crucial for accurate economic analysis and policy formulation.

Another limitation of the use of the simple C-VAR(1) model for Y is the absence of deter-

ministic components. A constant term or a deterministic trends in the dynamic of ΔX_t are typically added to vector error correction representations, both within and outside cointegration relations. Economic growth theories, such as the Solow growth model, often incorporate components of exponential growth to describe how capital accumulation and technological progress contribute to long-term economic growth rates.[5] Similarly, in financial economics, models also commonly utilize deterministic trends. For instance, asset pricing models often assume exponential growth in asset prices under idealized conditions. Furthermore, many economic variables are influenced by extra-economic factors that are also modeled with deterministic growth rates. For instance, in demography and population economics, models like the exponential growth model are commonly employed to describe how populations increase over time. For instance, a common mathematical representation is

$$P_t = P_0 e^{rT}$$

where P_t is the population at time t, P_0 is the population at time 0, and r is the growth rate.

These considerations challenge the assumption of the absence of deterministic components and the simplicity of the data generating process as a C-VAR(1), as previously illustrated. Thus, there is the need to include a constant term μ or a deterministic trend $\mu + \delta t$ within the representation of ΔY.

[5]It should be emphasized that these models typically also include factors that limit or modify exponential growth over time. For further information on Solow model, refer to the seminal paper Solow (1956).

Throughout the empirical applications of the thesis, the empirical approach we chose to face the problem related to the deterministic components by detrending variables to obtain series devoid of growth. However, while this approach appears to pose no immediate issues, it is crucial to investigate how it may impact Johansen's results on partially observed models. Furthermore, this methodological choice may lead to a loss of information concerning common deterministic components and those affecting cointegration relations.

To address the problems highlighted so far, an extension of Johansen's findings is necessary. Specifically, the analysis should start with the general model below

$$\Gamma_0 \Delta Y_t = \mu + \delta t + \alpha\beta' Y_{t-1} + \sum_{i=1}^{k} \Gamma_i \Delta Y_{t-1} + \varepsilon_t$$

and its state space representation

$$\Gamma_{0,o}\Delta X_{o,t} = -(\Gamma_{0,oT}\Delta T_t + \Gamma_{0,ou}\Delta X_{u,t}) + \mu_o + \delta_o t + M_o X_{o,t-1} + M_{ou} X_{u,t-1} + N_o T_{t-1} +$$

$$+ \sum_{i=1}^{k} \Gamma_{i,o}\Delta X_{o,t-1} + \sum_{i=1}^{k} \Gamma_{i,u}\Delta X_{u,t-1} + \sum_{i=1}^{k} \Gamma_{i,oT}\Delta T_{t-1} + \varepsilon_{o,t}$$

$$\Gamma_{0,u}\Delta X_{u,t} = -(\Gamma_{0,uT}\Delta T_t + \Gamma_{0,uo}\Delta X_{o,t}) + \mu_u + \delta_u t + M_{uo} X_{o,t-1} + M_u X_{u,t-1} + N_u T_{t-1} +$$

$$+ \sum_{i=1}^{k} \Gamma_{i,u}\Delta X_{u,t-1} + \sum_{i=1}^{k} \Gamma_{i,uo}\Delta X_{o,t-1} + \sum_{i=1}^{k} \Gamma_{i,uT}\Delta T_{t-1} + \varepsilon_{u,t}$$

$$\Gamma_{0,T}\Delta T_{u,t} = -(\Gamma_{0,Tu}\Delta X_{u,t} + \Gamma_{0,To}\Delta X_{o,t}) + \mu_T + \delta_T t + \sum_{i=1}^{k} \Gamma_{i,Tu}\Delta X_{u,t-1} +$$

$$+\sum_{i=1}^{k}\Gamma_{i,To}\Delta X_{o,t-1}+\sum_{i=1}^{k}\Gamma_{i,T}\Delta T_{t-1}+\varepsilon_{T,t}$$

where $\Gamma_{i,o}$, $\Gamma_{i,u}$, and $\Gamma_{i,T}$, indicates the submatrices of Γ_i constructed by considering the rows and columns relative to X_o, X_u, and T respectively, $\Gamma_{i,ou}$, $\Gamma_{i,oT}$, are the sumbatrices of Γ_i constructed by considering the rows of X_o and the columns of X_u and T respectively, $\Gamma_{i,uo}$, $\Gamma_{i,uT}$, are the sumbatrices of Γ_i constructed by considering the rows of X_u and the columns of X_o and T respectively, and $\Gamma_{i,Tu}$, $\Gamma_{i,To}$, are the sumbatrices of Γ_i constructed by considering the rows of T and the columns of X_u and X_o respectively. μ_T, μ_o, μ_u are the rows of μ corresponding to T, X_o, and X_u. Similarly, δ_T, δ_o, δ_u are the rows of δ corresponding to T, X_o, and X_u.

Starting from the state-space representation provided, one should follow a procedure analogous to Johansen's methodology to derive the representation of α_o and β_o in model

$$G_o\Delta X_{o,t}=C_o+D_o t+\alpha_o\beta_o X_{o,t-1}+\sum G_i\Delta X_{o,t}+\varepsilon_{o,t}$$

as a function of the coefficients from the fully observed VECM. This task immediately presents increased complexity compared to the standard procedure since the inclusion of additional coefficients can also make necessary adjustments to the methodology.

Once the formulae for α_o and β_o are derived, extending the findings of chapters 1 and 2 with these new representations becomes possible to verify whether the conclusions still hold under general conditions. However, several other challenges arise in this context. For instance, there is the critical issue of identifying the matrix G_0, which captures the

contemporaneous short-term effects of ΔX_o. This identification is essential to distinguish the structural coefficients α_o from $G_o^{-1}\alpha_o$. A way to address this problem could be the use of the techniques from graphical causal inference, already employed in econometric literature for uncovering contemporaneous causality in structural VAR models.

The introduction of short-term dynamics also raises questions about whether it is feasible to tackle a dual causal inference problem, specifically whether it is possible to infer certain properties of the causal graph $\mathcal{G}_s$, where $\mathcal{G}_s$ denotes the network that illustrates short-run causal connections, encapsulated in $\{\Gamma_0, \Gamma_1, \ldots, \Gamma_k\}$. Addressing this would be particularly pertinent for identifying which policy interventions could induce long-term impacts, permanently altering equilibrium positions, versus those that may only yield temporary benefits dissipating over time or potentially leading to adverse effects in the long run. To achieve this goal, it is necessary to extend the partially observed analysis beyond just the coefficients α_o and β_o; it also requires developing a representation for G_i as a function of the parameters in the fully observed model. These aspects are inherently complex and will necessitate substantial further research. Refinement of definitions and the construction of theoretical frameworks will likely be essential for establishing a comprehensive basis for the general analysis.

Thus far, we have outlined future developments aimed at relaxing the assumptions of the model. Now, we shift our focus to enumerate and analyse further advancements concerning the inferential procedures and test construction for identifying the causal aspects of variables within X_o.

The first extension on this matter concerns the test for strong causal exogeneity. Currently, there is a lack of studies evaluating the performance of tests using simulated data to understand how the structure of the causal graph influences test efficiency and power. Furthermore, the methodology proposed in this thesis does not allow testing subsets of $\aleph$; rather, it restricts analysis to the entire set $\aleph$ only. Consequently, it is possible to ascertain whether the set of weakly causally exogenous variables is also strongly causally exogenous, but it is not feasible to test whether strong causal exogeneity holds for only a subset of these variables. As illustrated in the examples from chapter 1, $\aleph$ can be subdivided into two subsets: $\aleph_w$ and $\aleph_s$. The former comprises variables within $\aleph$ that lack strong exogeneity, while $\aleph_s$ encompasses components of $\aleph$ that exhibit both weak and strong causal exogeneity. Identifying $\aleph_s$ when $\aleph_w$ is not empty necessitates the development of an alternative inferential procedure. This development is crucial as it would enable the differentiation of various degrees of exogeneity among variables within $\aleph$.

Regarding the existing tests relative to weak causal exogeneity, a crucial area requiring further investigation pertains to the precise asymptotic behaviour of the test statistic used for assessing causal endogeneity. The distributions employed in the thesis, such as the χ^2 and Dickey-Fuller distributions, are just approximations of the true ones. In fact, they hold if the variable $Q = \Sigma_o^{-1} R_0$ is observed. However, in practice, Σ_o must be estimated, leading to the substitution of Q_t with $\hat{Q}_t$. This substitution introduces an error on the left-hand side of the equations, potentially influencing the distribution of the test statistic. While it is theoretically expected that the bias decreases as the sample size N increases, due to

$\hat{\Sigma}_o$ converging to Σ_o, rigorous examination of this aspect is necessary. This is particularly critical for small sample sizes, where such biases can markedly impact the reliability of the test results. Therefore, further empirical and theoretical studies are warranted to accurately characterize these effects and refine the inferential procedures accordingly.

The impact of using estimates rather than exact values is evident from the simulations conducted in chapter 2. These simulations revealed a rejection rate slightly deviating from the chosen significance levels when the null hypothesis is true. Specifically, in the first test, which hinges on the linear independence of the rows of γ the rejection rate consistently fell below the specified significance level. Conversely, in the second test, which relies on the restrictions imposed on $\gamma_\perp$ the rejection rate consistently exceeded the significance level. To address this issue, a comprehensive examination of the asymptotic properties of the test statistic is warranted. Moreover, it is essential to explore potential small sample effects, especially given that macroeconomic datasets often comprise limited annual observations. Such an investigation is crucial for refining the inferential procedures and ensuring their robustness under various empirical conditions.

Other tests extensions should aim to address practical challenges arising from borderline results in cointegration rank tests. It is not uncommon to encounter test statistics very close to critical values for certain values of the cointegration rank r, where slight variations in the significance level could lead to different outcomes. This issue holds particular significance for the test on causal endogeneity proposed in chapter 1, since an accurate identification of the cointegration rank is crucial for determining whether the rank of the

modified model has decreased. Incorrectly setting the rank r, such as underestimating it, may fail to identify a reduction in rank. Conversely, overestimating the rank could erroneously identify a rank reduction that did not occur.

To address this issue, an alternative approach could be pursued by focusing on a standardization of the difference between the test statistics of the original model ($t1$) and the one of modified model ($t2$). The objective would be to investigate whether this standardized difference follows a specific distribution under the null hypothesis $t1 = t2$. Such an approach offers potential advantages, particularly in scenarios where the exact cointegration rank r is challenging to identify accurately. Instead of directly testing hypotheses about r, this method focuses on comparing the magnitudes of test statistics, which may provide robustness against mis-specification of r. Exploring the distributional properties of this standardized difference could enhance the reliability of causal inference procedures, especially when dealing with borderline cases where traditional rank determination methods may be ambiguous.

In the context of the third chapter, there exists potential for further advancements in devising a test to identify the driving forces within the set $\aleph_T$. As demonstrated, both weak exogeneity and weak causal exogeneity do not invariably correspond to the set of variables causally driving the dynamics of interest. Weak exogeneity serves merely as a sufficient condition for a variable to be included in $\aleph_T$, whereas weak causal exogeneity is just a necessary condition, given that $\aleph_T \subseteq \aleph$. Hence, there is a critical need to develop alternative techniques capable of distinguishing which components within $\aleph$ truly drive

the long-term dynamics of the variables under examination.

The third area of development concerns the construction of algorithmic procedures aimed at recovering causal structures. This entails developing iterative methods that leverage insights from the three preceding chapters to enable researchers to uncover segments of the causal graph $\mathcal{G}$. The overarching strategy involves integrating various tests to identify the skeleton of $\mathcal{G}$ and subsequently determining the directionality of its edges. This approach aligns with the principles of graphical causal inference, a burgeoning field that in the long-run framework finds application in Hoover (2020). Hoover's work lays a foundation for a long-run causal discovery algorithm by examining weak exogeneity and cointegration patterns through multiple inferential procedures applied to diverse subsets of observed variables. Exploring the potential adaptation of these ideas alongside the tests introduced in this chapter and integrating them with Hoover's findings could yield a robust causal discovery algorithm.

To have some preliminary ideas of how a causal discovery algorithm based on the findings of this thesis might function, consider a set of observed variables $X_o = \{A, B, C\}$ and assume that after testing for causal exogeneity, it is observed that $\aleph = A$ and $\beth = \{B, C\}$. Furthermore, after testing for strong causal exogeneity, it is determined that A is strongly causally exogenous. From this, it follows that causality from B and C to A is excluded, consistent with the definition of strong causal exogeneity. Moreover, given $\aleph$'s definition, A must directly or indirectly cause both B and C. Thus, from this initial round of tests, we infer $A \to \{B, C\}$, but the relationships between B and C remain unclear. To gain

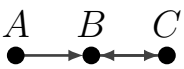

Figure C.1: Graph Estimated by Iterative Implementation of Causal Exogeneity Tests

further insights, we repeat the two tests for the subset $\{B, C\}$, excluding A from the analysis. Suppose we find that B belongs to ℵ while C belongs to ℶ. This suggests that B influences C, either directly or indirectly, and B blocks causal paths between A and C, thus mediating causality from A to C. Consequently, we exclude a direct edge from A to C. Suppose now that the hypothesis of strong causal exogeneity for B is rejected, implying a causal feedback from C to B. This scenario indicates that the estimated causal graph will have a chain-type structure: A on top of the causal order, B in the middle and C at the bottom, with causal feedback from C to B, as illustrated in Figure C.1

It should be noted that the estimated causal graph is just a "summary" of the true structure of $\mathcal{G}$. This summary is limited not only because it considers a subset of variables in Y, but also because it cannot reveal the precise transmission of causality among X_o. For example, it cannot ascertain whether causality from A to B is mediated by 2, 4, 9 variables, or whether it is direct. The same ambiguity applies to other causal connections within the graph. Figure C.2 illustrates three of the infinite possible true graphs $\mathcal{G}$ that can generate the patterns of weak and strong causal exogeneity among $X_o = \{A, B, C\}$ as previously assumed. While the graph in Figure C.1 may not perfectly match those in Figure C.2, it effectively summarises the counterfactual aspects of all the graphs in the latter figure. Consider for instance graph i): despite C not being directly connected to B, by modifying C there will be an effect on D which in turn influences D. Therefore,

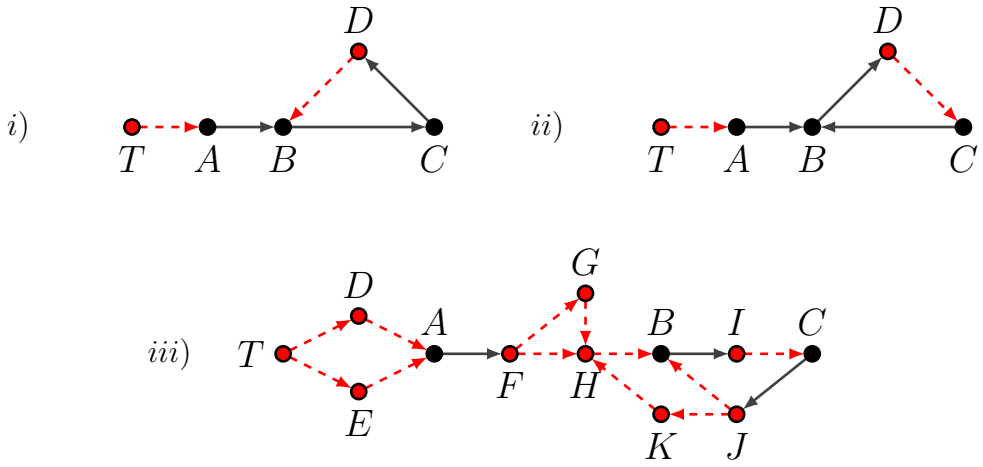

Figure C.2: Examples of Possible Causal Structures of the Fully Observed Model Given the Causal Exogeneity Patterns Generating Graph C.1

without controlling D causality can flow from C to B, and the path $C \to D \to B$ can be simplified to $C \to B$. A similar reasoning applies to the indirect connection between B and C in graphs *ii*): if D is uncontrolled, causality can flow from B to C through D, and the graph $B \to D \to C$ can be reduced to $B \to C$. The connections in graph *iii*) appear more complex, but the underlying logic remains the same. An intervention on A affects B through the mediation of the unobserved variables F, G, and H. Hence, $A \to B$. Similarly, an arbitrary modification of B has an effect on C via the influence on I, leading to the representation $B \to C$. Unobserved variables J and K transmit causality from C to B, resulting in $C \to B$. Notably, there is no direct path from C and A, indicating that modifications of C cannot influence A, thus $C \not\to A$. Furthermore, if the observed B is kept fixed, causality from A to C is blocked, in the sense that by keeping B constant, an intervention on A has no effect on C, thus $A \not\to C$. Combining these results yields the causal graph depicted in Figure C.1.

The example provided here was deliberately simplified for illustrative purposes. It is important to note that causal exogeneity patterns do not necessarily correspond to unique causal structures. In cases where multiple causal structures can produce the same exogeneity patterns, alternative techniques are required to distinguish between causal connections. One potential approach could involve integrating this analysis with previous methodologies, such as those proposed by Hoover (2020). Future research should explore the existence of observational equivalence in causal structures and develop strategies to address such scenarios effectively. Furthermore, there is a need for clearer guidelines to inform the development of causal discovery algorithms. These guidelines should delineate the key principles and methodologies that such algorithms should incorporate to accurately uncover causal relationships.

Throughout this final chapter, we have proposed three areas for further development. However, the avenues for research in long-run causal inference extend beyond the specific recommendations put forth here, encompassing topics that may not directly align with the methodologies advanced in this thesis. For instance, it would be valuable to explore connections between the concept of causal exogeneity and other econometric definitions, such as weak exogeneity with parameters of interest different from the cointegration matrix or the notion of super exogeneity, which has been traditionally related to causality in the econometric literature.

The causal inference literature plays a crucial role in facilitating robust empirical analy-

ses of economic theories and in formulating effective policy strategies. Its significance is underscored by two primary reasons. First, much of the macroeconomic discourse hinges on disagreements over causal relationships and the identification of key variables driving general economic dynamics. Second, causal inference allows data to speak freely, without requiring models that rely on identifying restrictions often shaped by the subjective beliefs of the modeler. By reducing reliance on these assumptions, econometric tools not only test economic theories but also uncover underlying economic mechanisms. This shift enables econometric tools to transition from passive tools to active contributors within the field of economics.

The findings presented in this thesis aim to contribute to the literature on causal inference by expanding the analytical toolkit available for understanding economic mechanisms. We strongly believe that greater emphasis on long-run causal inference, complementing traditional short-run analyses, is essential since long-run dynamics align more closely with the equilibrium dynamics widely accepted in macroeconomics. Our hope is that the methodology developed here can serve as a foundation for constructing new and more effective econometric tools capable of operating in settings with minimal assumptions.

As econometricians, we need to acknowledge that nearly every economic inquiry is rooted in causal reasoning, and that the problems that the economic literature is called to solve require counterfactual understanding. This observation is true despite some authors attempted to avoid explicit causal language; an exercise that often results in a clumsy strategy merely leading to the employment of synonyms for the sentence “is caused by”,

thereby hiding or making just implicit the causal logic, rather than eliminating it, much like sweeping dust under the carpet. In developing tools for causal inference, we need to consider that economics involves the study of complex systems where interconnected factors interact in intricate ways. The social world is perhaps more intricate than the natural world, comprising a multitude of interdependent elements that mutually influence each other, thereby forming a dynamic and perpetually evolving network of relationships. Adding to this complexity are human beings, complex entities whose interactions further contribute to the intricacies of these systems. To compound matters, econometricians must navigate contexts where variables are uncontrollable, rendering phenomena unreproducible in experimental settings. This inherent challenge underscores the complexity of the econometrician's work, which simultaneously presents stimulating intellectual opportunities as it compels us to grapple with the daunting task of discerning causation from mere passive observation of associations.

Appendix A

Data Sources Chapter 2

Long-term interest rate:

Organization for Economic Co-operation and Development, Interest Rates: Long-Term Government Bond Yields: 10-Year: Main (Including Benchmark) for United States

[IRLTLT01USQ156N], retrieved from FRED, Federal Reserve Bank of St. Louis. Percent, Quarterly, Not Seasonally Adjusted

Link: `https://fred.stlouisfed.org/series/IRLTLT01USQ156N`

Short-term interest rate:

Organization for Economic Co-operation and Development, Interest Rates: 3-Month or 90-Day Rates and Yields: Interbank Rates: Total for United States [IR3TIB01USQ156N], retrieved from FRED, Federal Reserve Bank of St. Louis

Link: `https://fred.stlouisfed.org/series/IR3TIB01USQ156N`

Consumer Price Index:

Organization for Economic Co-operation and Development, Consumer Price Index: All Items: Wage Earners: Total for United States [CPALWE01USQ661N], retrieved from FRED, Federal Reserve Bank of St. Louis. Index 2015=100, Quarterly, Not Seasonally Adjusted

Link: `https://fred.stlouisfed.org/series/CPALWE01USQ661N`

Economic Output:

U.S. Bureau of Economic Analysis, Gross National Product [GNP], retrieved from FRED, Federal Reserve Bank of St. Louis. Quarterly, Billions of Dollars, Seasonally Adjusted Annual Rate.

Link: `https://fred.stlouisfed.org/series/GNP`

Money Supply:

Organization for Economic Co-operation and Development, Monetary Aggregates and Their Components: Broad Money and Components: M3 for United States

[MABMM301USQ189S], retrieved from FRED, Federal Reserve Bank of St. Louis. Quarterly, US Dollar, Seasonally Adjusted.

Link: `https://fred.stlouisfed.org/series/MABMM301USQ189S`

Appendix B

Data Sources Chapter 3

Labour productivity:

Data Source: Hill, Thomas, Dimsdale (2016), A Millennium of UK Data (2016), Bank of England

Labor productivity per hour is measured as gross domestic product (GDP) per hour of work. The time series is measured relative to productivity perhour of work in 2013 (i.e. 2013 = 100).

Link: `https://ourworldindata.org/grapher/labor-productivity-per-hour?time=earliest..2016`

Other Data:

All the other data are souced from Òscar Jordà, Moritz Schularick, and Alan M. Taylor. 2017. "Macrofinancial History and the New Business Cycle Facts." in NBER Macroeconomics Annual 2016, volume 31, edited by Martin Eichenbaum and Jonathan A. Parker.

Chicago: University of Chicago Press.

Link: `https://www.macrohistory.net/database/`

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